MICROBIOLOGY FINAL REVISION FILE

Vaginitis	normal	Bacterial vaginosis	Candidiasis	Trichomoniasis
Etiology	--	Unclear: Gardenella vaginalis mobiluncus, Mycoplasma hominis	Candida albicans or Glabrata	Trichomonas vaginalis
Vaginal PH	< 4.5	> 4.5	≤ 4.5	> 4.5
Vaginal discharge	Clear to white	Homogenous Fishy-smelling, thin, milky-white or gray	Thick white cottage cheese like	Yellow-green, frothy, malodorous smelling
Clinical presentation	--	Itching and burning	irritation and pruritis Painful sexual intercourse and urination	Pruritus, painful urination and sexual intercourse
Clinical finding	--	Vulva erythma & labia edema	Inflammation and erythma	Cervical petechise “strawberry cervix”
KOH”whiff” test	Negative	Positive	Negative	Often positive
wet mount preparation	Lactobacilli	Clue cells ≥ 20%, no/low WBCs	Few to many WBCs pseudohyphae and budding yeast in C.albicans, budding yeast without pseudohyphae in C.glabrata	Motile flagellated protozoa, many WBCs
Treatment	--	Metronidazole & tinidazole	Floconazole & itraconazole	Metronidazole

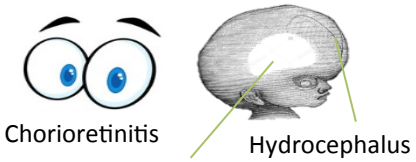
TOXOPLASMA GONDII

Transmission

- Cysts in raw meat
- Oocytes in animal feces
- Transplacental

❑ Highest transmission rate → 3rd trimester
❑ Highest fetal death rate → 1st trimester

Classic triad



Syphilis

(Treponema pallidum)

STD

Penicillin G

Congenital classification:
- **Late abortion**
- **Infantile** (rash & funisitis, osteochondritis, periostitis, liver & lung fibrosis)
- **Childhood** (interstitial keratitis, Hutchinson teeth, 8th nerve deafness, saddle nose,..)

OTHERS

Varicella

Available vaccine

- Chickenpox
- Shingles
- Limbs hypoplasia
- CNS & eye defects

1ry infection → greater risk of severe disease

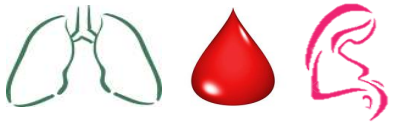
Acyclovir
for varicella pneumonia

Parvovirus P19

5th disease

Erythema infectiosum

Transmission



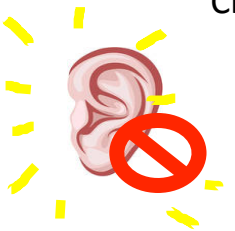
Highest fetal death → 2nd trimester
Minimal risk → 3rd trimester
Hydrops fetalis, severe anemia, CHF generalized edema

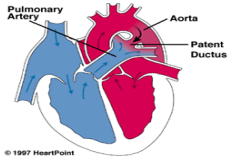
digoxin

RUBELLA

RNA enveloped virus
Spread by:
Respiratory droplets & transplacentally
- Available vaccine

Clinical features





- **Sensorineural hearing loss** (most common)
- "Salt & pepper" retinopathy
- Cataracts, glaucoma
- Patent ductus arteriosis
- HSM, thrombocytopenia
- "Blueberry muffin" lesions

TORCH

Transplacental Infections..

CYTOMEGALOVIRUS

Most common congenital viral infection!

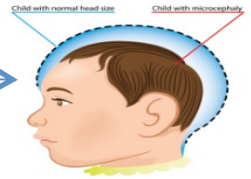
❑ Highest transmission rate → later in pregnancy
❑ Severe sequalae → earlier exposure (like toxoplasmosis)

ganciclovir

Clinical features

90% are asymptomatic

Microcephaly (not hydro-)
Periventricular calcification (not intracranial)
Neurological deficits
HSM, petichiae, jaundice, chorioretinitis



Complications: hearing loss, vision impairment

HERPES

Herpes simplex 1 or 2

Transmission

Maternal genital tract
1ry infection → greater risk of transmission than reactivation

Remember!

