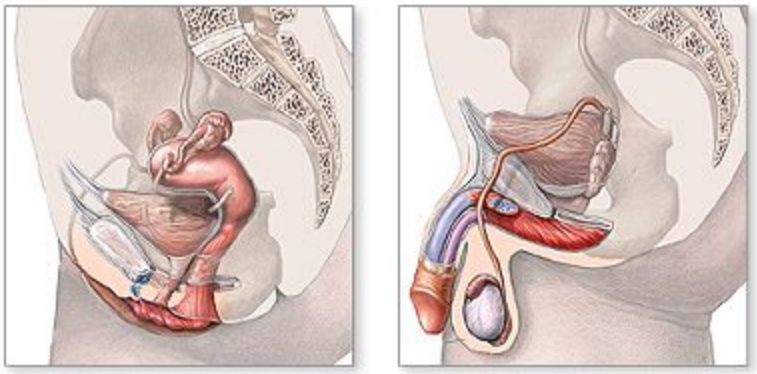
2- Transplacental infection

Microbiology 435's Teamwork
Reproductive Block



إِنَّا كُلَّ شَيْءٍ خَلَقْنَاهُ بِقَدَرٍ ﴿٤٩﴾

Learning Objectives:

- To recognize the different types of infant infections.
- To know major transplacentally transmitted pathogens causing congenital infections.
- To know the structure, epidemiology, clinical manifestations, diagnosis, treatment and preventive measures of (Toxoplasma, TP, ParvoV, VZV, Rubella V & CMV)

Please note that treponema pallidum (syphilis), and herpes will not be discussed in this lecture, as they will be in other upcoming lectures.

Important
Males notes
Females notes
Extra

Revised by
خولة العماري & هشام الغفيلي

Resources: 435 females only slides and notes (should be enough according to the doctor), Lippincott, Wikipedia, others...
Editing file: Here
Credit: Team members

Infant Infections

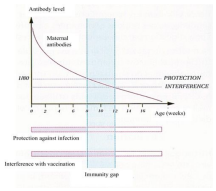
Classification	Timing of events	Mechanisms
Congenital	In utero	Trans placental
Perinatal	During labour and delivery through infected birth canal	Exposure to genital secretions & blood
Neonatal	After birth	• Direct contact with the mother • breast feeding or nosocomial exposure

Congenital Infections (Transplacental Infections)

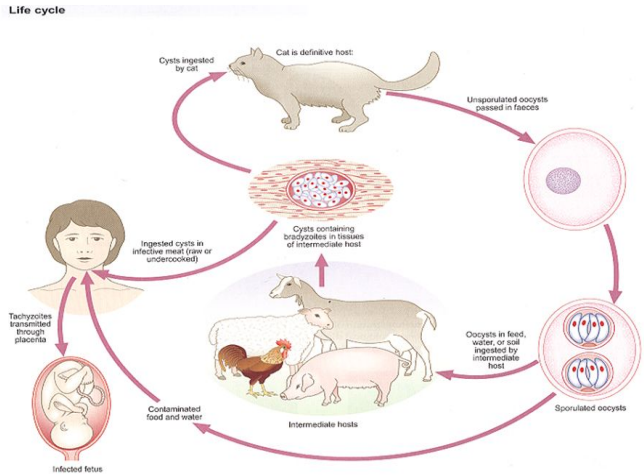
Introduction	
Etiology: 	• Mostly by **viruses**, previously known as (TORCH) infections: T= Toxoplasmosis (Toxoplasma gondii) the main pathogen cause TPI. it is a protozoan parasite O=Others: Treponema pallidum (causes syphilis), Parvovirus & Varicella Zoster Virus (VZV). R=Rubella Virus C=CMV H=Herpes, Hepatitis & HIV *mainly perinatal (during delivery), not congenital infection

Risk of Intrauterine infection & fetal damage	The ability of the organism to cross the placenta & cause fetal damage depends on:
	• **Type of organism** (Teratogenicity)
	• **Type of maternal infection**
	  ○ Primary "for the 1st time"
	  ○ Recurrent "the mother has been exposed to the infection before and she formed Ab., In case of reactivation of latent disease, these Ab. will protect the fetus."
	• **Time during pregnancy** (1 st "most severe", 2nd, 3rd Trimesters)
	Primary Maternal infection in the first half of pregnancy poses the greatest risk to the fetus, because cell division is intensive and organs are starting to develop, thus any disturbance during this period will cause congenital anomalies

