AIDS Manifestations

MED 443
King Saud University

Pathology Teamwork
MED 443

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TEAM 443
REPRODUCTIVE UNIT

Objectives

- Understand the pathogenesis of AIDS syndrome.
- Recognize the systemic manifestations of AIDS syndrome with a special emphasis on lymphoma, Kaposi sarcoma and opportunistic infections that could be encountered in AIDS patients.

THIS LECTURE WAS PRESENTED BY DR.MARIA ARAFAH & DR.HAMADA AL JAEDI

IF YOU WANT TO READ THE LECTURE FROM ROBBINS

IF YOU WANT TO READ THE LECTURE FROM FIRST AID

IF YOU WANT TO READ OSMOSIS SUMMARY

IF YOU WANT TO WATCH OSMOSIS VIDEO

Editing File

Color index :
Main text (black)
Female Slides (Pink)
Male Slides (Blue)
Important (Red)
Dr's note (Green)
Extra Info (Grey)

Sexually Transmitted Disease

" not part of the objectives"

EXTRA SLIDE 439

Gonorrhea/ Gonococcal infection السيلان البني	
Info	• It's a Bacterial diseases caused by N. gonorrhoeae (gonococci) • Occurs few days after suspicious intercourse...patient will deny • Chronic infection leads to infertility; rare, as it responds to therapy • Patient will usually go to private clinics to avoid scandals.
Symptoms	• **Female**: Less severe, Asymptomatic • **Male**: burning sensation in penis & urethra, along with pus and purulent discharge .
Treated by	Ampicillin & penicillin, with good prognosis

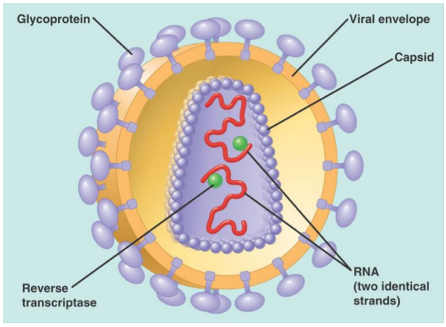
Non-specific Urethritis	
Info	Caused by Chlamydial infection (intracellular)
Symptoms	• **Female**: causes cervicitis • **Male**: burning sensation , secretions
Treated by	Erythromycin & tetracycline

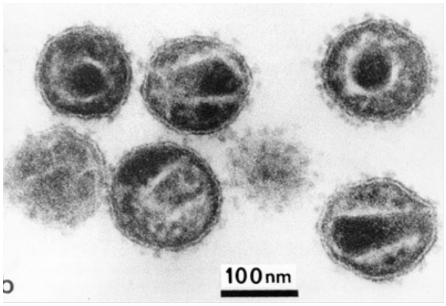
Syphilis الزهري	
Info	• Caused by: Treponema pallidum اللولبية الشاحبة • Usually in young males who likes travelling • Detected by dark-field microscopy
Primary syphilis	• **chancres" superficial painless ulcer**
Secondary syphilis 4 weeks after primary if not treated	• Mucocutaneous lesions (on palms & soles) Rich in plasma • Lymphadenopathy • **Condyloma latum**: elevated lesion on moist areas (around anus, inner thigh..)
Tertiary syphilis after more than one year if not treated	• **Neurological symptoms** Tabes dorsalis. • **CVS symptoms**
Diagnosis	VRDL: serological test that tests positive in syphilis, false positive in autoimmune disease
Treatment	large doses of penicillin

Introduction

General Points

- Human immunodeficiency virus (HIV) is the causative agent for AIDS.
- HIV is a retrovirus from lentivirus family that contains single stranded RNA.
- It was unknown until the early 1980's, but since then it has spread around the world to infect millions of people.
- All HIV infected patients are at a risk of developing deadly illnesses caused by opportunistic infections and tumors and the inevitable manifestations of AIDS.
- The most common type of HIV infection is known as HIV-1 and is the type that has led to the worldwide AIDS epidemic.
- There is also an HIV-2 that is much less common.
- The result of HIV infection is the destruction of the immune system





HIV Structure

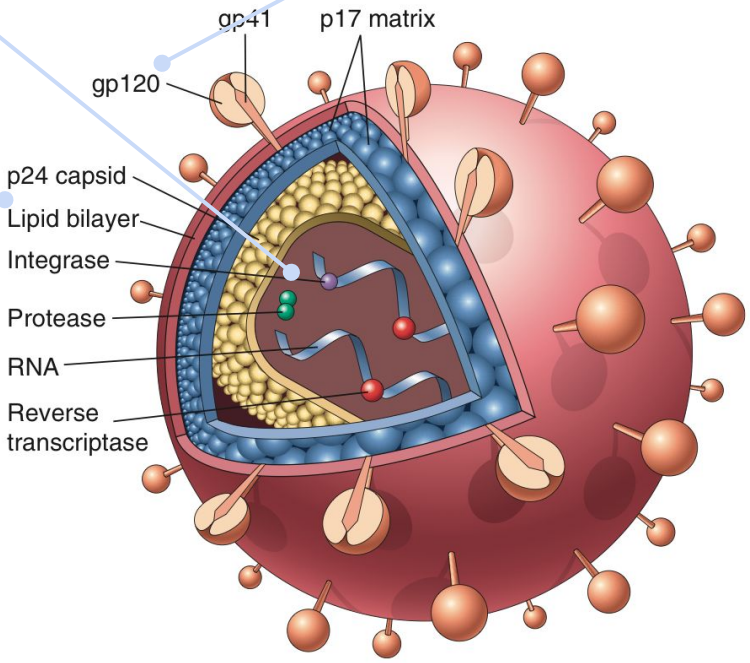
electron dense core: containing the viral genome consisting of the 2 short strands of RNA (ribonucleic acid)

All are encased by an outer lipid envelope.

