

HEPATITIS

cultivable	HAV	- Picorna viridae
Non cultivable	HBV	- Hepadna viridae
	HCV	- Flavi viridae
	HDV	- Delta viridae
	HEV	- Calci viridae

IgM ELISA (IOC) for all Hepatitis viruses except
HCV → PCR (IOC)

HAV · only cultivable Hepatitis virus
diagnosed by IgM ELISA
live vaccine - formalin inactivated

HCV · non cultivable
diagnosed by PCR IOC
∴ IgM not detected in 20-30% pts
mc transfusion associated - HCV

HCV : associated w Anti UKM-1 antibody

HDV

- non cultivable

IgM ELISA (Ioc)

Defective virus - cause disease $\approx$ HBV

HBV provides envelope protein HBsAg

Coinfection

IgM of HDV

IgM of HBV

Superinfection

IgM of HDV

IgG of HBV

HDV : associated $\approx$ Anti LKM-3 antibody

HEV

non cultivable

IgM ELISA (Ioc)

normal person $\rightarrow$ mortality 2-4%

pregnant woman $\rightarrow$ mortality 20-40%

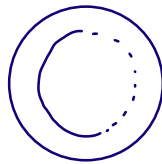
aka E-NANB - Enterically transmitted

Non A Non B hepatitis virus

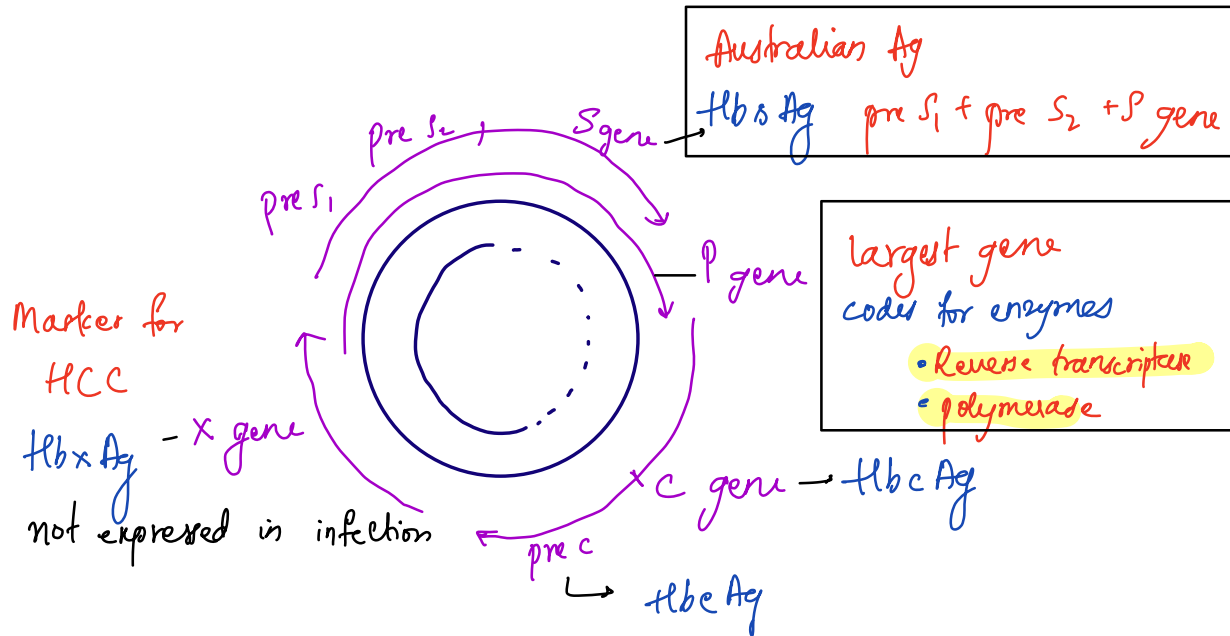
HBV → DANE PARTICLE

Structure

ds circular DNA



inner strand is incomplete
∴ partially ds DNA

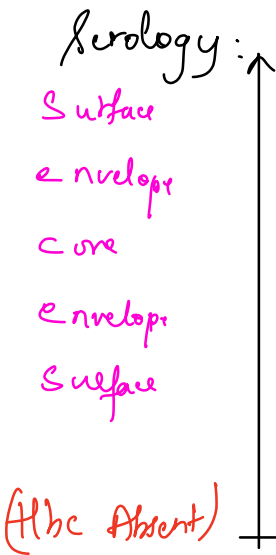


Apart from Retroviridae, virus having reverse transcriptase → **FLAV**

Hbs Ag
Hbc Ag
Hbe Ag } Infection marker

Hbx Ag - marker for HCC

Note: asymptomatic carriers are @ risk for developing HCC ∴ HBV DNA is integrated into cell genome



- Hb s Ag - active infection
- Hb c Ag - x
- Hb e Ag - active multiplication of virus (highly infective)

Anti-Hbc $\begin{cases} \text{IgM} - \text{acute} \\ \text{IgG} - \text{chronic} \end{cases}$

Anti HBe - No multiplication
(low infective)

