TEAM 443
MICROBIOLOGY

L1- Transplacental infections

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OBJECTIVES

1. **To recognize the different types of infant infections.**
2. **To know the major transplacentally transmitted pathogens causing congenital infections.**
3. **(Toxoplasmas, TP, parvovirus B19, VZV, Rubella V & CMV).**
4. **To describe their structures .**
5. **To know their major epidemiology features.**
6. **To describe clinical manifestations of their congenital infections.**
7. **To illustrate different laboratory diagnosis of maternal and congenital infections.**
8. **To know their treatment and preventive measures.**

Introduction

Terminology & Routes of transmission (2) (5)

Classification (On time of infection)	Timing of events	Mechanisms
Congenital	**Intra-uterine** (In utero)	❖Transplacental ❖Ascending infection 
Perinatal	**Intra-partum** (During labour & delivery)	❖Exposure to genital secretions and blood ❖Contact with infected material during delivery, faeces 
Neonatal	**Post-partum** (After birth)	❖Direct contact ❖Blood transfusion ❖Breastfeeding ❖Nosocomial exposure 

Congenital infections (3) (4)

❖Mostly due to viruses.
❖Previously known as (**TORCH**) infections: (1)
- ➢**T**= **T**oxoplasma gondii
- ➢**O**= **O**ther e.g. (Syphilis, parvovirus B19 & VZV)
- ➢**R**= **R**ubella virus *
- ➢**C**= **C**MV
- ➢**H**= **H**erpes, **H**PV, **H**BV, **H**CV & **H**IV

- **Primary maternal infection in the first half of pregnancy poses the greatest risk to the fetus.**

Important information for any congenital infection (1)

- ❖ 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.

Risk of IUI & fetal damage depends on:

- Type of Organism (Teratogenicity)
- Type of maternal Infection (Primary,recurrent)
- Time of infection during pregnancy (1st, 2nd, 3rd Trimester)

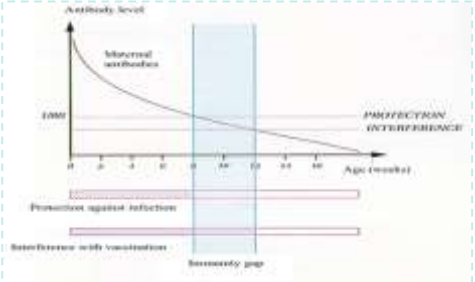
Common features of Congenital infections (5)

❖**Intrauterine growth retardation (IUGR)** small baby
❖**Microcephaly** Or hydrocephalus
❖**Hepatosplenomegaly (HSM)**
❖**Skin rash**
❖**Jaundice**
❖Generalized Lymphadenopathy
❖**Thrombocytopenia** may result in rash. There is also certain organ damage (eye, ear & the heart)
❖IgM, Persistent IgG
❖Fever

- **Majority of Congenital infections are asymptomatic at birth** , however some develop mental retardation and hearing loss later in life Preventative and therapeutic measures are possible for **some** of the agents.

Neonatal Serological Diagnosis

- IgM antibody (4)
- Absence of fetal IgM at birth doesn't exclude infection.(3)
- **Persistence** of specific **IgG** antibody **> 12 months** of age. Because the transplacentally acquired maternal IgG persist with infant and decrease within few months after birth. So persistence of IgG indicate the IgG is produced from the baby.



Toxoplasmosis (6)

Keywords: - Cat/undercooked meat - Intracranial calcifications - Chorioretinitis - Hydrocephalus - IgG avidity

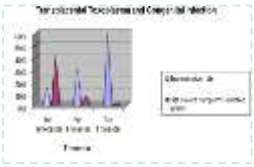
Clinical Features

- **Causative organism: Toxoplasma gondii**
- Obligate intracellular parasite.
- Definitive host is the domestic cat

Epidemiology

Prof.Ali: very important to remember

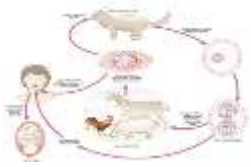
- European countries (ie France, Greece)
- Usually asymptomatic
- Most cases are due to primary maternal infection.
- Rarely, reactivation of a latent infection
- **Infection (Transmission) rate higher with infection in 3rd trimester**[2]
- **Fetal death higher with infection in 1st trimester**[3]
- Primary maternal infection in pregnancy



if the infection occurred in the first trimester, it will most likely lead to fetal death.
However, fetus had a better prognosis if the infection occurred in the 3rd trimester (which is fortunately more common)