Enzymes: Reverse transcriptase, protease, ribonuclease and integrase

gp120 aids in the binding of the virus to the target cells. Once the virus enters the human body it attaches itself to the target cell via the CD4 receptors on the surface of the target cell and therefore gains entry into the target cell.
gp120 is responsible for tropism/attraction to CD4+ receptors. This function helps in entry of HIV into the host cell.

antigen: expresses a cell surface protein/antigen called gp120



Pathogenesis

HIV infection

Any CD4+ cell can be infected by HIV. Target cells include:

- blood monocytes and tissue macrophages
- T lymphocytes & B lymphocytes
- Dendritic cell (i.e. the Langerhans cells of epithelial and follicular dendritic cells in lymph nodes)
- Natural Killer lymphocytes
- hematopoietic stem cells
- endothelial cells
- microglial cells in brain gastrointestinal epithelial cells.

In addition, HIV has the ability to mutate easily. This high mutation rate leads to the emergence of HIV variants within the infected person's cells that are more toxic and can resist drug therapy. Over time, different tissues of the body may harbor differing HIV variants

MACROPHAGE AND LANGERHAN CELLS (MACROPHAGES IN THE SKIN)

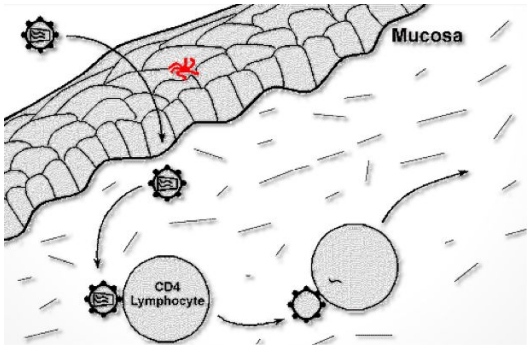
are both important as reservoirs and vectors for the spread of HIV in the body including the CNS and can be infected by HIV but are not destroyed themselves (unlike T-cells). HIV can then be carried via these cells elsewhere in the body.

LYMPH NODE

Once the infection extends to the lymph nodes, the HIV virions are trapped in the processes of follicular dendritic cells FDC's (Macrophages in the lymph nodes) , where they provide a reservoir and infect CD4+ T lymphocytes that are passing through the lymph node. The FDC's themselves become infected, but are not destroyed

Acquired immunodeficiency in not AIDS

Acquired (Secondary) immunodeficiency can be found in many conditions including cancer, metabolic diseases, malnutrition, and most importantly AIDS, however AIDs is not the only cause. Here are some common causes and their mechanisms.



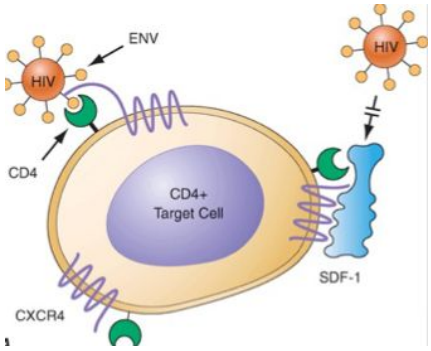
A Langerhans cell in the epithelium is shown in red in this diagram.

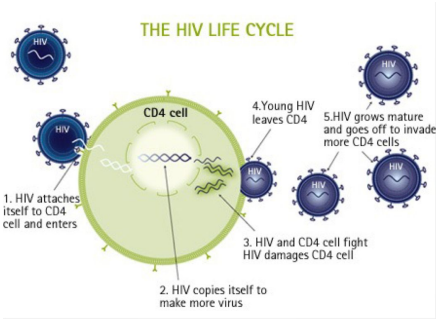
Pathogenesis

HIV Life Cycle

Virus Entry

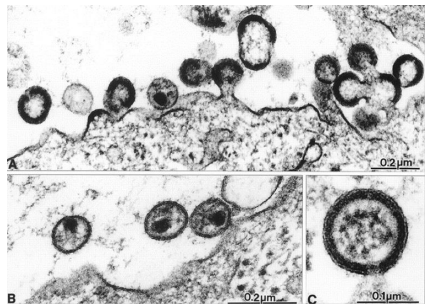
- HIV is shown to cross the mucosa of the genital tract and infect CD4+ + T lymphocytes.
- The probability of infection depends on both the number of infective HIV virions in the body fluid which contacts the host as well as the number of cells with CD4 receptors available at the site of contact.
- The **gp120** on the HIV **binds** to **CD4** receptors and facilitate the viral entry.
- In addition, gp120 also binds to two co-receptors CXCR4 and CCR5 on the host cell surface, which assist in the entry of the virus.
- The T-lymphocytes have surface CD4 receptors (CD4+ T lymphocytes) to which HIV can attach to promote entry into the cell.