Dna

E antigen HBsAg

Lipid bilayer membrane

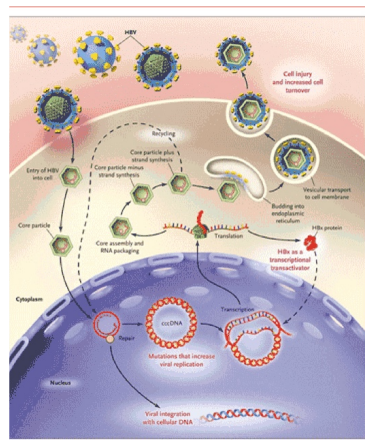
Dna polymerase

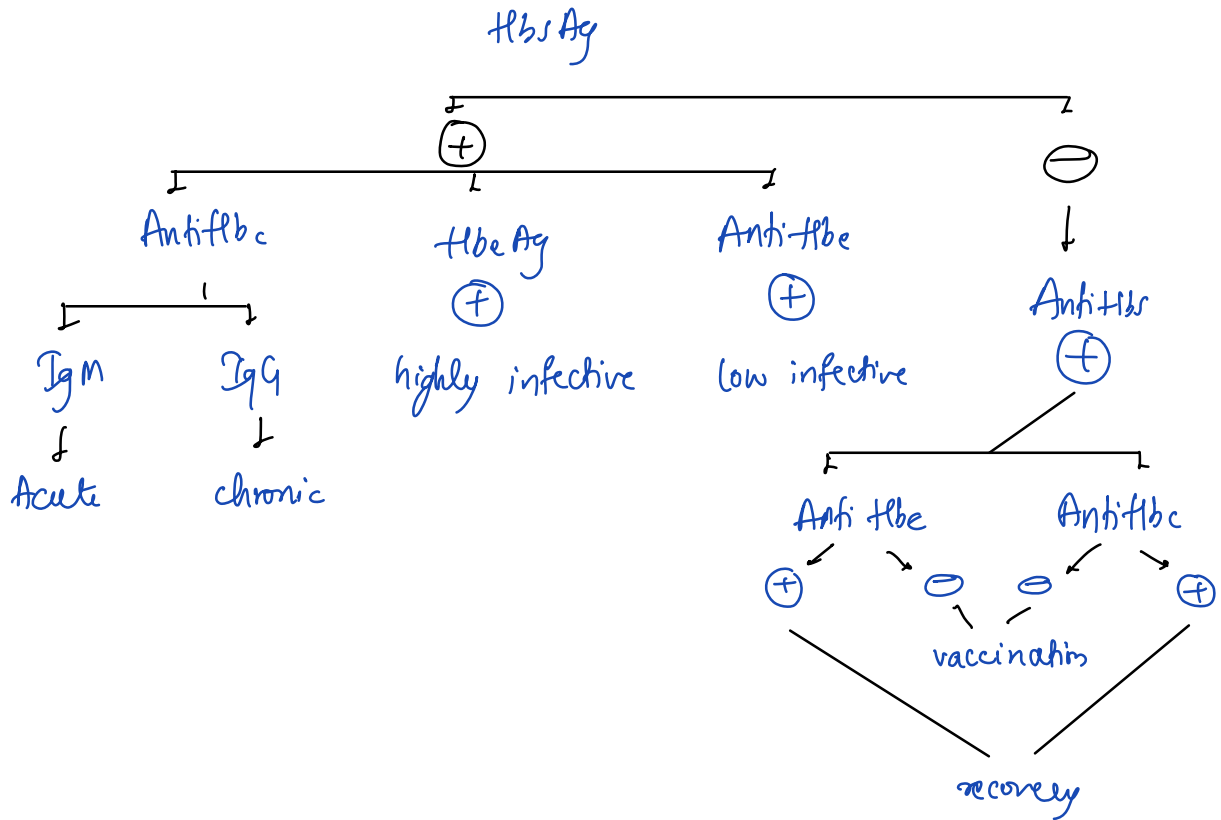
Core antigen HBcAg

Large surface protein HBsAg

Small surface protein HBsAg

Medium surface protein HBsAg





Vaccination : genetically engineered by cloning S gene in baker's yeast

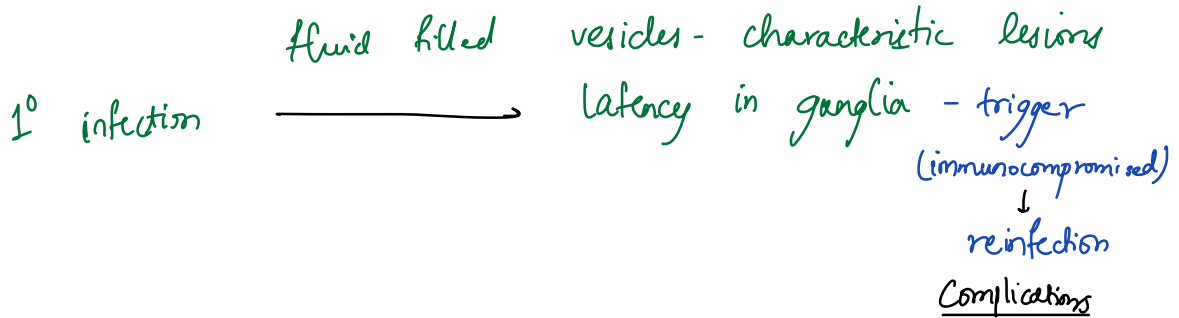
0, 1, 6 months

pregnancy is not a contraindication

If Ab titre > 10 mIU/ml No Rx on prick

Vaccination status of exposed worker		Source HBsAg +	Source HBsAg -	Source Unknown
Unvaccinated		HBIGx 1 and initiate HB vaccine series	Initiate HB vaccine series	Initiate HB vaccine series
Previously vaccinated	Known responder*	No treatment	No treatment	No treatment
Previously vaccinated	Known Unresponder	HBIG x 1 and initiate revaccination or HBIG x 2	No treatment	If known high risk source, treat as if source were HBsAg positive
Previously vaccinated	Unknown	Test exposed person for anti-HBs 1. If adequate(anti-HBs >10 mIU/mL), no treatment is necessary 2. If inadequate(anti-HBs <10 mIU/mL), administer HBIG x 1 and vaccine booster	No treatment	Test exposed person for anti-HBs 1. If adequate(anti-HBs >10 mIU/mL), no treatment is necessary 2. If inadequate, administer vaccine booster and recheck titer in 1–2 months

HERPES



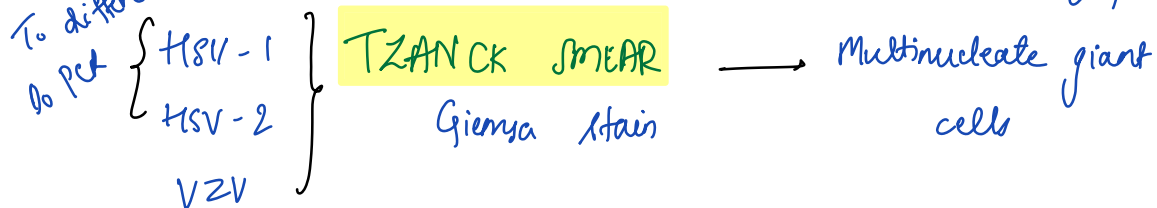
- HHV 1 HSV-1 (mc) - above umbilicus - orofacial - **mec of sporadic encephalitis (temporal lobe inv)**
- HHV 2 HSV-2 - below umbilicus - genital - neonatal herpes
- HHV 3 VZV 1° inf : Chicken pox

Reactivation: Herpes zoster (Shingles)

Congenital inf: Fetal varicella syndrome → limb hypoplasia, cicatrization

- HHV 4 EBV Infectious mononucleosis
- HHV 5 CMV Infectious mononucleosis **largest herpes virus**
- HHV 6 - } 6th disease
- HHV 7 - } Exanthema subitum / Roseola infantum, Pityriasis rosea
- HHV 8 - **Kaposi sarcoma**, Castleman disease, 1° effusion lymphoma