Common Findings of congenital infection	• Wide spectrum of manifestations. Ranging from asymptomatic infections to severe infections which result in death and cause abortion.
	• Majority of congenital infections are "**asymptomatic**" at birth
	• who are sick at birth tend to have significant signs and symptoms, including:
	  ○ Intrauterine **growth** retardation (IUGR) = small fetus " low birth weight"
	  ○ Hepatosplenomegaly (HSM) with jaundice
	  ○ Thrombocytopenia resulting in rash
	  ○ Microcephaly or hydrocephalus
	  ○ Risk of developing neurological or eyes abnormalities later in their life

Neonatal serological Dx in the infant IgM= acute IgG= chronic	• Detection of specific IgM antibodies (BUT the **Absence** of fetal IgM at birth does **not** exclude infection, because most of the anomalies are due to primary infection at ~20 week of gestation, so the baby's immune system haven't developed yet) IgM is a pentamer, it is too big to cross the placental barrier. If IgM is positive it is diagnostic, if it is negative we can't exclude the infection as the baby's immune system didn't develop fully yet. • **Persistence of specific IgG antibody more than 12 months[1].** حطوا تحتها عشرين خط The transplacental (maternal) Ab. appear during the first 3-6 months after birth and then decrease and disappear after 12 months. Thus if we detect IgG after 12 months, that means it is produced actively from the infected infant, not by the mother. ركزوا إننا قلنا لو استمر وجوده معناها بدأ يطلع من طفل " مصاب " لكن لو شفناه وعمره كان ٨ شهور ورجعنا اختبرناه وعمره سنه وشهر وما لقيناه معناها الطفل مافيه إلا العافية 
Management	Preventative and therapeutic measures are possible for **some** of the agents

1. Toxoplasma Gondii

Toxoplasma: Morphology	• Obligate intracellular parasite (protozoa) that has three forms: ○ **Oocysts:** Shed in **cat feces** عشان كذا دايم يقولون للحوامل لا تقربين القطط ○ **Tachyzoites:** (tachy = rapid) rapidly dividing forms, seen in body fluids (acute phase). ○ **Bradyzoites:** (brady=slow) slowly dividing forms that are contained in cysts in muscle and brain tissue and in the eye (chronic phase). Cell-mediated immunity will stop Tachyzoites and cause the formation of cysts containing Bradyzoites to limit its spread, if the patient is immunocompromised, the Bradyzoites "remember they are inside the cyst not in the blood" will activate in the form of Tachyzoites to cause an acute infection again "in the blood "
Transmission (zoonotic disease) Life cycle 1:12	• Ingestion of **oocyst** (through fecal oral route): fingers, soil, water contaminated with cat feces • Ingestion of **cyst** (containing bradyzoites) in undercooked **meat**. • Blood transfusion and organ transplant. • Transplacental route (by tachyzoites) 

[1] This applies for persons ≥12 months of age **when maternal antibodies are no longer present.**

<table>
<tr><td>Manifestations</td><td colspan="2">

Most cases of congenital toxoplasmosis are due to primary maternal infection.
Rarely caused by <u>reactivation</u> of a latent infection (bradyzoites transform into tachyzoites in the immunocompromised patient e.g. pregnancy, HIV, cancer)
The highest transmission rate is in the <u>third</u> trimester. The most severe symptoms are if transmission occurred during the <u>first</u> trimester. i.e. If the mother gets the infection in the third trimester, there is a high possibility that it will be transmitted to the fetus, but baby's symptoms are going to be the less severe "thanks god".
Most (70-90%) are asymptomatic at birth but are still at high risk of developing abnormalities later, especially of the eye (chorioretinitis) and neurologic disease (Mental retardation) .
The classic triad of symptoms:
Chorioretinitis[2], Hydrocephalus & Intracranial calcifications[3] .
Other signs include: rash, Hepatosplenomegaly, jaundice, lymphadenopathy, microcephaly, seizures, thrombocytopenia, deafness.
Abortion & intrauterine death higher with infection in 1st trimester.

