Sheet no. : 20

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Slide 4 :

The virus was discovered late, but there are certain immunological conditions which weren’t found before:

**1. Pneumocystis carinii pneumonia**; protozoma cause disease in the lung and lead to very severe pneumonia.

Normally, it isn’t fatal we can find it in normal flora sometimes.

**2.Kaposi's sarcoma** ( is tumor seen in skin )in elderly it`s very rare and not fatal )

Both of them seen in young adults, males and all are homosexual.

Slide 6 :

**HIV-1** (human immunodeficiency virus) structurally its similar to the **retrovirus** which contain double strand RNA and reverse transcriptase .

Slide 7:

Later on they found another virus: **HIV-2** similar to **1** to a certain conditions .

\*\*Also they found another virus seen in the semen of monkeys!

Slide 8 :

It contains **+ve** double strand RNA and reverse transcriptase enzymes.

The structure **is very complex** contain envelop and a lot of glycoproteins (glycoproteins projection 160 Dalton )

The genome also contain very complex genes as in slide 9

Slide 9:

-There are 3 main genes.

-Gp 160 divides into gp120, gp40 and extra proteins.

There are a lot of enzymes needed for the replication of this virus (reverse transcriptase, protease, integrase) these enzymes are very important in the treatment. Sometimes, the drugs used to inhibit some of these enzymes to prevent the viral replication inside the cells.

Slide 10 :

The receptor for this virus is the **CD4** which is found on the surface of the T-lymphocytes and macrophages .

\*So the virus infect only the ( T-lymphocytes and macrophages)

This receptor to be able to work there is another specific chemokine receptor **5 (CCR5)** on the dentritic cell .

When the virus enter the body, it will react with the CD4 in the presence of the (CCR5) which is found on macrophage,so the cell will be infected and the virus go inside.

When the cell starts to move in the blood, it will go to the lymph nodes. In the lymph nodes that cell will be destroyed because the virus will be replicated and released from the cell, this virus will be introduced to the other T-lymphocytes and here the T-lymphocytes will be infected in the presence of CD4 and CCR5 and so on.

If these two receptors (CD4 and CCR5) have mutation, the person will be resistance to this virus and won’t get the disease.

Slide 11:

**The viral life cycle :**

-gp160 associate with CD4 receptor(on the cell membrane).

-Reaction between the envelop (around the virus) and cell membrane of the host cell.

-The virus go inside the cell.

-In cytoplasm: the capsomers will be destroyed and reverse transcriptase will be released.

-That reverse transcriptase will code for a DNA synthesis which is complementary to the viral RNA .

-The DNA integrates to the host genome.

-The integration might stay for a long period (without function) .

-Or it might be function and produce new viruses, these new viruses will be released outside the cell and react with new T-lymphocytes and infect it.(This is seen in **acute infection) .**

Slide 12:

The main ways of transmission are:

**1)** blood **2)** pre-ejaculate **3)** vaginal secretion

Which mean the “**sexual transmission”.**

Slide 13:

the most danger thing is \* homosexuality and sex specially from( male to male and female to male), to transmit this virus there should be blood ,if there is no blood the virus won't survive for long period .

\*we as a dentists, it’s easily transmitted by droplet (from patient to dentist) if you didn’t take care as wearing masks .

Slide 14:

The highest amount of viruses found in the **blood**.

So blood transfusion is the main transmission way.

Slide 15 :

There are few cases (3-4) reported, transmitted by breast milk.

Slide 16-23:

**Immune response:**

**\*Acute infection** :

-As we said the virus go to the dentritic cell through CCR5.

-Then it will react with it and presenting it by **antigen presenting cell**, then it will be seen **by cytotoxic T-lymphocyte(CD8)**,or it will be seen by **CD4**.

-At that time, killing might be happen (by **CD8**) or might give a response (by **CD4**).

- When it response, it will activate B cell and the B cell will produce a lot of antibodies. That’s why at the beginning of the viral infection the number of the antibodies will increase.

-Memory cells will be produced which aren’t activated.

-Then the virus will be disseminated all over the body.

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**\*Latent infection:**

Then the virus established in the tissues and the cells contained the viral genome but without responding (T-lymphocytes aren’t active and aren’t reproducing the virus).