To differentiate
do PCR



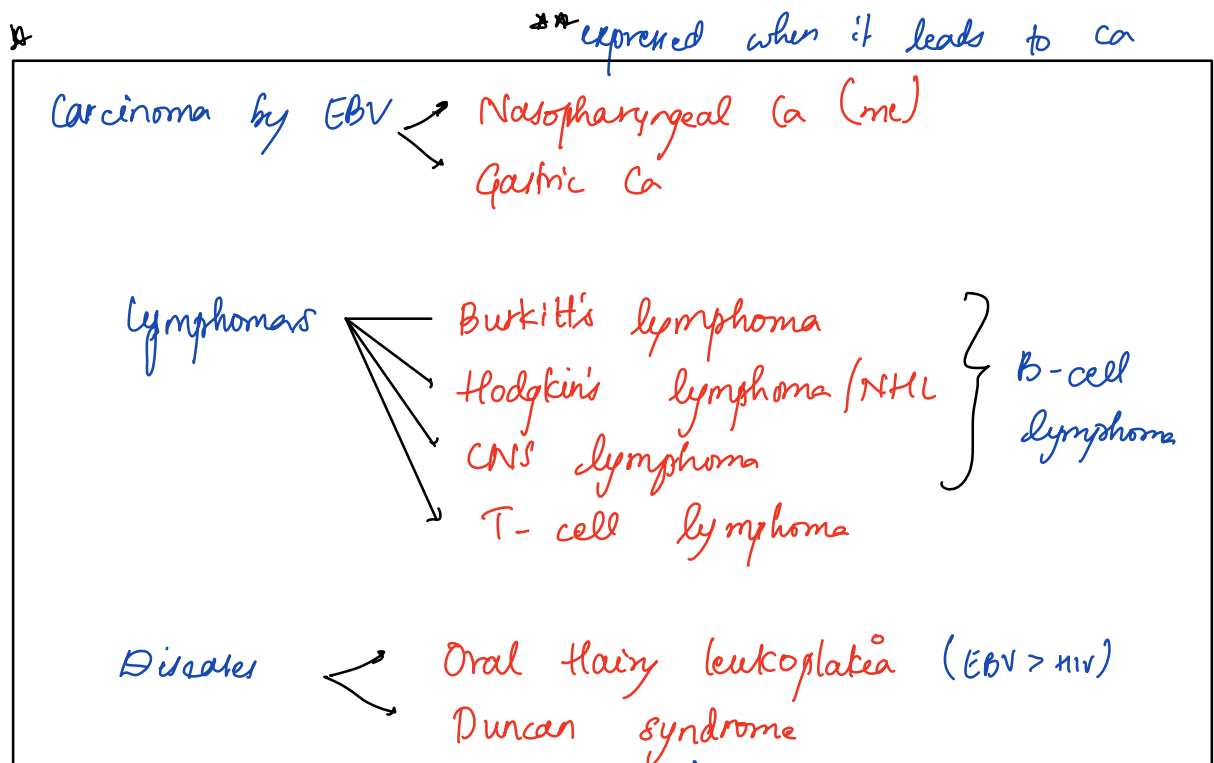
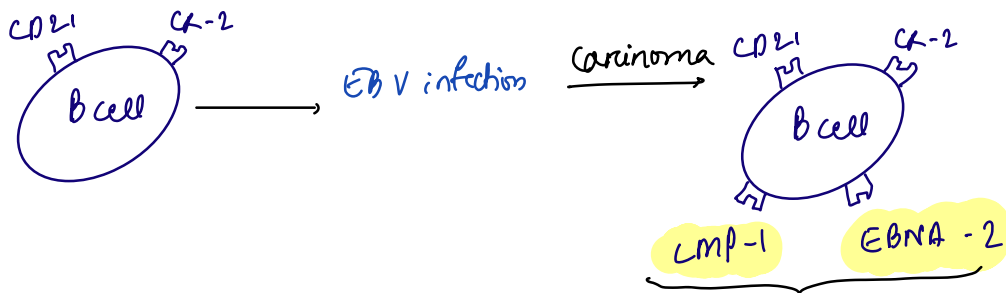
Rx: Acyclovir → Resistant → Foscarnet

EBV — Infectious mononucleosis (atypical lymphocytosis)
aka kissing disease / Glandular disease

Note: Glanders disease: *Burkholderia mallei*

C/F: pharyngitis (to diff IMN caused due to EBV & CMV)

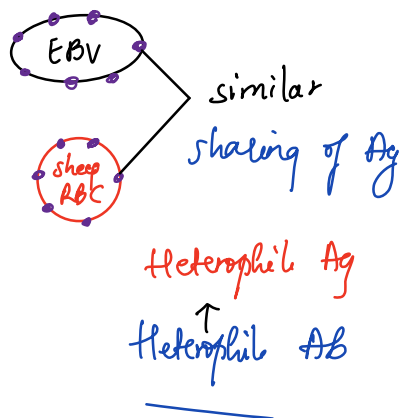
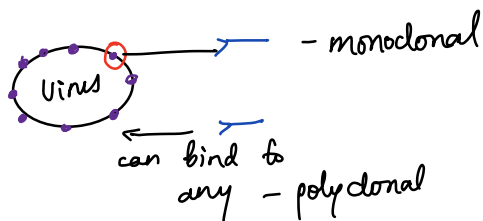
EBV mainly involve B cell $\because$ receptors on B-cell
CD-21 CR-2



Δ^{di} : Paul Bunnell test - detect EBV Abs + Sheep RBCs
Screening test

-ve in CMV IMN

Agglutination
Heterophile Agglutination Test



Confirmatory test : Differential absorption test
Monospot test (better)

Atypical lymphocytes (Downey Type 2 cells) - CD8+ Cytotoxic T cells

CMV - Infectious mononucleosis (atypical lymphocytosis)
largest herpes virus

- * No pharyngitis
- * Paul Bunnell test -ve
 - mc infection in post transplant
 - mc congenital infection (TORCH)

Ganciclovir resistance $\rightarrow$ $\therefore$ mutation in UL97 phosphotransferase gene

Pox

Variola (small pox)

only eradicated disease all over the world

May 8th, 1980

vaccine : Edward Jenner

infecting everyone & cowpox virus
($\therefore$ heterophilic antigen)

- Variola - Paschen bodies

Vaccinia (non pathogenic)

mild skin infection

- Vaccinia - Guarnieri bodies

used as vector in genetic engineering

Molluscum Contagiosum

pink color umbilicated lesions - characteristic

Serotypes: 1 - mc cause of infection

2 - can be sexually transmitted

3

4

- molluscum - HP bodies
contagiosum (Henderson Paterson)