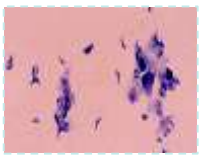
Life Cycle & Transmission

- Ingestion of **oocysts**: **cat feces contaminate fingers, soil, water**.
- Ingestion of **cysts** in **undercooked meats, garden products**
- **Blood transfusion** and **organ transplant**
- Vertical transmission: from mother to fetus (transplacental)



Morphology (3 forms)

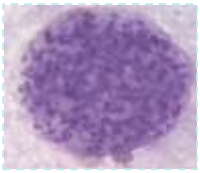
Oocysts
Shed in **CAT feces**
definitive host



Tachyzoites
- Rapidly dividing forms
- **Acute phase**

Immunity +

Immunity -



Bradyzoites
- Slowly dividing forms
- **Chronic phase**

Clinical Presentation

- Mostly (70-90%) are **asymptomatic at birth**, but are still at high risk of developing abnormalities, especially eye (chorioretinitis)/neurologic disease(MR) later.
  It's almost asymptotic in everyone EXCEPT pregnant women it's dangerous
- **Classic triad of symptoms:**
  - **A- Chorioretinitis** (inflammation of the choroid & retina)
  - **B- Intracranial calcifications**
  - **C- Hydrocephalus** (accumulation of fluid in the head)
- **Other symptoms include:**
  Fever, rash, HSM, **microcephaly**,seizures, jaundice, thrombocytopenia, lymphadenopathy
- Abortion & Intrauterine death. (IUD)

Diagnosis (6) (7)

Pregnant mother

1- Maternal serology: (7) (8)
- **IgM** /IgA
- IgG
- **IgG avidity**
- IgG seroconversion compared to booking blood.(8)(9)

Infant (9)

Prenatal:
1- Serial Ultrasound 2- PCR 3- Culture.

Postnatal/Newborn:
1- Serology I
- IgM
- High IgG **or** persistently +ve >12 months

2- PCR
3- Culture
4- Evaluation of infant e.g.neuroimaging

Treatment

- Spiramycin For the mother to decrease transmission
- Pyrimethamine and sulfadiazine after birth for one year

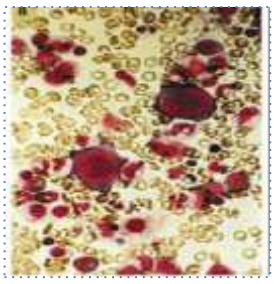
Prevention

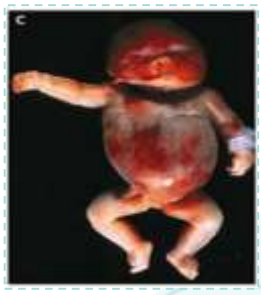
1- Avoid: Exposure to cat feces and contaminated food and water.
2- Wash: - Hands with soap and water. - Fruits/vegetables - Surfaces that touched fruits/vegetables/raw meat.
3- Cook: all meats thoroughly.

Parvovirus B19

Keywords:
- Hydrops fetalis
- Anemia, rash
- Slapped cheek (المعنى أنه الخدود تكون لونها احمر)

General (14) information	❖ **Family:** Parvoviridae ❖ Non-enveloped V, Icosahedral capsid and **S.S DNA** genome ❖ Causative agent of Fifth disease(1) (erythema infectiosum)	
Epidemiology	❖ Worldwide distribution, Most of the population is eventually infected. ❖ Half of women of childbearing age are susceptible to infection. ❖ Humans are known hosts ❖ No evidence of teratogenicity , Risk of **fetal death highest** during 2nd trimester	
Transmission (15)	❖ **Respiratory route** ❖ **Blood transfusion** ❖ **Transplacental route**	
Clinical presentation	**Acquired infection**	**Congenital infection**
	Immunocompetent host • **Erythema infectiosum (Rash)** Usually self-limiting Immuno-compromised host	• Risk of congenital infection is greatest when infection occur in 1st 20 weeks (1st and 2nd trimesters): 1. **Infection in 1st trimester** → IUD (Intrauterine death) 2. **Infection in 2nd trimester** → **HF (Hydrops fetalis)** Massive edema 3. **Infection in 3rd trimester** → Lowest risk (16) • Causes fetal loss through hydrops fetalis, **severe anaemia,** Congestive heart failure CHF, generalized edema and fetal death. Hemolysis of RBCs → Anaemia → CHD → HF
Diagnosis	**Pregnant mother**	**Prenatal (19)**
	- **Specific IgM (17) (18)** - IgG seroconversion - Persistent IgG	- **Ultrasound (hydrops)** - **Not grow in cell culture** - PCR
Treatment	**Intrauterine blood transfusions** (blood transfusion transplacentally) Administration of digoxin to the fetus	
Prevention	• Hygiene practice • No vaccine (TRIAL)	





