Human Retinoviruses (1c)

Genome: +ssRNA, linear, (7-11) kbP, diploid / Enveloped

contain RNA directed DNA polymerase also called RT — RNA → DNA

Some important genes: pol, env, Pro and gag

* Infection do not kill infected cell cause latent infection
* Many members are tumor viruses

we can classify retinoviruses into 2 type based on their behavior:
① Exogenous ② Endogenous (non pathogenic) was

HIV

Human Immunodeficiency virus

- HIV-1 common worldwide
  - 4 Groups
    - O, P — from SIV from gorilla
    - N
    - M → cause HIV pandemic from SIV from chimpanze
- HIV-2 (endemic in africa)

Transmission:

1- Heterosexual transmission, 2- Men homosexual
3- Vertical transmission, 4- Injection drug abuse (IDA)
5- Blood brone

Pathogenesis:

1- HIV infect mainly $CD4^+$ T cell and it can infect macrophages & DC through (gp 120)
2- The infection begins locally for 10 days before systemic spread
3- → lymphoid tissue (gut-associated lymphoid tisse) → Blood (viremia) which remains at high levels for about (8-12) weeks
4- The adaptive immune system response take over at this stage to control viral replication → viral load decline (viral-set-point) which is important prognostic marker
5- clinical latency (3-20) years - avg (8-10) years.
6- AIDS when the $CD4^+$ count is less than 200/μL / AIDS ≠ HIV infection

Diagnosis:

HIV-2 / HIV-1 (group M, N & O) → screening relies on enzyme immune assays with forth generation assays combining the detection of Abs with detection of p24. followed by confirmatory test mostly western blot or detection of HIV-1 RNA

Managment:

For managment of the HIV-1 infected

Individual CD4 Tcell count and plasma viral load measurement.

The cornerstone of HIV-1 managment is so called HAART

Treatment:

Combination of antiretroviral drugs ART classified into 6 classes

- NRTI: Zidovudine, Didanosine, Stavudine, Lamivudine, Abacavir, Tenofovir, Emtricitabine
- PI: Saquinavir, Indinavir, Nelfinavir, Atazanavir, Tipranavir, Darunavir
- NNRTI: Nevirapine, Delavirdine, Efavirez, Etravirine, Rilpivirine — العم آثار جانبية
- Integrase Inhibitor: Raltegravir, Dolutegavir and Elvitegavir
- Fusion Inhibitor: Enfuvirtide
- CCR5 Antagonist: Maraviroc

تم اعطاء ٣ أنواع من الأدوية وأهمها المجموعة (1) ثابتة و المجموعة 2 و 3 اختياريات مع 4 - يعني نستخدم 1 مع 2 او 3 او عكس استبدال مجموعة 4 .

Roaa Abuarab.

HTLV

Human T Lymphotropic virus
- HTLV-1 منتشر أكثر
- HTLV-2 more restricted

Transmission: The most common one
① Vertical transmission (breast milk or Placentally)
② Sexually ③ Blood.

pathogenesis:

① HTLV infect & stimulates both mitosis and immortalize T lymphocytes.
② The virus become integrated in the host cell as provirus.
③ over a period of many years, the infected T cell accumulate many chromosomal aberrations → leading to malignant phenotypes.

The viral Tax gene is the critical viral onco-protein.

clinical manifestations: Mostly Asymptomatic

(HTLV-1) associated disease:

1- Adult-Tcell leukemia.
2- Cutaneous Tcell lymphoma
3- Hairly cell leukemia (HTLV-2)
4- HTLV associated myelopathy topical spastic paraparesis

Diagnosis

* ELIBA
* Western blot.

Treatment

Agressive chemotherapy

for prevention screening of blood unit can be done

Vaccines are tested.