Virus Replication

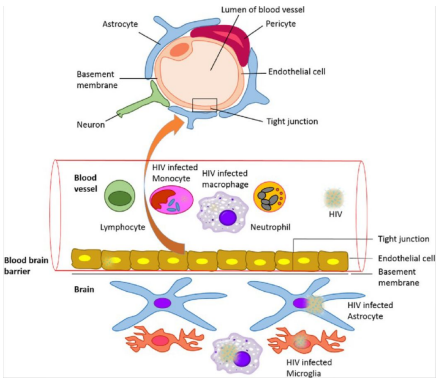
- Retroviruses are unable to replicate outside the living host cells because they only contain RNA and do not contain DNA
- Therefore once HIV infects a cell, it must use its reverse transcriptase enzyme to transcribe/convert its RNA to host cell proviral DNA for replication.
- The enzyme, reverse transcriptase in the HIV helps in the reverse transcription (i.e. conversion) of RNA to proviral DNA. The proviral HIV DNA is then inserted into host cell genomic DNA by the integrase enzyme.
- Once the HIV proviral DNA is within the infected cell's genome, the HIV provirus is replicated by the host cell to produce additional HIV virions which are released by surface budding.



HIV viral particles are seen adjacent to the cell surface in this electron micrograph

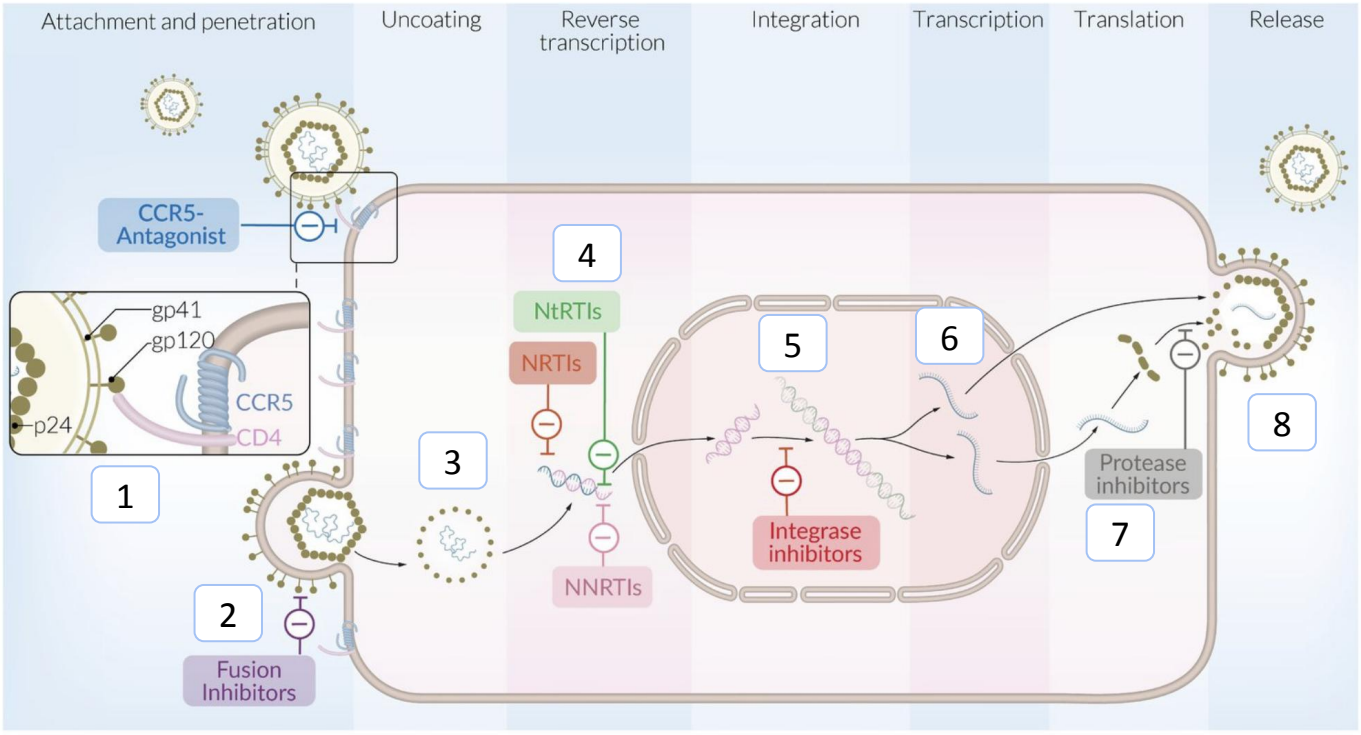
Virus Release

- Alternatively the infected cells can undergo lysis with release of new HIV virions which can then infect additional cells.



Extra Slide

Pathogenesis



CCR5-Antagonist: maraviroc

Fusion inhibitors: enfuvirtid

NtRTIs: tenofovir, adefovir

NRTIs: abacavir, didanosine, emtricitabine, lamivudine, stavudine, zidovudine

NNRTIs: efavirenz, nevirapine

Integrase inhibitors: raltegravir, dolutegravir

Protease inhibitors: lopiravir, ritonavir, indinavir, nelfinavir

1 Attachment: The viral envelope glycoprotein gp120 (cap) binds to CD4 receptors, which are present on certain cells. The gp120-CD4 complex then binds to the host chemokine receptor 5 (CCR5).
– CCR5 antagonists prevent viral attachment to the host cell.