Hydrops fetalis

erythema infectiosum

Varicella Zoster Virus

Keywords:
- **Vesicular rash, VZIG**
- **Chickenpox, Shingle**

General information (21)

- **Family:** Herpesviridae which will cause latent infection (22)
- dsDNA , Enveloped, Icosahedral Virus
- Chicken pox,or shingle is an Apha-herpesvirus
- 90% of pregnant women **already immune**
- Primary infection during pregnancy carries a greater risk of severe disease

Transmission

-Respiratory route.
-Transplacental route.

Clinical presentation

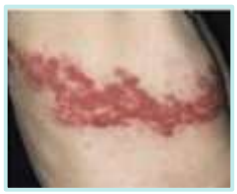
Acquired infection

Varicella (Chickenpox): (10)
عنقز

- Primary illness
- Generalized vesicular rash
- Common in children.
- Itching

Zoster (shingles):
الحزام الناري

- Recurrent infection
- **Localized painful unilateral vesicular rash**



Continental infection

Primary infection carries a greater risk (for both mother and fetus) of severe disease, in particular pneumonia.

1-Intrauterine infections (Majority asymptomatic)**:**
Congenital varicella syndrome(CVS):
-1st 20 weeks of Pregnancy
-The incidence of CVS is ~ 2%
-Up to 3% chance of transmission to fetus, recognised congenital varicella syndrome/ **Symptoms:**
-Scarring of skin
-Hypoplasia of limbs
-CNS & Eye defects e.g retinitis

2-Neonatal varicella(20): (Occurs during delivery)
-Less than 5 days before delivery → severe disease encephalitis and pneumonia in neonatal period
-More than 5 days before delivery → mild disease
why? Because of mother Ab

Diagnosis (23)

Pregnant mother (26)

Direct from the vesicles:
- cells scraping from the base of vesicles: ImmunoFluorescent(Ag)
- DNA-VZV by **PCR**

Serological:
- IgM AB

Infant

Prenatal:
- Ultrasound and MRI.
- VZV DNA in fetal blood or amniotic fluid or placenta villi (usually the sample is amniotic fluid) by PCR

Postnatal/Newborn:
- VZV IgM
- Virus isolation (culture)
- VZV DNA in VF or CSF (CSF infection) by PCR

Prevention (24)

- **Pre-exposure:** Varicella live-attenuated vaccines before or after pregnancy but not during pregnancy.
- **Post-exposure :** VZIG (Varicella zoster immunoglobulin)(25): Susceptible pregnant women(not immune either by previous infection or by not taking the vaccine) have been exposed to VZV. Not for pregnant women who is infected with vzv. they should be treated with acyclovir
infants whose mothers develop Varicella during the last 5 days of pregnancy or the first 2 days after delivery and premature baby <28 weeks of gestation

Treatment

Acyclovir at first sign of varicella pneumonia for pregnant women and neonates

Rubella Virus

Keywords:
- Routine antenatal screening
- Maculopapular rash
- Blueberry muffin
- Cardiac malformations (PDA)

General information (27)

- **Family:** Togaviridae
- SS RNA genome, Icosahedral capsid, Enveloped Virus
- Rubella = German measles الحصبة

Epidemiology

- **Vaccine-preventable disease in human** → No longer considered endemic
- Mild, self-limiting illness
- Transmission can occur with primary infection or reactivation of virus but 40% of transmission in primary infection.
- Increased risk of transmission later in pregnancy but more severe complication associated with earlier acquisition.