</td></tr>
<tr><td rowspan="2">Diagnosis

*Important to differentiate between primary and recurrent inf.</td><td>Pregnant mother</td><td>

Serology:

IgM, IgG, IgG avidity[4], and IgG seroconversion[5] compared to booking blood[6]
VERY IMP EXPLANATION: If the maternal blood positive for IgM but negative for IgG indicate primary infection, however if the maternal blood positive for IgG & IGM We can't know so we have to do IgG avidity test, if IgG avidity test shows LOW avidity that indicate primary infection, But if it shows HIGH avidity that indicate recurrent infection..هالكلام ينطبق على جميع اللاورقانزم بهالدرس
</td></tr>
<tr><td>Infant</td><td>

<u>Pre</u>natal Dx: PCR (detection of the Toxoplasma genome from amniotic fluid), Culture or Serial Ultrasound (to detect anomalies)
<u>Post</u>natal Dx:

Serology by detecting IgM (again, negative results doesn't exclude infection), or persistent IgG more than 12 months
PCR
Culture (isolation of Toxoplasma)
Evaluation of infant (e.g., neuroimaging, ophthalmic/pituitary/CNS functions). Because as we said, most infants are asymptomatic at birth.

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<tr><td>Treatment</td><td colspan="2">

Spiramycin[7]. indicated to <u>MOTHER</u> to treat primary maternal infection, it decreases the transmission of Toxoplasma to the fetus but it doesn't treat the fetus once the infection has occurred
Pyrimethamine combined with sulfadiazine. once transplacental infection has been confirmed, we must shift the treatment to these drugs. It is also recommended for infant with symptomatic and asymptomatic congenital infections for one year after delivery.

يعني اكتشفنا إن الجنين أصيب نبدأ نستعمل هذا الدواء لين تولد بالسلامة بعدها نعطيه للطفل بعد الولادة لمدة سنة
</td></tr>
<tr><td>Prevention</td><td colspan="2">
No vaccine is available.

Avoid exposure to cat feces;
Wash: hands with soap and water, wash fruits & vegetables, wash surfaces that touch fruits, vegetables & raw meat.
Cook all meats thoroughly

</td></tr>
</table>

[2] inflammation of the choroid (thin pigmented vascular coat of the eye) and retina. Presenting as edema & bleeding
[3] Calcification **anywhere** in the Skull
[4] IgG antibodies produced following primary infection have low avidity (low binding strength). Two to four months following infection, IgG antibodies mature to high-avidity (high binding strength). Therefore, avidity assays can be used to assess low avidity (which indicates recent infection) versus high avidity (past infection).
[5] seroconversion (1st sample IgG negative, 2nd sample IgG positive) is clear evidence for recent primary infection.
[6] بوكينق بلود :اللي هو عينة دم تؤخذ من الحامل اول ماتحمل (وهي معافاة مافيها شي)),,فنقوم نقارن هذي العينه بعينة دم جديدة تؤخذ خلال المرض
[7] spiramycin is a macrolide antibiotic and antiparasitic It is used to treat toxoplasmosis and various other infections of soft tissues.

2. Parvovirus B19

Morphology

- **Family:** Parvoviridae. parvo=small. (the smallest of the DNA viruses)
- **Structure:** nonenveloped, Icosahedral capsid & **ssDNA** genome.
- في الغالب الفايروسات اللي تحتوي على DNA يكونون دبل لكن البارفو فايروس استثناء

Epidimiology

- Worldwide distribution
- Humans are known hosts. the only reservoir, so it's not zoonotic like the Toxoplasma.
- **Transmission:**
  1. Respiratory route
  2. Transplacental route
  3. Blood transfusion

Clinical presentation

Acquired infection (after birth)

- **Immunocompetent host:**

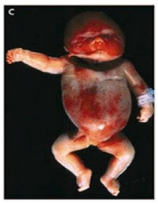
Erythema infectiosum[8] is the most common clinical presentation (slapped-cheek appearance, acute febrile illness).

- **Immunocompromised pts[9]**

Congenital infection (before birth)

Risk of congenital infection is greatest when infection occur in 1st 20 wks..