2 Penetration: Viral binding to CCR5 causes a conformational change in the envelope gp41 (stalk), which enables HIV to fuse with and release its capsid into the host cell.
- Fusion inhibitors competitively bind to gp41 and.

3 Uncoating of the viral capsid to release viral RNA

4 Reverse transcription: Viral RNA is transcribed into viral DNA by viral reverse transcriptase enzymes.
Nucleoside reverse transcriptase inhibitors (NRTI), nucleotide reverse transcriptase inhibitors (NtRTI), and non-nucleoside reverse transcriptase inhibitors (NNRTI) inhibit the viral reverse transcriptase enzyme..

5 Integration: Newly transcribed viral DNA is integrated into host DNA by viral integrase.
– Integrase inhibitors inhibit viral integrase.

6 Transcription: Newly integrated DNA is used as a template to create copies of viral RNA that will encode for viral proteins.

7 Translation: Newly transcripted viral RNA acts as templates for the synthesis of new viral proteins synthesis. Viral protease enzyme cleaves the long polypeptide chains into shorter, functional viral proteins (e.g., envelope proteins, enzyme proteins).
– Protease inhibitors inhibit viral protease and prevent the generation of functional viral proteins.

8 Release: New viral RNA and viral proteins use a portion of the host cell membrane as a viral envelope and then leave the host cell.

Made by : Lama Alotaibi

Modes Of Transmission

Sexually Transmissible disease

- HIV is primarily spread as a sexually transmissible disease.
- Transmission of HIV can occur from
a. male to male
b. male to female
c. female to male.
d. Female to female transmission remains extremely rare.

Parenteral Routes

- **IV drug users** sharing infected needles. Less common practices like the use of instruments such as tattoo needles that are not properly disinfected also carry a potential risk.
- **Health care workers** with percutaneous exposures (needle puncture) to HIV-containing blood.
- Persons receiving multiple blood transfusions, e.g. hemophiliacs.
  - Screening of blood products for HIV has significantly reduced HIV transmission by this means.
- HIV can be present in a variety of body fluids and secretions. They include **genital secretions, blood, breast milk, saliva, urine, tears, and sweat.**
  - However, saliva, urine, tears, and sweat are of no major clinical importance, as transmission of HIV through these fluids does not routinely occur because of the low concentration of HIV in these fluids.

Congenital Infection

- Congenital Infection: perinatally or in infancy
- Mothers with HIV infection can pass the virus:
  - Transplacentally i.e. in utero
  - At the time of delivery through the birth canal
  - Through breast milk
- HIV infection is not spread by casual contact in public places, households, or in the workplace. HIV does not spread by insect vectors.
- There is no vaccine to prevent HIV infection,because of its mutations

Diagnosis

Diagnosis

Better detailed info will be discussed in microbiology

ELISA

- Test for HIV antibodies is done with a rapid test using an (ELISA) technique.
- if rapid test is positive, then the next step is to: Confirm HIV infection with Western blot or immunofluorescence assay (IFA)

WESTERN BLOT OR IMMUNOFLUORESCENCE ASSAY (IFA)

- **Confirm** HIV infection with Western blot or immunofluorescence assay (IFA)
- The average HIV-infected person may take up to several weeks to become seropositive (Window period), and then may live up to 8 or 10 years, on average, before the development of the clinical signs and symptoms of AIDS.
- PCR (polymerase chain reaction): most sensitive.

Clinical Behaviour

Clinical Presentation

Primary HIV infection may go unnoticed (asymptomatic) in at least half of cases produce a mild disease which quickly subsides, or produce an acute HIV infection, followed by a long clinical "latent" period lasting years. CD4 is still not markedly decreased, so no infections will happen

Primary acute HIV infections may include fever, generalized lymphadenopathy, pharyngitis, rash, arthralgia and diarrhea. These symptoms diminish over 1 to 2 months.

Clinical AIDS

01

The primary target of HIV is the immune system, which is gradually destroyed.

02

Clinically, HIV infection may appear "latent" for years. During this period there is ongoing immune system destruction but still enough of the immune system remains intact to provide immunity and prevent most infections.

03

Eventually, when a significant number of CD4+ T lymphocytes have been destroyed and when production of new CD4+ cells cannot match destruction, failure of the immune system leads to the appearance of clinical AIDS.

04

The progression to clinical AIDS is also marked by the appearance of syncytia-forming (SI) variants of HIV in about half of HIV infected patients.

05

These SI variants are associated with more rapid CD4+ cell decline.

06

The development of signs and symptoms of AIDS correlates with the CD4+ lymphocyte count.

07

When the CD4+ lymphocyte count drops **below 200/microliter,** then the stage of clinical AIDS has been reached. This is the point at which the characteristic opportunistic infections and neoplasms of AIDS appear.

08

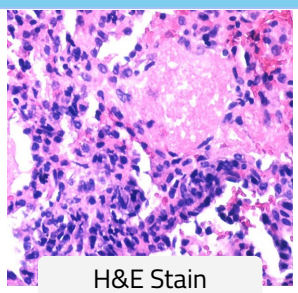
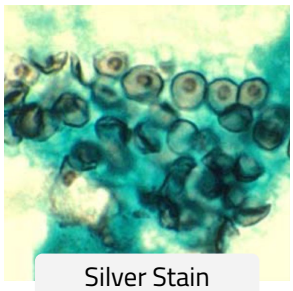
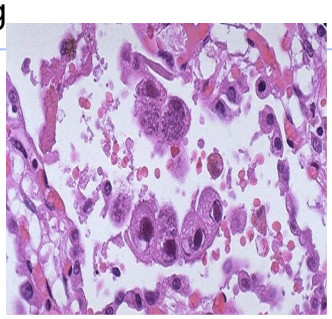
The CD4+ T-cells to CD8+ T-cells ratio is also greatly reduced, often to less than 1.0 where the normal ratio is about 3:1.