PAPOVA

warts characteristic lesion

HPV

HPV 6, 11 - Anogenital warts [Condyloma acuminata]
HPV ^{m.c.} (16), 18 - Carcinoma cervix
31, 33, 45, 52, 58

Vaccine

- Bivalent HPV 16, 18 → Cervarix
- Quadrivalent HPV 16, 18, 6, 11 → Gardasil

Nine valent HPV 16, 18 + 31, 33, 45, 52, 58, 6, 11
(L₁ component used in vaccine)
other 5 serotypes causing carcinoma

3 doses: 0, 2, 6 months

Rx: warts - Cryotherapy
Rx is not recommended (∴ recurrence)

ADENOVIRUS

Serotypes :

1, 2, 5, 6 - RTI in children

4, 7 - RTI in adults

3, 7 - Acute follicular conjunctivitis
(keratoconjunctival fever)

Note: Acute hemorrhagic conjunctivitis - Enterovirus 70
Coxsackie A 24

11, 21 - Hemorrhagic cystitis (cyclophosphamide)

40, 41 - Diarrhoea

PARVO

only serotype inf. to humans - parvo B-19

C/F: Immunocompetent

- child - erythema infectiosum
5th disease (slapped cheek appearance)
- adults - arthralgia, arthritis

Immunocompromised — Aplastic anemia
Sickle cell anemia

Pregnant (congenital) — abortion
Non immune fetal hydrops

RETROVIRIDAE

HIV

- | HIV - 1 | HIV - 2 |
|---|---|
| <ul style="list-style-type: none">- all over the world- more virulent- 1st line 2 NRTI + 1 NNRTI | <ul style="list-style-type: none">- restricted to some places- more drug resistant- intrinsically resistant to NNRTIs- presence of gp 36 |

HIV - 1 : 4 groups M, N, O, P

M group - 9 subgroups A B C D F G H J K

HIV 1 Group M subtype C - mc worldwide

Highest risk : blood transfusion > drug abuse needle sharing
> receptive anal intercourse

mc mode of spread - sexual transmission

Structure :

structural genes

- env
- gag
- pol

codes for

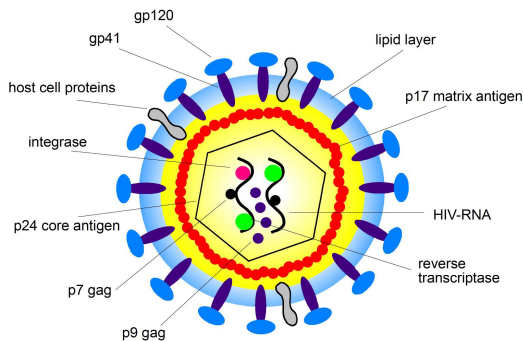
- envelope gp120, gp41
- core p24
- enzymes p31
 - reverse transcriptase
 - polymerase
 - integrase

Non structural genes

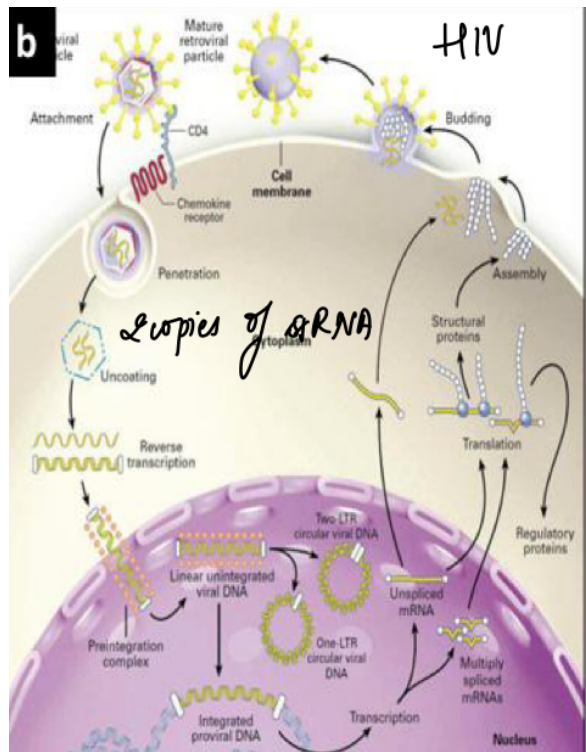
Tat, Rev, Nef, Vif, LTR, Vpr

common for both

Vpu → HIV 1
Vpx → HIV 2



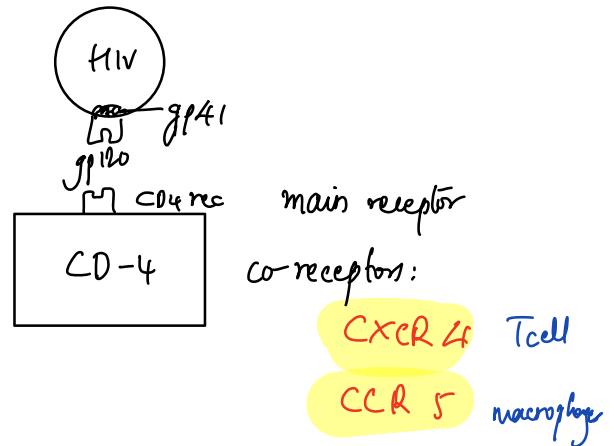
has 2 single strands of RNA
thermolabile



Replication

1) Attachment

binding via gp120
cell fusion via gp41



Note: If CCR-5 mutated → resistant to HIV

2) penetration - membrane fusion

3) Uncoating -

ss RNA ~

↓ RT

ss DNA ~

↓ polymerase

ds DNA ~

↓ integrase

integration into
host DNA



Release - budding (∴ enveloped virus)

C/f :

Early manifestation

like glandular disease

late manifestation

AIDS

WHO staging - 4 stages

Stage I

- Asymptomatic
- generalized lymphadenopathy

Stage II

- Superficial fungal infections
- Reactivation of viruses (herpes zoster)
- Aphthous ulcers in mouth