It still for years in the body unless there is another infection or other immune suppression happens.

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**\*AIDS :**

If **reactivation** happen, the virus will be reproduced and almost all the T-lymphocytes will be destroyed(the # is reduced).

And here we can see the AIDS picture

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Slide 24:

**The number of virus**: at beginning it increase and the symptoms are like cold flu (no specific picture we can see), then the virus go down and we start to see latency problems.

**T-lymphocytes** : at early stages are high but with time they go down.

**Antibodies :** IgM and IgG stay for a long period of time and in the incubation period we see high amount of antibodies because the release of viruses happen slowly.

When the T-lymphocytes and antibodies go down and the virus number increase again, here the AIDS condition appear .

There are markers we can see:

- **P24** antigen which will be high at beginning then go down and again will go up.

-**Anti-HIV IgM** will go up at beginning and then go down

“As in the figure”

There is a window 3-5 days in this period we can't see the virus or the antibodies because the immune response is not started yet.

Slide 32:

Opportunistic infection is an infection caused by pathogens ,those that usually do not cause disease in a healthy host in this situation with immunodeficiency person it will cause a lot of complications for example in the upper respiratory tract we will see infection of herpes simplex. All the bacteria present in the normal flora it will produce infection.

The treatment here almost impossible.

Slide 34:

HIV have high mutation rate ( change its antigenicity) 1 mutation per replicated genome . That’s why there is no protective vaccine are available .

Slide 36:

During the window period we might get –ve results (no antigens or antibodies) So the **Seroconversion** is important to do.

Slide 38:

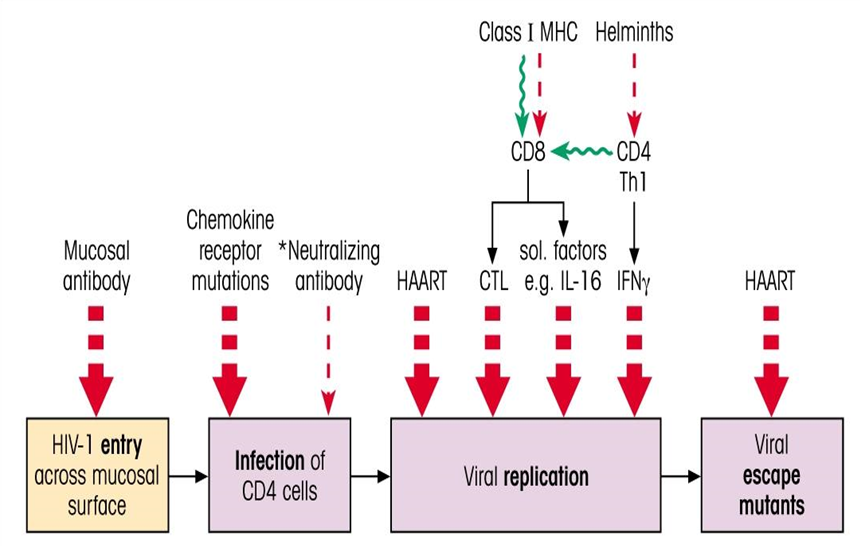
Treatment by Antiretroviral drugs;it target certain areas in the replication of the virus . (Reverse transcriptase inhibitors, Nucleotide Reverse Transcriptase Inhibitors,…..etc “as in the slide”)

-At least 3 drugs should be given for treatment.

-The treatment in Jordan is free.

Slide 40 :

**H**ighly **A**ctive **A**nti-**R**etroviral **T**herapy, different types of treatment depend on the level of the virus .



Slide 42:

There is no effective vaccine till now because of the different components of the virus seen in different patients.

-Still under investigation.

-Subunit vaccine as gp160 or gp40 ,,, etc

Slide 44:

If the patient has a mutation in CCR5 this mean he has resistance to the infection.

So we can use him as a donor(CCR5- ) to the patient who has(CCR5+).

-1st they do total body irradiation to kill the whole **CCR+** in the patient.

-Then they transfer  **CCR-** to him .

-At the end he will have CCR- and might become resistant to the virus .

These are just trials some are success and some aren’t.

Slides 45-53 :

The doctor just read the numbers!

The epidemiology is changing not constant!

\*\*You have to refer to the slides.

\*\*Thank you ^\_^