Complications of AIDS

Acquired Immunodeficiency Syndrome (AIDS) (Final stage)

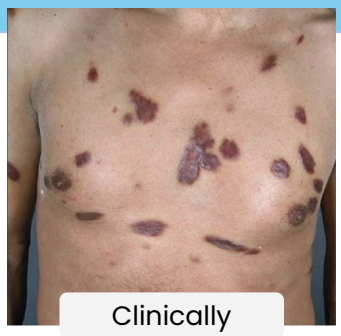
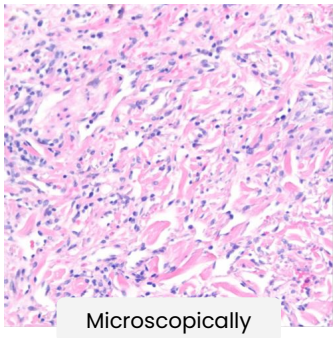
The stage of clinical AIDS is reached years after the initial infection and is marked by the development of one or more of the typical opportunistic infections or neoplasms common to AIDS.
Following are some of the more common complications seen in AIDS:
- **Infections** e.g. pneumocystis jiroveci, CMV, mycobacteria, fungi.
- **Neoplasms**
- **Miscellaneous** e.g. lymphoid interstitial pneumonitis which is a condition involving the lung that can be seen in AIDS in children

Infection

Fungal	Pneumocystis Jiroveci	Pneumocystis jiroveci (P. carinii) is the most frequent opportunistic infection seen in AIDS. It commonly produces a **pulmonary infection.** (Pneumonia) • The diagnosis is made histologically by finding the organisms in cytology (bronchoalveolar lavage) or biopsy (transbronchial biopsy) material from the lung. • In the lung, there is soap bubble like intra-alveolar exudate and the organism appears as cyst like structures that are positive to silver stain.(GMS)	 H&E Stain  Silver Stain
	Others	• Candidiasis of the esophagus, trachea, bronchi, or lungs. • Cryptococcus neoformans (produces pneumonia and meningitis), Histoplasma capsulatum, and Coccidioides immitis. • Aspergillosis especially in the lung	
Viral	Cytomegalovirus	CMV infection is seen in AIDS. • It causes pneumonia and it can also cause serious diseases in the brain and gastrointestinal tract.(CMV colitis). • It is also a common cause for retinitis and blindness in persons with AIDS.	
	Others	• Herpes simplex Virus infection in the mucosa • Viral HIV encephalitis	

Complications of AIDS

Infection		
Bacterial	Mycobacterial infections	• Mycobacterium tuberculosis. • Mycobacterium avium complex (MAC) infection. • Definitive diagnosis of mycobacterial disease is made by culture and PCR. • No caseating granuloma; as there is depletion of CD4+ cells. هجت الكي ورد من سنة أولى وهي تنحرق
	Syphilis	primary, secondary and tertiary
Protozoa	Toxoplasmosis	Toxoplasmosis caused by Toxoplasma gondii is a protozoan parasite that most often leads to infection of the brain with AIDS.
	Cryptosporidium and Microsporidium	Cryptosporidium and Microsporidia produce voluminous watery diarrhea in patients with AIDS

Neoplasms		
Kaposi's sarcoma (KS)	• Sarcoma : malignant mesenchymal tumor • It is a sarcoma of the blood vessels. (Endothelial origin) • Visceral organs can also be involved with KS. • produces reddish purple patches or nodules over the skin and can be diagnosed with skin biopsy. • It is associated with **HHV-8** and on histology, it shows malignant spindle cells of vascular origin.	 Clinically  Microscopically
Lymphoma	• Malignant lymphomas are seen with AIDS, and related to EBV • They are commonly B-cell non-Hodgkin's lymphoma. • They are typically of high grade and often in the brain. • They are very aggressive and respond poorly to therapy.	

Keywords

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

2 short strands of RNA (ribonucleic acid).
Reverse transcriptase : to convert its RNA to host cell proviral DNA for replication.
protease : synthesis of viral proteins
integrase : proviral DNA is inserted into the host genomic DNA by it
Sexually transmitted , Parenterally or Congenitally
For diagnosis : Test for HIV antibodies is done with a rapid test using an (ELISA) & confirm by Western blot or immunofluorescence assay (IFA)
PCR (polymerase chain reaction): most sensitive.
Clinical aids spear when significant number of CD4+ T lymphocytes have been destroyed and when production of new CD4+ cells cannot match destruction
progression marked by syncytia-forming (SI)
Stages of clinical AIDS : CD4+ lymphocyte count drops below 200/microliter