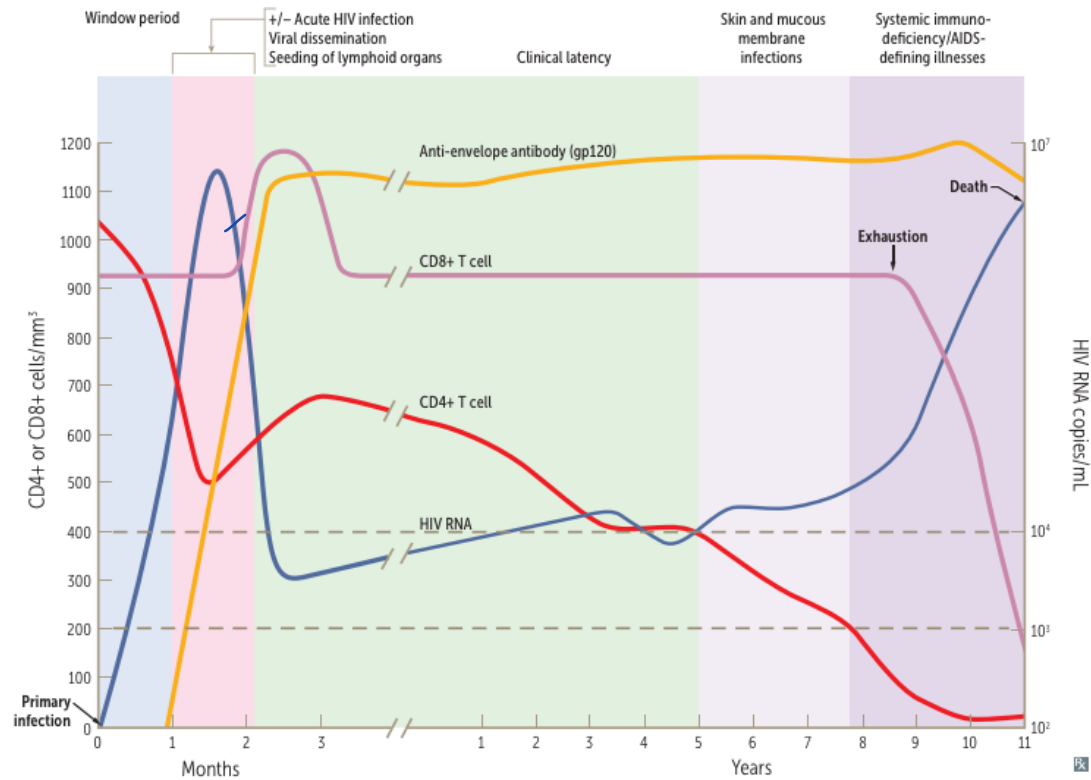
Stage III

- fever > 1 month
- diarrhoea > 1 month
- wt loss > 10%
- oral candidiasis
- pulmonary TB
- unexplained anaemia

Stage IV

- oesophageal candidiasis
- Extrapulmonary TB
- all carcinomas (Kaposi sarcoma)
- Opportunistic infections
- Wasting syndrome

Time course of untreated HIV infection



Dashed lines on CD4+ cell count axis indicate moderate immunocompromise (< 400 CD4+ cells/mm³) and when AIDS-defining illnesses emerge (< 200 CD4+ cells/mm³).

Most patients who do not receive treatment eventually die of complications of HIV infection.

Four stages of untreated infection:

1. Flu-like (acute)
2. Feeling fine (latent)
3. Falling count
4. Final crisis

During clinical latency phase, virus replicates in lymph nodes

Δ_{in} : earliest Ag to appear - p24
 earliest Ab to appear - Ab against gp41

screening test - ELISA
 confirmatory test - Western blot

Western blot tests are no longer recommended by the CDC for confirmatory testing. HIV-1/2 Ag/Ab testing is not recommended in babies with suspected HIV due to maternally transferred antibody. Use HIV viral load instead.

Treatment: HAART (Highly Active Antiretroviral therapy)

HIV 1 $\rightarrow$ 1st line: 2 NRTI + 1 NNRTI

L E T
 Lamivudine + Efavirenz + Tenofovir
 NRTI NNRTI NRTI

2nd line: 2 NRTI + PI

L L T
 Lamivudine + Lopinavir + Tenofovir
 NRTI PI NRTI

HIV 2 $\rightarrow$ 1st line 2 NRTI + PI

L L T
 Lamivudine + Lopinavir + Tenofovir
 NRTI PI NRTI

No use of NNRTI

PEP : Lamivudine + Tenofovir + Dolutegravir
1st dose $\bar{c}$ in 2hr (max $\bar{c}$ in 72 hrs)
continue for 28 days
protection rate $\sim 70\%$.

[to $\uparrow$ the conc of lopinavir in blood - Ritonavir added in low conc]

Pregnant women : L E T regimen

Window period - period in which Abi cannot be
detected in patient
3 wks to 3 months
(i.e period b/w infection & detection of Ab)

Ag test can be done - Ag test (p24 - core Ag)
- PCR

New born - Nevirapine

1^o CNS lymphoma - Steroids after Bx

HIV therapy

Antiretroviral therapy (ART): often initiated at the time of HIV diagnosis.

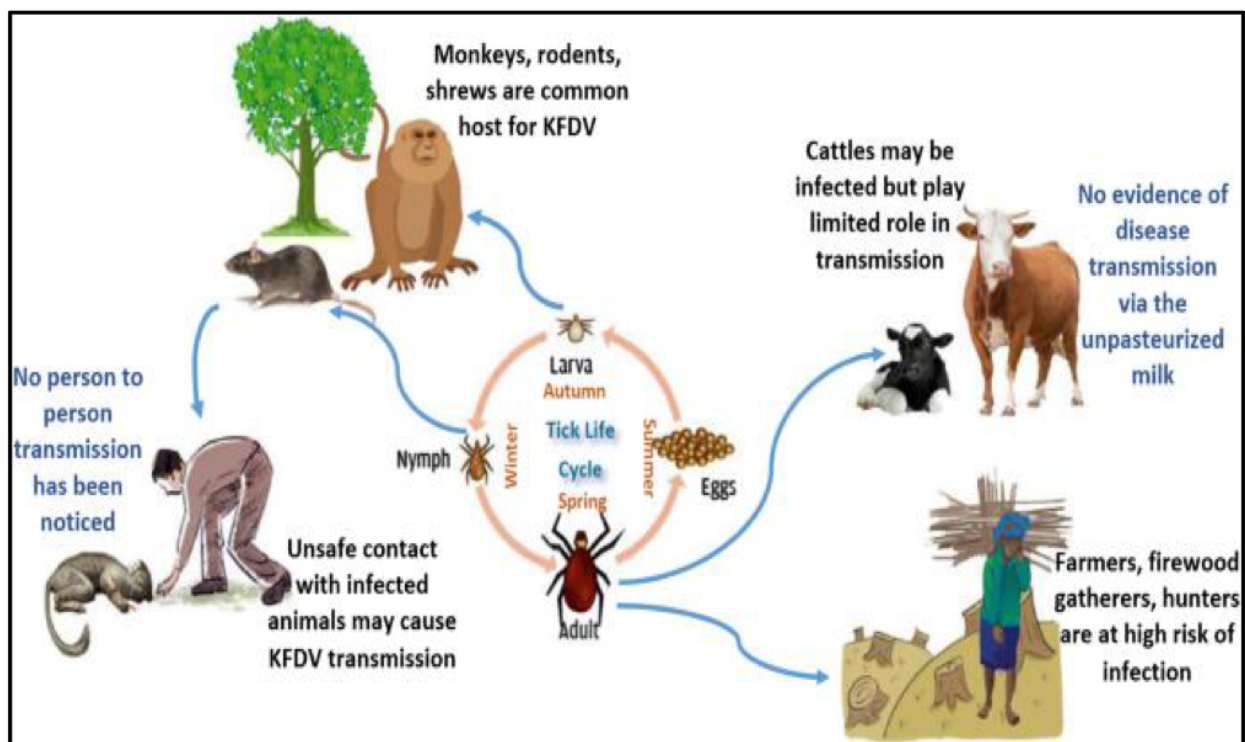
Strongest indication for use with patients presenting with AIDS-defining illness, low CD4+ cell counts (< 500 cells/mm³), or high viral load. Regimen consists of 3 drugs to prevent resistance: 2 NRTIs and preferably an integrase inhibitor.