- Infection in the 1st trimester → IUD (Intrauterine death) 2-6 %
- Infection in the 2nd trimester → HF (Hydrops fetalis)*
- Infection in the 3rd trimester → Lowest risk



*Parvovirus is known to cause fetal loss (hydrops fetalis) through: **severe anaemia** (due to the destruction of the RBCs by B19) → **congestive heart failure** (myocarditis) → **generalized oedema & fetal death.**

Daignosis

Pregnant mother

Investigations of mother in general always by serology

- Specific Ig**M**, Ig**G** seroconversion.

Prenatal

Prenatal investigations in general always by PCR or culture "but you should look at the case first, could we culture or not"

- Ultrasound (to detect hydrops "edema")
- Doesn't grow in cell culture.
- PCR should be performed to detect the viral DNA

The diagnosis based on PCR only in this case because the hydrops detected by ultrasound is not always caused by the parvovirus.

Treatment

No specific treatment. Intrauterine blood transfusion provides blood to fetus when fetal RBCs are being destroyed. We can treat symptoms e.g. digoxin for CHF

Prevention

*Hygiene practice * No vaccine available (still in TRIAL تجريبي)

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[8] Fifth disease is a mild rash illness caused by parvovirus B19. This disease, also called erythema infectiosum, got its name because it was fifth in a list of historical classifications of common skin rash illnesses in children.

[9] B19, has been isolated and identified as the cause of transient **aplastic crisis** in patients with **sickle cell disease** and implicated in adult acute polyarthritis.

3. Varicella Zoster Virus (VZV)

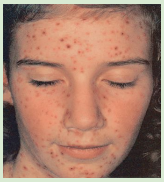
Morphology

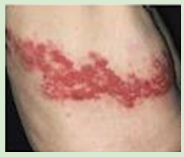
- **Family:** Herpesviridae
- **Structure:** dsDNA, Enveloped, Icosahedral Virus

Transmission

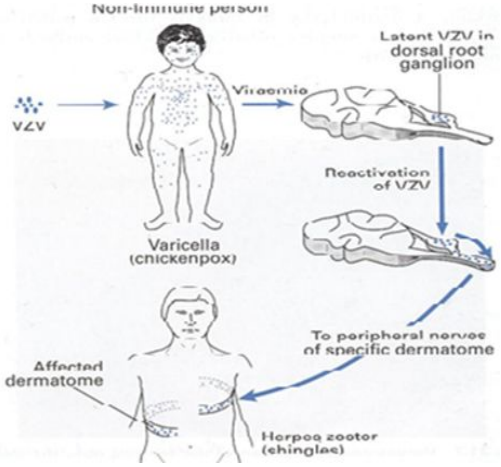
- Respiratory & Transplacental routes

Clinical presentation





Acquired infection[10]:



1) **Varicella (Chickenpox العنقز):** (Primary illness) follows initial exposure to the virus and is typically a relatively mild, self-limited childhood illness with **Generalized vesicular Rash.**
   * Primary infection in a pregnant lady carries a greater risk of severe disease, in particular: pneumonia (rare)
2) → Then the virus travels to dorsal root ganglia and stay there for years (become latent)
3) **Zoster (Shingle الحزام الناري):** (Recurrent infection) When there is diminished immunity, the virus may reactivate & spread to the corresponding dermatome causing **localised** vesicular painful Rash.

Intrauterine infections:

- **Congenital infection:** **Congenital varicella syndrome (CVS)** incidence is ~1- 2%
  - Occur when mother acquires the infection during ***1st*** **20 weeks** of Pregnancy
  - Characterised by:
    - Scarring of skin
    - Hypoplasia of limbs
    - CNS defects with mental retardation and microcephaly
    - eye defects with Chorioretinitis/ blindness
- **Neonatal varicella:** When mother acquires the infection….
  - **Less than 5 days** before delivery or 2 days after delivery:
    the neonate is more likely to have **severe** infection (there is no time for the maternal Ab to be produced and cross the placenta to protect the baby)
  - **More than 5 days** before delivery:
    the neonate is more likely to have **mild** disease عشان الأجسام المضادة اللي اكتسبها من جسم أمه تحميه

Diagnosis

Pregnant mother

- **Direct Examination:**
  - Culture: Vesicular **fluid (VF)** for virus isolation (take long time)
    بتكون على شكل حويصلات جلدية فيها سائل يتم عزل الفايروس من هذا السائل لغرض التشخيص chicken box
  - More rapid: **Cells** scraping from the base of **vesicles** (from the lesion itself) → **I**mmuno**F**luorescent test to detect viral antigen **(Ag)**
  - **PCR** to detect DNA-VZV (rapid results)
- **Serological test:** Ig**M** Ab.