Transmission

- Respiratory route.
- Transplacental route

Clinical Presentation

Acquired infection

- Ex. Maculopapular rash

Congenital infection

- Normal → Congenital rubella syndrome (CRS) → IUD
- Risk of acquiring congenital rubella infection varies and depends on gestational age of the fetus at the time of maternal infection:

0-12 weeks → 70%
13-16 weeks → 20%
>16 weeks → infrequent

- **Congenital rubella syndrome:** Triad of abnormalities affect → Ears, Eyes and Heart.
- Eyes: **Cataracts**(most common) **and glaucoma , "Salt and pepper" retinopathy.**
- **Heart: Cardiac malformations** e.g.Pulmonary artery stenosis (PAS), Patent ductus arteriosus(PDA) ***
- Skin: Skin rash.
- Neurologic defects (Less common)
- Others: Growth retardation, Bone disease, HSM, Thrombocytopenia, **"blueberry muffin" lesions/rash**

Diagnosis (28)

Pregnant mother

- Viral isolation virus from nasal secretions, throat, blood, urine, CSF.
- Serological diagnosis:

1- Rubella specific IgM

2- IgG seroconversion (Maternal IgG is useless!)

Infant

Postnatal/Newborn:
1– Serology:
- IgM : recent postnatal or congenital infection.
-Persistence of IgG >9-12 months (Rising monthly IgG titers suggest congenital infection)
2- Culture
3- PCR

Prenatal:
1- Ultrasound e.g heart lesions
2-Culture
3- PCR

Prevention (29)

- **Rubella vaccine (Live attenuated vaccine)** for non immune people (12)
- Routine Antenatal screening(11): Rubella Specific IgG

+ve igG (no risk of congenital anomalies to the baby and she is immune)
-ve igG (at risk and they should take the live attenuated vaccine but after delivery)

- Non-immune women → vaccination (avoid pregnancy for 1-3 months) .

Cytomegalovirus (CMV)

Keywords: from 441
- **Owl's eye, intranuclear inclusion bodies**
- **Urine**
- **Deafness**

General information (30)

- **Family:** Herpesviridae has the ability to cause latent infection
- dsDNA , Enveloped ,Icosahedral Virus.
- Establishes in latent form → reactivation → Recurrent infection.
- Most common congenital viral infection~40,000 infants per year.
- Mild, self limiting illness
- ★ Most common congenital infection

Epidemiology

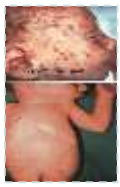
- Human ,worldwide.

Transmission

Horizontal transmission	Vertical transmission
Young children: saliva (in day care) Later in life: sexual contact , Blood transfusion & organ transplant	• Primary CMV infection (40%) • Recurrent CMV infection (~1%)

Clinical Presentation

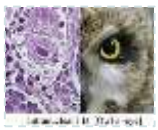
- **Clinically normal (80%)** / **asymptomatic at birth (90%)**
- **develop symptoms later** (Hearing defect and mental retardation) **(15%)** :
  -CNS abnormalities → **microcephaly**, periventricular calcification (31), neurological deficits.
  -Eye → chorioretinitis, Vision Impairment
  -Ear → >80% develop long term complications: sensorineural deafness.(Most common)
  -develop delay
  -Liver → HSM and jaundice.
  -Lung → pneumonitis.
  -Thrombocytopenic purpura (petechiae)



- **Cytomegalic inclusion disease (4%)** small baby due to intrauterine growth retardation
- **Death (1%)**

Diagnosis (34)

Pregnant mother	Infant
Serological diagnosis: (same as Toxoplasmosis) **1-** CMV IgM **2-** CMV IgG (shows only past infection) **3-** CMV IgG avidity	**Postnatal:** **1-** By isolating CMV or detection of its genome in first 3 (why ? To differentiate between congenital,prenatal or postnatal infection) weeks of life. 2-Viral load and DNA copies can be assessed by PCR in urine. Body fluid(Sample) : Urine (32), saliva, blood. Using: - Standard cell culture method - Shell vial assay - PCR **2-** Histology; Detection of Cytomegalic **intranuclear Inclusion Bodies in affected tissue (Owl's eye).** **3-** Serology; CMV IgM / serologies not helpful given high antibody in population. **Prenatal:** 1- Ultrasound **2- Culture** **3- PCR**