Most ARTs are active against both HIV-1 and HIV-2 (exceptions: NNRTIs and enfuvirtide not effective against HIV-2).

DRUG	MECHANISM	TOXICITY
NRTIs		
Abacavir (ABC) Emtricitabine (FTC) Lamivudine (3TC) Tenofovir (TDF) Zidovudine (ZDV, formerly AZT)	Competitively inhibit nucleotide binding to reverse transcriptase and terminate the DNA chain (lack a 3' OH group). T enofovir is a nucleoside; the others are nucleosides. All need to be phosphorylated to be active. ZDV can be used for general prophylaxis and during pregnancy to ↓ risk of fetal transmission. Have you dined (vudine) with my nuclear (nucleosides) family?	Bone marrow suppression (can be reversed with granulocyte colony-stimulating factor [G-CSF] and erythropoietin), peripheral neuropathy, lactic acidosis (nucleosides), anemia (ZDV). Abacavir contraindicated if patient has HLA-B*5701 mutation due to ↑ risk of hypersensitivity.
NNRTIs		
Delavirdine Efavirenz Nevirapine	Bind to reverse transcriptase at site different from NRTIs. Do not require phosphorylation to be active or compete with nucleotides.	Rash and hepatotoxicity are common to all NNRTIs. Vivid dreams and CNS symptoms are common with efavirenz.
Integrase inhibitors		
Bictegravir Dolutegravir Elvitegravir Raltegravir	Inhibits HIV genome integration into host cell chromosome by reversibly inhibiting HIV integrase.	↑ creatine kinase.
Protease inhibitors		
Atazanavir Darunavir Lopinavir Ritonavir	Assembly of virions depends on HIV-1 protease (<i>pol</i> gene), which cleaves the polypeptide products of HIV mRNA into their functional parts. Thus, protease inhibitors prevent maturation of new viruses. Ritonavir can "boost" other drug concentrations by inhibiting cytochrome P-450. Navir (never)tease a protease.	Hyperglycemia, GI intolerance (nausea, diarrhea), lipodystrophy (Cushing-like syndrome). Rifampin (potent CYP/UGT inducer) reduces protease inhibitor concentrations; use rifabutin instead.
Entry inhibitors		
Enfuvirtide	Binds gp41, inhibiting viral entry.	Skin reaction at injection sites. Enfuvirtide inhibits fusion .
Maraviroc	Binds CCR-5 on surface of T cells/monocytes, inhibiting interaction with gp120.	Maraviroc inhibits docking .

KYASANJUR FOREST DISEASE

- Reservoir : Rats & squirrels
- Amplifier : Monkey
- Vector : Tick (Hard tick > soft tick)
- Vaccine : Killed



JAPANESE ENCEPHALITIS

- Reservoir : Birds (herons & ducks)
- Amplifier : Pig
- Vector : Culex mosquito → Filarialis, JE
- Vaccine : [
 - live : strains used : JA 14-14-2
 - killed : Nakayama & Beijing strains