[10] Reye syndrome, an acute encephalopathy accompanied by fatty liver, can sometimes follow VZV or influenza infections in children. Epidemiological evidence suggests that use of aspirin or other salicylate-containing compounds to treat pain and fever during the viral illness is associated with the development of Reye syndrome. It is also important to avoid aspirin following vaccination against chickenpox.

	Infant	• **Prenatal Dx:**
		  ○ Ultrasound
		  ○ PCR: VZV DNA in fetal blood[11] or amniotic fluid or placental villi
		  ○ VZV Ig**M** in fetal blood.
		
		• **Postnatal Dx:**
		  ○ VZV **IgM** (again, negative results doesn't exclude infection)
		  ○ virus isolation (culture)
		  ○ PCR: VZV DNA in vesicular fluid or CSF (in case of CNS infection "encephalitis")

Treatment	**Acyclovir** (antiviral) for the mother and the fetus	

Prevention	• **Pre exposure: varicella vaccine:** live-attenuated vaccines "at 1 year + pre-school"
	• **Post exposure: VZIG** (Varicella zoster **immune globulin**). who needs to take **VZIG**?
	  ○ Susceptible (non immune) **pregnant** women who have been **exposed** to VZV.
	  ○ Infants whose mothers get infected by VZV < 5 before to 2 days after delivery.	

4. Rubella Virus

Morphology

- **Family:** Togaviridae
- **Structure:** ssRNA, Icosahedral capsid, Enveloped Virus

Epidemiology & Pathogenesis
Picture

- Humans are the **only** reservoir
- **Transmission:**
  - Respiratory route
  - Transplacental route
- A world wide distribution, but now decreased due to vaccination.

Manifestations

في هذي المحاضرة أغلب الأعراض تتراوح ما بين عادية إلى شديدة

Acquired infection

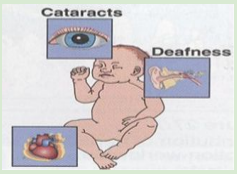
- Maculopapular rash (Rubella=German measles الحصبة الالمانية)

ما نقدر نعزل الفايروس من هذا النوع من أنواع الطفح الجلدي، ترجمتها طفح بقعي " البقعة ما نقدر نطلع منها شيء لأن من الأساس ما فيها سائل "

Congenital infection

Risk of acquiring congenital rubella infection varies and depends on gestational age of the fetus at the time of maternal infection, Ranging from normal ~ to congenital rubella syndrome (CRS) ~ to intrauterine death (IUD).

gestational age	risk to fetus
0-12 wks	70-80%
13-16 wks	20%
>16 wks	Infrequent



Congenital Rubella Syndrome:
Triad of abnormalities Affecting Ears,eye & heart:

- **Ears:** Sensorineural hearing loss. most common manifestation, can be unilateral or bilateral, present at birth but usually detected later
- **Eyes:** Cataracts, glaucoma, some time blindness
- **Heart:** Cardiac malformations (patent ductus arteriosus)
- **Others: Neurologic defects**, growth retardation, bone disease, hepatosplenomegaly, thrombocytopenia (result in rash called "blueberry muffin" lesions)

[11] Fetal blood sampling (FBS) (cordocentesis) is the collecting of fetal blood directly from the umbilical cord or fetus.