Prevention

No vaccine, Education about CMV & how to prevent it through hygiene; hand washing

Treatment

Symptomatic infants → Ganciclovir (x6 weeks) To improve neurological development

Male Slides

Syphilis & Herpes simplex

Herpes simplex (36)

General information	❖ HSV1 (in oral cavity causing Herpes labialis) ❖ HSV2 (genital infections) ❖ Is an enveloped double stranded DNA virus.
Epidemiology	❖ Primarily transmitted through infected maternal genital tract ❖ Primary infection with greater transmission risk than reactivation ❖ Indications for **C-section delivery** prior to membrane rupture
Clinical presentation (37)	❖ 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. ❖ Molecular PCR viral DNA detection in blood or Urine
Diagnosis	❖ Culture of maternal lesions if present at delivery ❖ Cultures in infant ❖ CSF PCR (Mainly) ❖ Serologies is useless
Treatment (38)	❖ Maximum dose of acyclovir

❖ **Pregnant lady having simplex for the first time (primary) → C section is indicated**

Syphilis (10)

General information	❖ Treponema pallidum (spirochete) ❖ Transmitted via sexual contact[3] ❖ Mother with primary or secondary syphilis[4] ❖ Typically occurs during second half of pregnancy ❖ Intrauterine death in 25%
Clinical presentation	**3 major classifications**: ❖ Late abortion or stillbirth ❖ **Infantile**: - **Anal Rash/ulcer** and Funisitis (umbilical cord vasculitis) - Osteochondritis, periostitis, liver and lung fibrosis ❖ Childhood: - Interstitial keratitis, Hutchinson teeth, 8th nerve nerve deafness - Frontal bossing, Short maxilla, High palatal arch, Saddle nose and Perioral fissures (but these presentation not specific only for congenital syphilis)
Diagnosis (11) (12)	❖ **RPR** (rapid plasma reagin)/**VDRL** (Venereal disease research laboratory test, it's a screening test): non-treponemal test [6] ❖ **MHA-TP** (microhemagglutination assay–Treponema pallidum) /**FTA-ABS** (fluorescent treponemal antibody test absorption test): specific treponemal test ❖ Confirmed if T. pallidum identified in skin lesions, placenta, umbilical cord, or at autopsy
Treatment	❖ **Penicillin G**
Prevention (13)	❖ **RPR/VDRL screening** in ALL pregnant women early in pregnancy and at time of birth

❖ Prof: if you see a lesion like this in ER immediately start the maximum dose of acyclovir and do PCR. If negative, you stop the acyclovir.
❖ Only mother with primary or secondary syphilis not tertiary can thair baby develop congenital syphilis
❖ Baby present with ulcer around the anus (distinctive characteristic for syphilis)

Prof Ali's notes:

1- there are multiple factors that determine when we should worry about the baby if he is at high risk to be infected or at low risk:

A- The prevalence of the disease :
if the disease is common or not , like CMV is very common among people so they have been infected ,therefore they have immunity against it .

B- The time of exposure ; early pregnancy or late pregnancy

C-the immunity of the host (the mother):
If the mother was infected at the first time in pregnancy there will be adverse effects on the baby in contrast to if she was immunized and already exposed nothing will happen to the baby.

2- general info(overview):
-some infections if they occurred early in the pregnancy they will be transmitted rapidly
- in early pregnancy no problem, in late pregnancy there is complications
-it is not necessary to do screening to everythings , cuz even if we did and the result was positive there is nothing we can do to help the patient .

3- we screen for syphilis, toxoplasmosis , hepatitis B, HIV, Rebulla.

4- CMV, parvovirus , VZV—> we don't screen.

5- in congenital infections there are symptoms that are common most of the time—> fever , rash, organomegaly.

6- toxoplasmosis:
- prevalence depends on where you live (in KSA—> high prevalence).
- The only infection that is high in 3rd trimester , but in case it occurred at early trimester it will cause fetal death immediately .

7- IgM —> stay for one year

8- in toxoplasmosis virus we do not depend on IgM only, because it's stay for one or two years , for instance ,she is five months pregnant and the IgM is present but maybe she has been exposed before she get pregnant , so because of this we must do IgG and other test.

9- in Newborn—> it is rare to do these tests , instead if there were abortion or the baby was very sick.