incubation period - 15 days

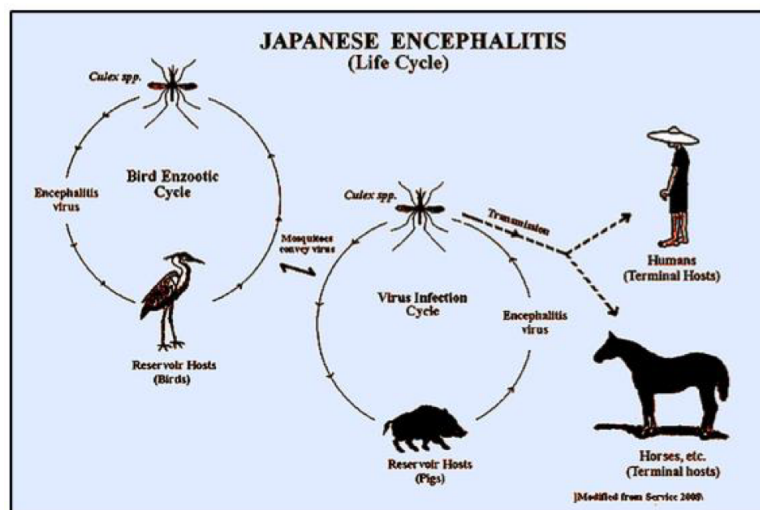
Age group - upper limit is 15 yrs

Apparent : Inapparent infection - 1:1000
no clinical manifestation

∴ difficult to eradicate

Gorakhpur in UP - epidemic going on

In case of polio → 1:1000 : 75
↓ ↓
 adults children



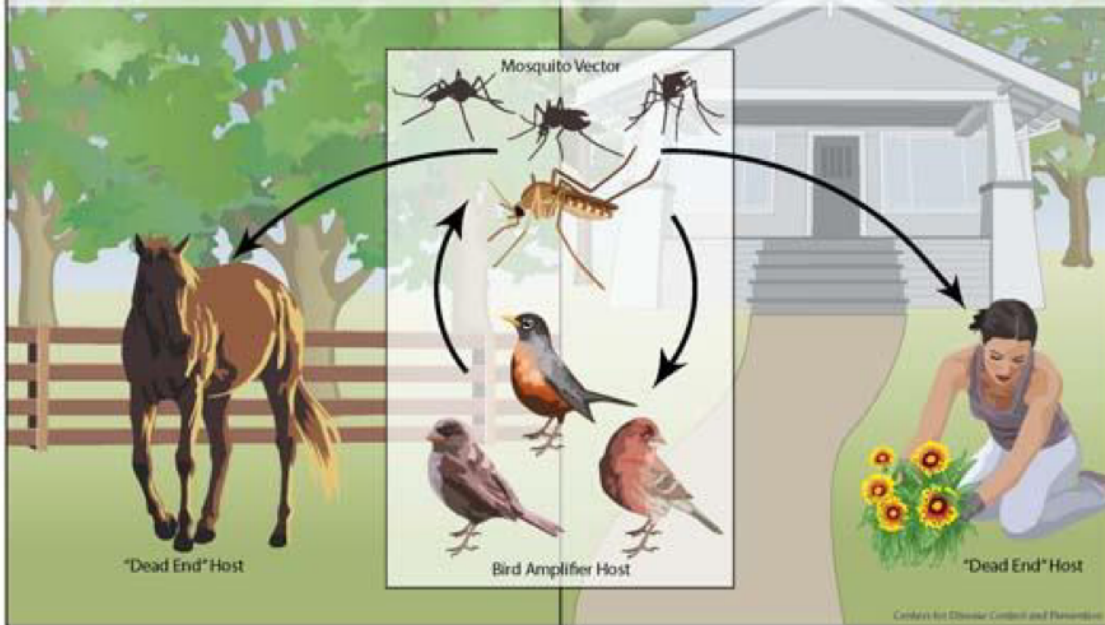
1. JAPANESE ENCEPHALITIS- Vector- *Culex* spp.; Natural reservoir: Herons, egrets, stork and cranes; Amplifying reservoir-pigs

2.WEST NILE VIRUS- Vector- Culex spp.; Reservoir- CROWS, SPARROWS AND JAYS; Man & horse are dead end hosts

West Nile Virus Transmission Cycle

In nature, West Nile virus cycles between mosquitoes (especially *Culex* species) and birds. Some infected birds, can develop high levels of the virus in their bloodstream and mosquitoes can become infected by biting these infected birds. After about a week, infected mosquitoes can pass the virus to more birds when they bite.

Mosquitoes with West Nile virus also bite and infect people, horses and other mammals. However, humans, horses and other mammals are 'dead end' hosts. This means that they do not develop high levels of virus in their bloodstream, and cannot pass the virus on to other biting mosquitoes.



DENQUE

- vector $\begin{cases} \text{Aedes aegypti} \\ \text{Aedes albopictus (new vector)} \end{cases}$
- vaccine : live **CYD - TDV**
(not licensed in India)

used in endemic places prevalence $> 10\%$.

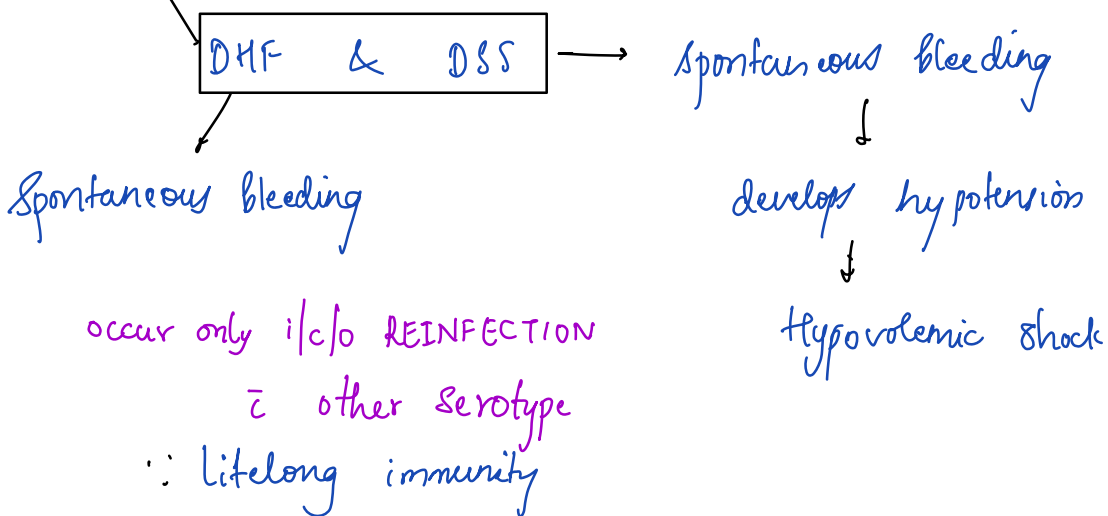
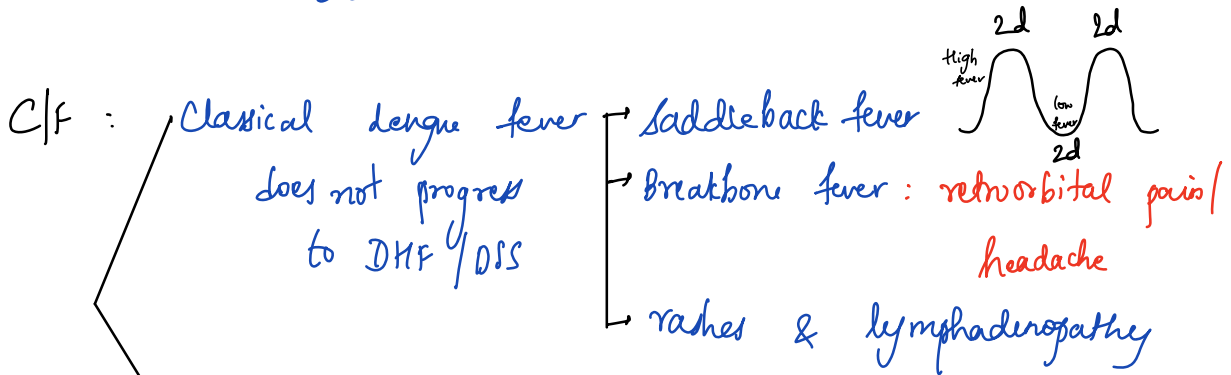
Serotypes : DEN 1

DEN 2 — most virulent

DEN 3

DEN 4

DEN 5



Reinfection $\bar{c}$ other serotype



Antigenic sin (Abs act against different Ag)