Diagnosis	**Pregnant mother**	**Serological diagnosis:** Rubella **specific IgM** or **IgG seroconversion**

	Infant	**Prenatal Dx:**
		• Ultrasound (to detect signs of the congenital anomalies)
		• Culture (from amniotic fluid)
		• PCR (from amniotic fluid, fetal blood)
		Postnatal Dx:
		• Serology:
		  ○ Rubella specific IgM but the absence of IgM does not exclude the infection.
		  ○ Persistent IgG in the infant's serum beyond 9-12 ms of age
		• Culture
		• PCR

Treatment	No antiviral available *مافيه علاج محدد إلى الآن*	

Prevention	• **Routine antenatal screening:**
	All pregnant ladies should do Routine antenatal screening to detect Rubella specific Ig**G**.
	Note that we are looking for IgG indicating immunity. Not IgM which would indicate infection.
	بدايةً من البوكينق بلود نشوف عندها الأجسام المضادة الخاصة بالروبلا : موجودة؟ الحمدلله ماعليها ضرر بس نراقبها كمان مو موجودة؟ هنا نحرص عليها وما نخليها تقرب من أي شخص مصاب ونراقبها
	• **vaccination :**
	  ○ Usually given with MMRV " (Measles, Mumps, Rubella & Varicella) Vaccine "
	  ○ Non immunised **women** should take the vaccine before or after pregnancy but not during pregnancy, bc it's live attenuated vaccine should not given to the immunocompromised.
	  ○ women who got vaccine should avoid pregnancy for **3 months.**	

5. Cytomegalovirus (CMV)		
Characteristic	• **Family:**Herpesviridae • **Structure:** dsDNA, Enveloped, Icosahedral Virus After the initial exposure to the virus → the virus Establishes in latent form → When there is diminished immunity, the virus may reactivate and cause recurrent infection.	
Epidemiology	Humans are the reservoir, worldwide distribution.	
Transmission (tn)	**Horizontal transmission**	• **Young children:** saliva by shared cups and spoons • **Later in life:** sexual contact, Blood transfusion & organ transplant
	Vertical transmission	Vertical transmission is the transmission *from mother to baby,* can be: 1) transplacentally (in utero/congenital), 2) during delivery (prenatal) 3) by breastfeeding (postnatal/neonatal) • 40% transmission in **primary CMV infection.** • Only 1% transmission in **Recurrent CMV infection** due to presence of maternal antibodies

<table>
<tr><td>Manifestations
  </td><td colspan="2">The majority of cases are <u>asymptomatic</u> at birth, BUT 15% of cases may develop <u>Hearing defect and mental retardation</u>, 4% <u>Cytomegalic inclusion disease</u>* & 1% may die (abortion).

Cytomegalic Inclusion Disease is characterised by:
CNS abnormalities: microcephaly,Ventriculomegaly, and periventricular calcification (like toxoplasma, however CMV causes specifically periventricular calcification).
Eye: chorioretinitis
Ear: sensorineural deafness
Liver: Hepatosplenomegaly (HSM) and jaundice.
Lung : pneumonitis
Heart: myocarditis
Thrombocytopenic purpura (“blueberry muffin” spots)</td></tr>
<tr><td rowspan="2">Diagnosis</td><td>Pregnant mother</td><td>Maternal serology: remember how to differentiate between 1ry & recurrent... CMV IgM, IgG, IgG avidity (low: primary infection, high: reactivation)</td></tr>
<tr><td>Infant</td><td>Prenatal: PCR, culture (from the amniotic fluid, takes time thus mostly PCR is used), CMV specific IgM, Ultrasound (to detect any anomalies)

Postnatal:
Isolation of CMV or detection of its genome: in first 3 wks of life From Body fluids: urine, saliva, blood, after 4 weeks doesn't mean congenital infection, because it is possible to acquire the infection during delivery or postnatally. By using: Standard tube culture method, Shell vial assay (modified cell culture) or PCR). PCR is very rapid and sensitive but it is not available in all hospitals.
Histology: Detection of Cytomegalic intranuclear Inclusion Bodies in affected tissue (<u>owl’s eye</u>)
Serology: CMV IgM</td></tr>
<tr><td>Treatment</td><td colspan="2">Symptomatic infants: Ganciclovir for at least 6 weeks
Asymptomatic infants: not recommended, because of the sides effect of Ganciclovir</td></tr>
<tr><td>Prevention</td><td colspan="2">Education about CMV & how to prevent it through hygiene and handwashing
Vaccine is not available (TRIAL)</td></tr>
</table>

The doctor asked these questions in the end: very imp.