C-SECTION

Clinical features

Most are asymptomatic at birth
Symptoms between birth and 4wks: skin, eyes, mouth, CNS disease, disseminated disease (earlier)

Acyclovir

Done by: Lina Aljurf

Lab diagnosis of tranplacental Infections

TOXOPLASMA GONDI

Pregnant mother:
Serology: IgM,**IgG**,**IgG** (AVIDITY),**IgG** seroconversion (compare)
IgG X 3

Infant:
Prenatal:PCR,Culture, Serial U/S

Postnatal:
Serology:IgM,IgA,IgG
Persistently +ve >12 ms

PARVOVIRUS B19

Pregnant mother:
Specific IgM,IgG seroconversion
Prenatal DX:
Not grown in c/c ,
PCR, U/S to detect (hydrops)

VARICELLA ZOSTER VIRUS

Pregnant mother:
A-Direct:
Vesicular fluid for isolation, cell scraping from base of vesicle for IF (Ag) , DNA-VZV by PCR
B- serological test: IgM AB
Infant:
Prenatal: VZV DNA in FB or AF or placenta villi, VSV IgM in FB, U/S
Postnatal:
VSV IgM, virus isolation , VSV DNA in VF or CSF (CSF INF)

RUBELLA VIRUS

Pregnant mother:
Serological Dx:
Rubella specific IgM, IgG seroconversion
Infant:
Cell culture & RT-PCR: from AF, chorionic villi (fetus),or from nasal secretion , throat, urine, blood(newborn).
Serological Dx: Rubella specific IgM , persistent & rising titers of anti- rubella IgG in infant's serum beyond 9-12 months of age

CYTOMEGALO VIRUS

Maternal:
Serology:CMV IgM, CMV IgG, CMV IgG avidity

Prenatal: PCR,culture, CMV specific IgM , ultrasound
Postnatal:
1-isolatio of CMV& CMV DNA in first 3 wks of life from:
(urine , sliva, blood) BY:
A-standard tube culture method B-SHELLS vial assay C-PCR
2-Histology: detection of CMV inclusion bodies in affceted tissue (OWL's EYE)
3- Serology : CMV IgM

	CHLAMYDIA	GONORRHEA
	➢ obligate intracellular bacteria ➢ No rigid cell wall. ➢ Fail to grow on artificial media ➢ Uses host cell metabolism for growth &replication. ➢ Spread by genital secretions , anal or oral sex. ➢ Species: C. trachomatis (A,B,C) → Trachoma C.trachomatis (D,K) → Inclusion Conjunctivitis, Genital Infection C. trachomatis (L1,L2,L3) →Lymphogranuloma Veneruem C.psittaci →Psittacosis (By parrots) C.pneumoniae →Respiratory Infections	➢ A STD disease acquired by direct genital contact. ➢ It is localized to mucosal surfaces with infrequent spread to blood or deep tissues. ➢ Caused by Neisseria gonorrhoeae. ➢ A Gram negative diplococci grows on chocolate agar and on selective enriched media and CO_2 required. ➢ Not a normal flora. ➢ Invasion by IA and Opa proteins
SYMPTOMS	➢ Female Symtoms: cervicitis, salpingitis, urethral syndrome, endometritis & proctitis. ➢ Male Symptoms: urethritis epididymitis & proctitis. ➢ Most infants develop inclusion conjunctivitis, 5-10% develop infant pneumonia syndrome.	➢ **Pharyngitis, urethritis with discharge ,proctitis.** ➢ **Pelvic inflammatory disease: (women)** fever, lower abdominal pain, adnexal tenderness, leukocytosis ➢ **Disseminated gonococcal infection:** Fever. migratory arthralgia and arthritis. Purulent arthritis involving large joints. Petechial, maculopapular rash.
DIAGNOSIS	➢ (PCR) or (LCR): are the most sensitive methods of diagnosis. ➢ Isolation on tissue culture (McCoy cell line) ➢ iodine or Giemsa stained smear.	Culture on Thayer-Martin Isolates identified by sugar fermentation of glucose only Coagglutination test.
TREATMENT	**Azithromycin** single dose for non- LGV infection. **Erythromycin** for pregnant women. **Doxycycline** for LGV.	**Ceftriaxone IM** (or oral Cefixime recommended). **Ciprofloxacin** or Ofloxacin **Azithromycin** Doxycycline

SYPHILIS

A chronic systemic infection caused by a spiral organism called Treponema pallidum.
-Transmission by contact with mucosal surfaces or blood and less commonly by contacts with a lesion, sharing needles or transplacental transmission
-Slow multiplication produces **endarteritis** and **granulomas**.

Stages

Primary Syphilis: painless, indurated ulcer on external genitalia or cervix ,
anal or oral site appear after an Enlarged inguinal lymph nodes.

Secondary Syphilis:
symmetric mucocutaneous rash , snail track ulcers
generalized non-tender lymph nodes enlargement (full of spirochete)
Skin lesion distributed on trunk extremities and face.
1/3 develop condylomata lata

Latent Stage:
no clinical manifestations
Risk of blood-borne transmission or from relapsing infection or mother to fetus continue.