</td></tr>
<tr><td rowspan="8">Complications</td><td rowspan="2">Fungal</td><td>Pneumocystis Jiroveci :
produces a pneumonia.
In the lung, there is soap bubble like intra alveolar exudate
positive to silver stain</td></tr>
<tr><td>Other :
Cryptococcus neoformans (produces pneumonia and meningitis) , Candidiasis , Aspergillosis</td></tr>
<tr><td rowspan="2">Viral</td><td>Cytomegalovirus :
causes pneumonia</td></tr>
<tr><td>Herpes simplex Virus infection in the mucosa , HIV encephalitis</td></tr>
<tr><td>Bacterial</td><td>Mycobacterial :
Mycobacterium tuberculosis : No caseating granuloma
Mycobacterium avium complex (MAC) infection.
Syphilis</td></tr>
<tr><td rowspan="2">Protozoa</td><td>Toxoplasmosis :
caused by Toxoplasma gondii
Lead to infection of the brain</td></tr>
<tr><td>Cryptosporidium & Microsporidium : produce voluminous watery diarrhea</td></tr>
<tr><td>Neoplasms</td><td>Kaposi's sarcoma :
sarcoma of the blood vessels.
reddish purple patches
malignant spindle cells of vascular origin.
Associated with HHV-8</td></tr>
<tr><td>Lymphoma</td><td>commonly diffuse large B-cell non-Hodgkin's lymphoma.
typically of high grade and often in the brain.</td></tr>
</table>

YOU vs MCQs

Question 1

Which of the following is not a receptor for binding of HIV?

- CD4+ receptors
- CXCR4
- CCR5
- TM4SF5

Question 2

Which one of the following tests is used to confirm HIV infection?

- ELISA
- CD4 Count
- Immunofluorescence assay (IFA)
- Microscopic testing

Question 3

When does the signs and symptoms and infections of AIDS begin to show?

- CD4 & CD8 below 200/microliter
- CD4 count below 200/microliter.
- CD8 count below 200/microliter
- CD4 count below 100/microliter

Question 4

Which one of the following is a characteristic clinicopathological feature about Aids-associated Kaposi's sarcoma?

- Multiple skin lesions indicate a poor prognosis
- Is a spindle cell tumor of lymphoepithelial origin
- Caused by infection of human herpesvirus 6
- The commonest site of visceral spread is the brain

1- D / 2-C / 3- B / 4-B

YOU VS MCQs

Question 5

What is the most common lymphoma in AIDS patient

- B cell non Hodgkin lymphoma
- B cell Hodgkin lymphoma
- Polycystic ovarian syndrome
- Chronic lymphocytic leukemia

Question 6

Kaposi sarcoma is usually associated with which of the following?

- HCV
- HHV-8
- HHV-6
- HPV

Question 7

A patient with AIDs came to the clinic with fever, vomiting , and he also mentioned that his vision is decreasing significantly , which of the following might be the problem?

- Kaposi sarcoma
- Pneumocystis Jiroveci
- Cytomegalovirus
- Aspergillosis

Question 8

A 31 year old male presented with HIV. His history showed he had fever, cough, and difficulty breathing. The diagnosis of pneumonia was made. Transbronchial biopsy shows bubble soap appearance and small cyst which are positive to silver stain. Which one of the following is the most likely causative organisms?

- Aspergillus fumigatus
- Cryptococcus neoformans
- Pneumocystis jiroveci
- Mycobacterium avium

5- A / 6-B / 7- C / 8-C

Cases

1.A 31-year-old man with AIDS complains of difficulty swallowing. Examination of his oral cavity demonstrates whitish membranes covering much of his tongue and palate. Endoscopy also reveals several whitish, ulcerated lesions in the esophagus. These pathologic findings are fundamentally caused by loss of which of the following immune cells in this patient?

A.B lymphocytes	B.Helper T lymphocytes	C.Killer T lymphocytes	D.Monocytes/macrophages

2.Which of the following enzymes converts the HIV genome into double-stranded DNA in host cells in the patient described in Question 1?

A.DNA polymerase (Pol-1)	B.DNA polymerase (Pol-2)	C.Integrase	D.Reverse transcriptase

3.A 28-year-old woman with a history of drug abuse presents with an infectious mononucleosis-like syndrome and lymphadenopathy. Blood tests subsequently indicate that she is HIV-positive. Which of the following lymphocyte-associated proteins mediates the entry of HIV into host cells in this patient?

A.CD4	B.CD8	C.GP41	D.GP120

4.A 45-year-old man presents with a 9-month history of a reddish nodule on his foot. Biopsy of the nodule discloses a poorly demarcated lesion composed of fibroblasts and endothelial-like cells lining vascular spaces. Further work-up identifies similar lesions in the lymph nodes and liver. The tumor cells contain sequences of human herpesvirus-8(HHV-8). This patient most likely has which of the following diseases?

A. Acquired immunodeficiency	B.Ataxia telangiectasia	C. Li-Fraumeni syndrome	D.Neurofibromatosis type I

Here is the pathology Alien , for the last 2 years I have been explaining every pathology case for you , and you have probably seen me jumping around from anatomy , microbiology , pharmacology , haematology to embryology and more asking you questions as my hobby or explaining things .
And now our journey in the pathology has come to an end ..
see you in clinical years