10- syphilis :
- has phases => - primary —> 3 weeks after exposure
  - secondary —> 3 months
  - tertiary
- if she was infected by primary and secondary in first 20 weeks of pregnancy , the baby will be highly exposed to syphilis.

11- Diagnosis of syphilis :
non treponemal test => Is a screening test,highly sensitive but not specific, cuz sometimes the test will be positive in a pregnant leady who took the vaccine (not infected) ; so we have to confirms it by treponemal test.

Prof Ali's notes:

12- (dr said that he will not ask about this, but me I am telling you have to study it :) :
In some cases that we suspect that the baby has been infected by syphilis once he was born, so we test and find the following:

-mother => positive

-baby=> RPR positive (non treponemal test)

But we have to know that IgG can cross the placenta from the mother to the baby, in this case we have to wait days , if the IgG level decreased then it was from the mother , if it was still high we know that the baby has been infected and give penicillin G.

13- we do screening after pregnancy

14-parvovirus B19:

- it is transplacental but not congenital
- If the pregnant woman was primary exposed to this virus in first 20 weeks —> it will be very severe.

15- the pathogenesis of parvovirus B19 : it can go to the bone marrow of the mother and cause destruction to RBC, in case of sickle cell disease patient —> can lead to hemolytic anemia.

The same thing occur to the baby , go to bone marrow —> destruction of the RBC—> anemia —> heart failure.

16- if the infection occurred at late pregnancy nothing will happen(no symptoms)

17- any virus we look for IgM , except in syphilis(it is a bacteria -bacterium treponema pallium- ,not virus)

18- IgM cannot cross the placenta , if we found IgM in baby's blood it is from the baby himself.

19- if we found IgG in baby's blood , how do we know if it is from the mother or from the baby? we repeat the blood test again after two weeks=>

- still at high level(persistence IgG)—> from the baby
- returned to low level —> from the mother

20- neonatal varicella (هو ما يعرف بالعنقز):
This type of infection normally we don't worry about unless the patient was—> immuncompersed, pregnant , chemotherapy or organ transplant.

21- why do we fear varicella:

A- it is highly infectious

B- we have to do a lot in order to protect the baby

C- long incubation period —>symptoms will take 2-3 days to appear, for example: baby came to ER with fever only after 3 days he started to suffer from rash.

22- it is a type of herpes simplex virus.

Prof Ali's notes:

23- the diagnosis is a little bit hard: if we have a sample from vesicles it will be easy, no vesicles , we mainly depend on serology IgM and IgG.
24- An example (you can consider it as a story):
Let's imagine that you are married and you are waiting for your first child, one day you and your wife went to the hospital for checkup and you found that your wife ,her immunity against vericila is negative(did not take the vaccine and has nerve been infected before), what are going to do? We have two options:

- Number one=> not to be exposed ; be careful not to visit people
- Number two=> depends when we give her the vaccine ,in early pregnancy or in late pregnancy, or give her immunoglobulin.
- Number three=>Treatment : we start it once we confirm the infection.

25- immunoglobulin can not cross the placenta.
26- pregnant women will have pneumonitis and hepatitis.
27- Rubella (German measles):

- we must do screening
- Very fatal disease
- Early pregnancy the risk is high, late pregnancy the risk is low.
- Can cause rash hemorrhagic

28- Diagnosis => in Rubella we don't do culture , we do serological test.
29- no treatment for Rubella.
30- CMN:

- very common disease and we have to do screening for all babies
- The most common cause of sensorineural hearing loss
- it is preventable
- Late pregnancy transmission
- This virus is like HIV, EPV, hepatitis which are all secreted from body fluids like: saliva (by kissing) and genitalia, can be transmitted easily.
- This virus most of the time affect the second baby; cuz the first child transmitted to the mother from daycare.

31-the difference between calcification in brain in toxoplasmosis and in CMV:

- toxoplasmosis=> the calcification in all the brain
- CMV=> only periventricle (lateral ventricles)

32- when we diagnose the baby we find that the virus has been secreted in large amount in the urine , we can obtain it from blood but from urine is much more easy .
34- diagnosis :

A- viral isolation
B- urine
C- blood
D-PCR for CMV in urine (used most of the time)

35- in the past we used to urine in culture.