Tertiary Syphilis:
Neurosyphilis: chronic meningitis, with increased cells and protein in CSF, leads to degenerative changes and psychosis.
-Demyelination causes peripheral neuropathies.
-Most advanced cases result in paresis

Clinical Manifestations

Cardiovascular syphilis:
Aneurysm of aorta and aortic valve ring.
Gumma on skin, bones, joints → local destruction.

Congenital syphilis:
- if the mother utreated→ fetus susceptible after 4th month of gestation:

➢ Fetal loss, congenital syphilis result (Rhinitis ,rash and bone changes, anemia thrombocytopenia, and liver failure.)

Diagnosis

A. IgM used to diagnose congenital syphilis.
B. Dark field microscopy of smear from primary or secondary lesions. May be negative
C. Serology Test (Common):
   1. Nontreponemal Test: (RPR & VDRL)
      POSITIVE during primary stage ,screening, follow up therapy
   2. Treponemal tests: (FTA-ABS) & (MHA-TP)
      POSITIVE at all stages , confirm RPR & VDRL

Treatment

❖ Penicillin
❖ For Hypersensitive patients: Tetracycline, Erythromycin or Cephalosporins

HERPES (LINEAR DS. DNA)

- HSV 1: targets Trigeminal Ganglia causes encephalitis, oral herpes
- HSV 2: targets Sacral Ganglia, causes genital herpes.
- **Transmission:** sexually, auto-inoculation, perinatal 85% (50% chance if mother has 1° or 8% in recurrent) -> do c-section, Intrauterine 10% (1st trimester-> abortion, after 20 weeks of gestation-> malformation (what are they?)
- **Neonatal Herpes Infection**(caused by HSV-2): 1) Localized skin infection 2) Localized brain infection 3) Generalized Infection (usually fatal)
- Once the virus enters it remains for life (latency)
- **Symptoms:** genital -> Inguinal lymphadenopathy, vesicular herpetic lesion, itching. Extra-genital-> aseptic meningitis. Herpetic proctitis in homos.
- **Lab diagnosis:** ELISA, IF, PCR, tissue culture.
- NO VACCINE
- **Treatment:** Acyclovir

HUMAN PAPILLOMA VIRUS
(CIRCULAR DS. DNA)

- **Cutaneous** (1,2,3,4,10): common, plantar, flat warts.
- **Ano-genital:**

I. Condyloma acuminata (benign HPV 6,11)
II. Cervical carcinoma (HPV 16, 18, 31, 45)
III. Penile & anal carcinoma in men (HPV 16, 18)

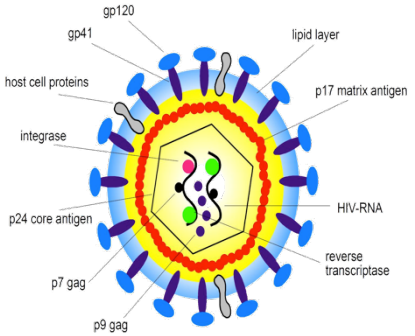
- HPV 16 & 18 are associated with great dysplasia and progression to invasive carcinoma.
- Persistent HPV -> cervical cancer.
- **Symptoms:** 3-4 weeks IP, pain, discomfort, vaginal bleeding, warts.
- **Lab diagnosis**: PCR, pap smear, in situ hybridization. Doesn't grow in tissue culture!
- **Vaccines:**

I. Gardasil -> protection against genital warts
II. Cervarix -> protection against cervical cancer.

- **Treatment:** Cryotherapy, laser, Elctrocautery.

HIV

It is a retrovirus causes AIDS, mainly infects **T-helper cells (CD4)**, resulting in the loss of **cell mediated immunity** , which leads to severe immunologic impairment, leading to multiple opportunistic infections, unusual cancers and death.



Characteristics

Retrovirdae family, viron consist of:

- Glycoprotein envelope (**gp120, gp41**)
- Matrix layer (p17)
- Caspid
- Identical ssRNA (p24)
- Enzymes (Reverse transcriptase, integrase and protease

HIV-1	HIV-2
Causes HIV infection worldwide	Causes infection in regions e.g west africa
Highly virulent	Less virulent
Highly susceptible to mutation	Less susceptible to mutation

Transmission

1- Sexual (STD) direct contact with infected blood, semen and vaginal secretion

2- Parentrally: direct exposure to infected blood or body fluids

3- From mother to child:

- Vertical (25%) transplacentally, but treatment may reduce transmission, given **Zidovudine**
- Perinatal (50%) during delivery , given **Nevirapine**
- Breastfeeding (25%)

Course of HIV Infection

Acute Phase IP= 2-4 weeks, lasts about 12 weeks

- Rapid viral replication, high viral load in serum
- Gradual decrease in CD4 cell count
- 25-65% develop symptoms like infectious mononucleosis or Flu-like syndrome. 13% will be Asymptomatic