AND NOW FOR THE LAST TIME , NEED EXPLANATION ? CLICK HERE

Best of luck
Pathology Alien

1-B / 2-D / 3-A / 4-A

Cases

EXTRA CASES MAY REQUIRE EXTRA INFO

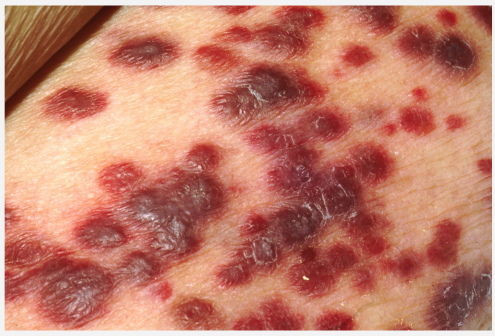
1.A 32-year-old primigravid woman at 34-weeks of gestation comes to the office for her first visit. She has not had vaginal bleeding or cramping. She immigrated from Ghana six weeks ago and has not had any prenatal care. The patient has a history of intravenous drug use, including heroin. She does not use tobacco or alcohol. The patient had sexual intercourse with an HIV-infected individual one year ago and did not receive post-exposure prophylaxis. The patient's blood type is AB positive. Temperature is 37.0°C (98.6°F), pulse is 99/min, respirations are 20/min, and blood pressure is 110/75 mmHg. Physical examination reveals anterior and posterior cervical lymphadenopathy. Ultrasound confirms a 34-week intrauterine pregnancy. The toxicology screen is negative. After birth, which of the following is the most appropriate diagnostic investigation to obtain for suspected neonatal human immunodeficiency virus (HIV)?

A.HIV antibody-only immunoassay	B.No further workup for the mother or fetus	C. Nucleic acid amplification test for HIV RNA	D.Anti D immunoglobulin assay

2.A 28-year-old man comes to his primary care physician for three days with fatigue, fever, and a sore throat. Past medical history is significant for Chlamydia urethritis as a teenager, and two months ago, he was diagnosed with herpes simplex genitalis. The patient is sexually active and uses condoms inconsistently. Temperature is 38.11°C (100.6°F), pulse is 99/min, respirations are 20/min, and blood pressure is 120/75 mmHg. Physical examination reveals anterior and posterior cervical lymphadenopathy and a well-circumscribed, oval-shaped maculopapular rash over the abdomen, trunk, and face. Oral examination reveals mucocutaneous ulcers on the tongue, oral mucosa, and palate. Pharyngeal edema and hyperemia are present without exudates or tonsillar enlargement. Abdominal examination is noncontributory. Heterophile antibody screening is negative. Which of the following best describes the most likely microbiological agent responsible for this patient's symptoms?

A.Nonenveloped, single-stranded, linear DNA virus	B.Enveloped, double-stranded, linear DNA virus	C.Nonenveloped, double-stranded, linear DNA virus	D.Enveloped, single-stranded, linear RNA virus

3.A 45-year-old man comes to the clinic with skin lesions over his trunk, abdomen, and face. The patient also notes significant weight loss over the last six months. Past medical history is significant for gastroesophageal reflux disease, alcoholic cirrhosis, and esophageal varices. The patient was admitted two months ago for bleeding esophageal varices and underwent endoscopic variceal ligation. The patient is sexually active and uses condoms inconsistently. The patient uses intravenous drugs, including morphine, and consumes alcohol regularly. Temperature is 36.6°C (98.0°F), pulse is 99/min, respirations are 20/min, and blood pressure is 120/75 mmHg. Physical examination reveals anterior and posterior cervical lymphadenopathy. Oral examination reveals white mucosal plaques that cannot be scraped off easily. Skin lesions on the right arm are depicted below. This patient's clinical presentation suggests which of the following underlying conditions?



A.Pancreatic cancer	B.Gastric carcinoma	C.Acquired immunodeficiency syndrome	D.Parkinson's disease

SUMMARY

IF YOU WANT A SUMMARY CLICK HERE

1-C / 2-D / 3-C

Pathology Team

Leader
زياد العتيبي

- هياء العجمي
- ريماز المحمود
- لؤي الحديثي
- فيصل الشويعر
- محمد السلامة
- عبدالمحسن الدايل
- أريج القريني
- عبدالله الزامل
- رزان السطيحي
- رغد المصلح
- ألين الكلية
- مريم الغنام
- محمد العرفج
- رند ابا الخيل
- وجد المطيري
- ريوف الأحمري
- سلطان البقمي
- شوق الخليفة
- عروب المحمود

Leader
لمى العتيبي

- الجوهرة الوهيبي
- ريناد صالح الشهري
- دانه المحيسن
- الدانه عبدالله
- ساره الشهراني
- أفنان الأحمري
- هيا الزير
- محمد معشي
- يزيد المطيري
- ريما القرني
- سديم اليحيى
- عائشة إبراهيم
- شادن الهزاني
- ساره الدوسري
- هدى الجدعان
- ليان الرويلي
- ساره العجاجي
- رغد الحامد
- مشعل الدخيل
- سلمى السعدون

Pathology Team

All Basic Years !