Dr's note:

36- herpes simplex virus (HSV):
- is primary infection
- no serological test, not very helpful , but sometimes we do it for epidemiological reason.
- In diagnosis we mainly use PCR for the two types : HSV1 and HSV2.

37- has wide range of complications:
- appearance of viscles
- Sepsis
- Hypotensive

38- in case we have an infected husband and a non infected pregnant wife, how can we protect the pregnant wife?
by giving suppression treatment => famciclovir => transmission is low.

Dr Malak's notes:

1- TORCH:
A- TORC=> all by transplacental
B- H=> all by prenatal.

3- negative IgM result does not exclude the diagnosis ; the baby is unable to develop antibodies before 20 weeks.

4- presence of IgM indicate acute infection.

5- very imp note:
the risk of intrauterine transmission increases with gestational age of the fetus => highest risk of transmission is in 3rd trimester
however, the risk of fetal damage in first trimester due to cell division and organs being developed.

6- it is imp to differentiate between primary and recurrent menstrual infection , why?? due to the risk of fetal damage with primary.

7- maternal (the mother):
- igM positive and IgG negative=> primary infection
- IgM positive and IgG positive=> we can not differentiate , so we have to do IgG avidity test
- IgG avidity test : low level=> primary , high level=> recurrent

8- booking blood mean→ the first blood obtained from pregnant woman.

9- seroconversion means→ it was negative then turn to be positive.

10- after recovery the virus become latent in dorsal root ganglia , so when immunosuppression occurs this leads to reactivation .

11- we should do screening for all pregnant ladies m for what?? detection og IgG which indicates immunity.

12- MMRV→ Measles- Mumps-Rubella-Varicella.

Summary

Infection	Congenital infection/ Clinical sign	Infant U/S to look for:	Vaccine	Treatment
Toxoplasma Gondii	- Chorioretinitis - Hydrocephalus - intracranial calcification	- Hydrocephalus - Intracranial calcification	-	-Spiramycin -Pyrimethamine & Sulfadiazine
Parvovirus	Hydrops fetalis(2nd trimester)--> Anemia + Edema	Hydrops		
Congenital varicella syndrome (1st trimester)& Neonatal varicella (around delivery)	Scarring of skin , Hypoplasia of limbs	Hypoplasia of limbs	Pre exposure: varicella vaccine(LAV) Post exposure: VZIG	Acyclovir
Congenital Rubella Syndrome	-Sensorineural hearing loss -Cataracts and glaucoma -Cardiac malformations	Heart defect	-Rubella vaccine(LAV) (Given after delivery -life attenuated)	No treatment (but there is routine antenatal screening)
Cytomegalic Inclusion Disease	Microcephaly + calcification , then mental retardation & deafness	Microcephaly + intracranial calcification	-	Symptomatic infants: Ganciclovir

Special thanks to Rafan Ahazani for writing this summary

MCQs - SAQ

Q1 - **Which one of the following can cause cataract , pda and cardiac malformations?**			
A- varicella zoster	B- chlamydia	C- HSV	D- Rubella
Q2 - **Which one of the following is the best test for postnatal infant for CMV?**			
A- ultrasound	B- serology of CMV igM	C- isolating CMV by body fluid sample	D- spiramycin
Q3 - **What is the treatment of CMV?**			
A- Ganciclovir	B- penicillin	C- acyclovir	D- spiramycin
Q4 - **Cyst of toxoplasma gondii found in?**			
A- Feces of infected cat	B- skeletal muscle of chronic patient	C- myocardium of chronic patient	D- both B&C
Q5 - **Spiramycin used to treat?**			
A- CMV	B- HSV	C- VZV	D- Toxoplasmosis
Q6 - **Transmission of toxoplasmosis is higher in?**			
A- 1st trimester	B- 2nd trimester	C- 3rd trimester	D- after birth

A 35 years old business woman went to the hospital suffering from vaginal discharge , after taking a sample to the laboratory the result shown ; gram negative diplococci

Q : what is the most likely diagnosis?

Answer: Neisseria gonorrheae

Answers: Q1:D Q2:C Q3:A Q4:D Q5:D Q6:C. Special thanks to 442.

TEAM 443
MICROBIOLOGY

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اللهم إني استودعتك ما قرأت وما حفظت فرده إليّ عند حاجتي إليه انك على كل شيء قدير

Any future corrections will be in the editing file, so please check it frequently