Chronic Phase lasts forr 10 years in adults, 5 in children

- Low viral load, CD4 count > 500/ml
- Totally **asymptomatic** but patient is still contagious at the end of this stage patient develop PGL (Presistent generalized lymphadenopathy and ARC (AIDS-Related Complex)

AIDS end stage of the disease

- Continuous viral replication, high viral load
- Marked decrease in CD4<200
- Persistent or multiple frequent opportunistic infections like **Pneumocystis Pneumonia**, and develop unusual cancer (**Kaposi Sarcoma**

Diagnoses

- Detection of both HIV Ag & Ab in the patient serum by **ELISA**, If result is +ve, repeat the screening test in duplicate.
- If still giving +ve result will do confirmatory tests **Western Blot** OR detect Blood viral load by **PCR**

Treatment

High Active Antiretroviral Therapy (HAART)

HAART is usually composed of :

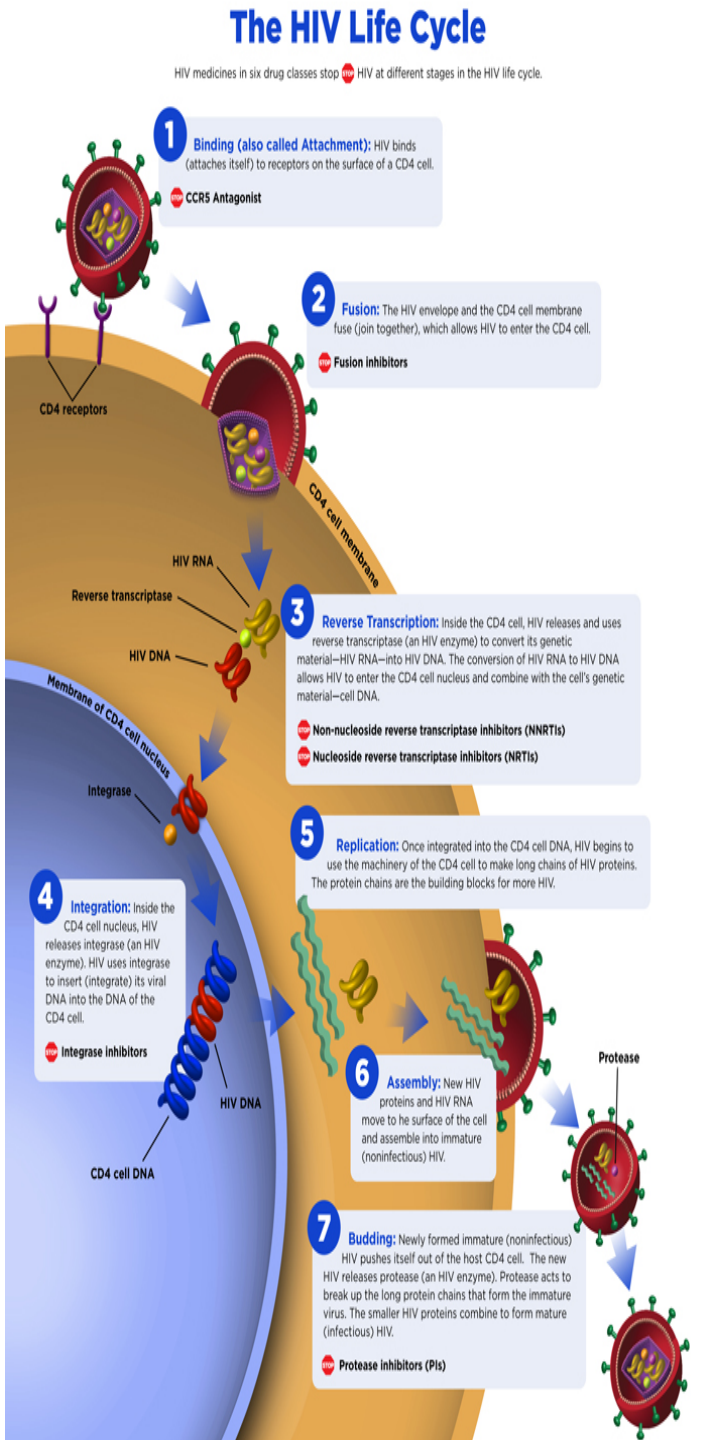
- 2 reverse transcriptase inhibitors:

Zidovudine (AZT) , Lamivudine (3TC)

- 1 protease inhibitor:

Saquinavir, Indinavir, Nelfinavir,

Treatment will never eradicate HIV virus, and it has no vaccine.



THANK YOU FOR CHECKING OUR WORK

DONE BY

Ahad Awadh

Abdullah Albasha

Najla Al draiweesh

Hanan Khushaim

Lina Aljurf

Amal Afrah