Renal Block

Leaders	
لمى العتيبي	زياد العتيبي
ليان الرويلي اسيل الشهري ساره العجاجي عروب المحمود رغد المصلح دانه المحيسن الجوهرة الخليفة رهف السلمه ريماز المحمود عائشة بورقه فيصل الشويعر	محمد العرفج قاسم الوائلي سلطان البقمي محمد معشي عبدالرحمن الأحيدب عبدالله الكودري يزيد ال طلحه عبدالعزيز القرني يزيد المطيري عبدالرحمن الأحمري

Cardiovascular block

Leaders	
لمى العتيبي	عبدالله القرني
ليان الرويلي ريما القرني عائشة إبراهيم عروب المحمود رغد المصلح ريماز المحمود هياء العجمي رهف السلمه رغد الحامد شادن الهزاني سلمى السعدون وعد الركابي مريم الغنام	زياد العتيبي محمد معشي أبو عويد قاسم الوائلي سلطان البقمي لؤي الحديثي محمد العرفج يزيد ال طلحه ريان الشهري فهد المغيصيب شوق الخليفة ليلى الفرحان افنان الضيفان

Respiratory block

Leaders	
لمى العتيبي	عبدالله القرني
عروب المحمود هياء العجمي شادن الهزاني فرح ابو خلف ريما القرني رزان السطيحي افنان الضيفان رهف السلمه شوق الخليفة سلمى السعدون ساره العجاجي	عبدالمحسن الدايل مشعل الهلال محمد معشي الوليد الزهراني قاسم الوائلي نواف العمار زياد. العتيبي ريان الفايز عبدالرحمن العصلب موده الصبي ليلى الفرحان

Foundation block

Leaders	
ساره العجاجي موده الصبي	بدر الغامدي عبدالعزيز القرني
نورة العتيق وسن العنزي ريما السحيباني رهف السلمة الدانه عبدالله رغد المصلح لمى.العتيبي ساره الشهراني روان القحطاني رغد القحطاني وعد القحطاني	فيصل الشويعر طلال الغدير أبان بصفر زياد حكمي محمد العرفج محمد الصعيري حمد الزيادي عبدالله العويس يوسف بادغيش أحمد السويلم الجوهرة الوهيبي

Reproductive block

Leaders	
لمى العتيبي	زياد العتيبي
هياء العجمي رغد المصلح الجوهرة الوهيبي سديم اليحيى ريماز المحمود ألين الكلية ريناد صالح الشهري عائشة إبراهيم لؤي الحديثي مريم الغنام دانه المحيسن شادن الهزاني فيصل الشويعر محمد العرفج الدانه عبد الله ساره الدوسري محمد السلامة رند ابا الخيل ساره الشهراني	هدى الجدعان عبد المحسن الدايل وجد المطيري أفنان الأحمري ليان الرويلي أريج القريني ريوف الأحمري هيا الزير ساره العجاجي عبد الله الزامل سلطان البقمي محمد معشي رغد الحامد رزان السطيحي شوق الخليفة يزيد المطيري مشعل الدخيل عروب المحمود ريما القرني سلمى السعدون

Gastrointestinal block

Leaders	
لمى العتيبي	زياد العتيبي
عائشة إبراهيم عبد الرحمن المسلم نورة المحيميد الدانة عبد الله رغد المصلح ليان الرويلي ريما المطيري فيصل الشويعر ريماز المحمود زياد حكمي سلطان البقمي عروب المحمود خالد الرشيد عبد الله الضويحي عبد المحسن الدايل نوره المالك شوق الخليفة دينا المهوس	الجوهرة الوهيبي لؤي الحديثي هيا الزير عبد الله الكودري محمد السلامه معاذ الحضيف إيلاف معتبي يوسف بادغيش ساره العجاجي يزيد ال طلحه أفنان الأحمري رند ابا الخيل رزان السطيحي هدى بن جدعان منصور العتيبي زياد السويلم محمد العرفج دانه المحيسن عبدالرحمن الأحيدب

Central nervous system block

Leaders	
لمى العتيبي	زياد العتيبي
عبد الله الكودري هياء العجمي شوق الخليفة رند ابا الخيل محمد العرفج عمر المقرن محمد معشي ريما المطيري ساره العجاجي أفنان الأحمري وعد العنزي نوره المحيميد رزان السطيحي يزيد المطيري	عائشة ابراهيم فيصل الشويعر منصور العتيبي رغد المصلح معاذ الحضيف ريماز المحمود الحوراء العوامي عروب المحمود ليان الرويلي يزيد ال طلحه لؤي الحديثي نوره المالك سلطان البقمي الجوهرة الوهيبي

Musculoskeletal block

Leaders	
لمى العتيبي	عبدالعزيز القرني
عائشه ابراهيم هيا العجمي منار الجنوبي فرح ابو خلف سمر العنزي مها السحيباني ساره العجاجي	مروان المالك مهند العتيبي بدر الغامدي عبدالمحسن الدايل قاسم الوائلي ماجد القطاني رزان السطيحي

Academic & Batch Leaders

Batch

1st year	2nd year
ليان الرويلي عمر المقرن محمد معشي	نوره العتيق طلال الغدير عبدالله الربيعة

Academic

1st year	2nd year
ريماس الجعيدي رغد الحامد محمد العرفج يوسف بادغيش	لمى المطيري عبدالعزيز الفهيد محمد العيسى

Endocrine block

Leaders	
لمى العتيبي	زياد العتيبي
رغد المصلح ليان الرويلي سلطان البقمي ريماز المحمود محمد العرفج عروب المحمود عائشة إبراهيم	يزيد آل طلحه شوق الخليفة إيلاف معتبي ريما المطيري دينا المهوس عبد المحسن الدايل	أفنان الأحمري رند ابا الخيل ساره العجاجي معاذ الحضيف دانه المحيسن نوره المحيميد فيصل الشويعر	الجوهرة الوهيبي يزيد المطيري رزان السطيحي ألين الكلية لؤي الحديثي