1. The organisms that are transported through the placenta? TORC
2. what is toxoplasma and what are the congenital infections caused by it?
3. what is parvovirus and what does it cause?
4. what does the Varicella Zoster Virus cause?
5. what are the congenital triad for rubella?
6. what are the most important manifestations caused by CMV?
7. Which of these infections can be prevented "by vaccine"? rubella + VZV
8. what is the infection that the human is not the reservoir? toxoplasma

EXTRA SUMMARIES. Here

L2: SUMMARY OF TRANSPLACENTAL INFECTIONS

Notes:
All are mostly asymptomatic, All viruses have Icosahedral capsid, toxoplasma is zoonotic,
In neonates, Serology by detecting IgM (-ve doesn't exclude), or persistent IgG > 12 months
In transmission, we mentioned routes other than transplacental route (for all)
Common Findings: Chorioretinitis, **growth & mental** retardation, Hepatosplenomegaly, Thrombocytopenia, Microcephaly, risk of intrauterine death(highest in 1st trimester/primary inf.)

	Toxoplasma Gondii	Parvovirus B19	Varicella Zoster Virus (VZV)	Rubella Virus	Cytomegalovirus (CMV)
Morphology	intracellular parasite	Parvoviridae, **non-enveloped**, **ssDNA**.	Herpesviridae dsDNA, Enveloped,	Togaviridae ssRNA, Enveloped	Herpesviridae dsDNA, Enveloped
Route	Ingestion of cyst/ oocyst, Blood	Respiratory Blood	Respiratory	Respiratory	Saliva, sexual, Blood, & Vertically
Congenital inf.	**The classic triad of symptoms:** Chorioretinitis, **Hydro**cephalus & Intracranial calcifications	Hydrops fetalis (**anaemia, CHF, oedema & fetal death)**	Scarring of skin Hypoplasia of limbs CNS & eye defects	Deafness, Cataracts, glaucoma, patent ductus arteriosus, **CNS**, "blueberry muffin" lesions	Ventriculomegaly, **periventricular** calcification, deafness,pneumonitis, myocarditis, "blueberry muffin"
Acquired inf.		Erythema infectiosum	Varicella (Chickenpox) Zoster (Shingle)	Maculopapular rash (German measles)	
Maternal investi.	IgM, IgG, IgG avidity, and IgG seroconversion	IgM, IgG seroconversion.	IgM, Culture (vesicular fluid), IF (Ag in cells), PCR	**IgM, IgG seroconversion**	IgM, IgG, IgG avidity
Prenatal	PCR, Culture or US	PCR, US	PCR, US, IgM (fetal blood)	PCR , Culture or US	**PCR**, culture, US, Ig**M**,
Postnatal	IgM, IgG , PCR, Culture,Evaluation		IgM , Culture, PCR	IgM, IgG, PCR, Culture	**PCR**, culture, histo (owl's eye), Ig**M**,
Treatment	Spiramycin **Pyrimethamine + sulfadiazine.**	Intrauterine blood transfusion	**Acyclovir**		Ganciclovir only if symptomatic
Prevention	Preventive measures	Preventive measures	**Preexposure:** live-attenuated vaccines **Postexposure: Ig for pregnants,** Infants	**screening for** IgG. **vaccination :** for Non immunised **women +** avoid pregnancy for **3 months.**	Preventive measures

MCQs

1- Majority of congenital infections are asymptomatic at birth :

a) True
b) False

2- what is the route of transmission of Toxoplasma gondii:

a) Ingestion of oocyst
b) Ingestion of cyst in undercooked meat.
c) Blood transfusion and organ transplant
d) All

3- congenital infection could be because of reactivation of a latent infection:

a) True
b) False

4- To prevent Toxoplasma Gondii congenital infection you should:

a) Stay home when ill
b) share personal items
c) Avoid exposure to cat feces
d) Avoid touching your eyes

5- Risk of congenital infection with Parvovirus B19 is greatest when infection occur in:

a) 1st trimester
b) 2nd trimester
c) 3rd trimester

6- Congenital Rubella Syndrome abnormalities :

a) Sensorineural hearing loss
b) Cataracts and glaucoma
c) Renal failure
d) A+b

7- Intranuclear inclusion bodies [Owl's -eye] is seen in:

a) CMV
b) Rubella virus
c) HIV
d) All

Ans: a, d, a, c, a, d, a