Mechanism: **Antibody dependent enhancement**



Exaggerated immune response



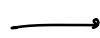
DHF & DSS

Δ^{14} : Ag detection - NS-1 (highly specific)
(Non-structural proteins) } ELISA

Ab detection - IgM (highly sensitive)

not very specific $\therefore$ cross reactivity

Gold standard
(highly sensitive &
highly specific)



Neutralization Test

(not 2oc)

Gold std for all viruses

YELLOW FEVER

- endemic in Central & South America
- Incubation period - 6 days
($\therefore$ if not vaccinated - quarantine for 6 days)
- vector - Aedes
- vaccine line $\rightarrow$ 17D strain
- killed $\rightarrow$ Dakar strain

Diseases not seen in India - • Yellow fever

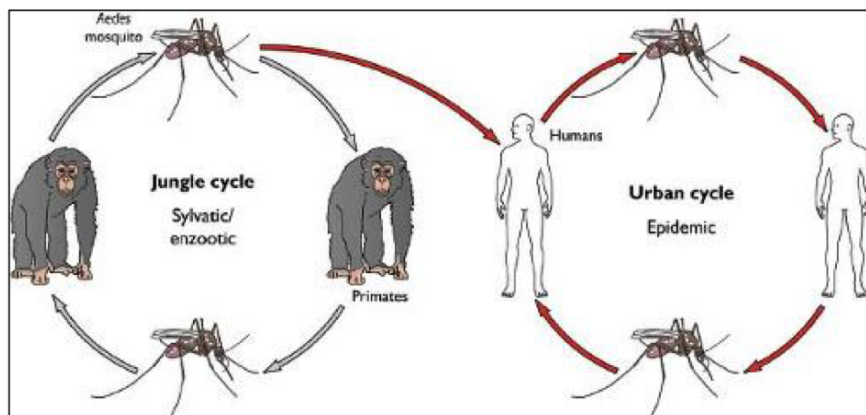
Filoviridae

- Ebola
- Marburg

⁹
Reference centres for yellow fever in India :

- National Institute of Virology Pune
- Central Institute, Kasauli

3. DENGUE & YELLOW FEVER TRANSMISSION CYCLE - Vector- Aedes aegypti, A. albopictus;
Reservoir-Non-human primates



- Vector - Aedes } Adult - GBS
- sexual transmission }
- vertical transmission → Newborn - Microcephaly (not always ⊕)

no cases for long time

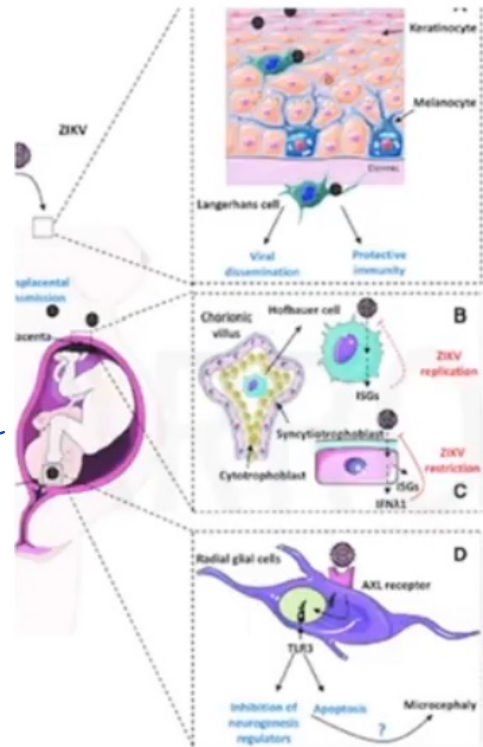
This type of occurrence : Re-emergence

- attaches to epidermal keratinocytes & dermal fibroblast

Vertical transmission $\rightarrow \therefore$ Zika affects Hoffbauer cells

Fetal manifestations of Zika virus

- Loss of brain matter
- Cortical atrophy
- Microcephaly
- Ventriculomegaly
- Ocular symptoms
- ↑ Tone of limbs
- Congenital limb contracture

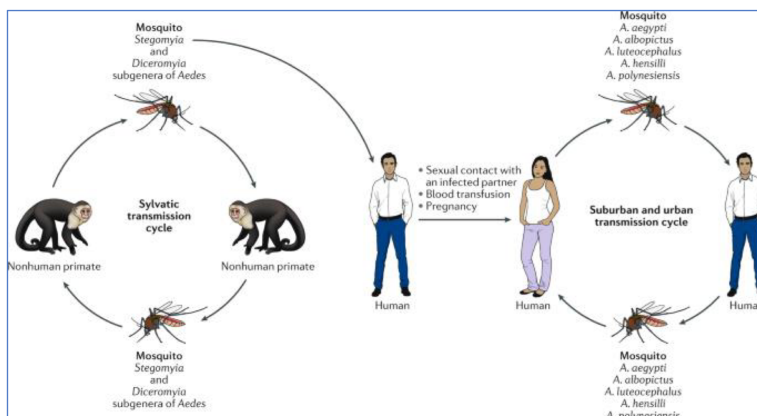


Q

Not
petechial
rash



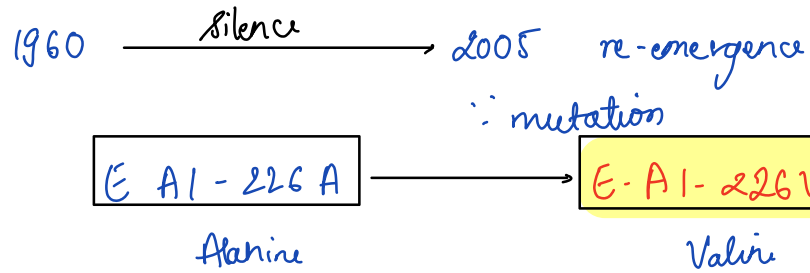
4. ZIKA VIRUS- Vector- *Aedes aegypti*, *A. albopictus*



CHIKUNGUNYA

Vector - Aedes

Vaccine - No vaccine



Aedes aegypti

spindle shape
no stripes on ventral part



Aedes albopictus

cylindrical shape
stripes (+) on ventral part

Rash on Day 1

RUBELLA

GERMAN MEASLES

Vaccine - Live RA 27/3 strains

C/F: adults

Forscheimer spots

petechiae on soft palate



Congenital

Congenital Rubella syndrome (1st Trimester)

- salt & pepper retinitis > Cataract > Glaucoma
- SNHL
- PDA

Transient congenital infections (3rd trimester)
only petechial spots

also seen in

CMV, Parvovirus B19.
AML, Neuroblastoma
Rh hemolysis

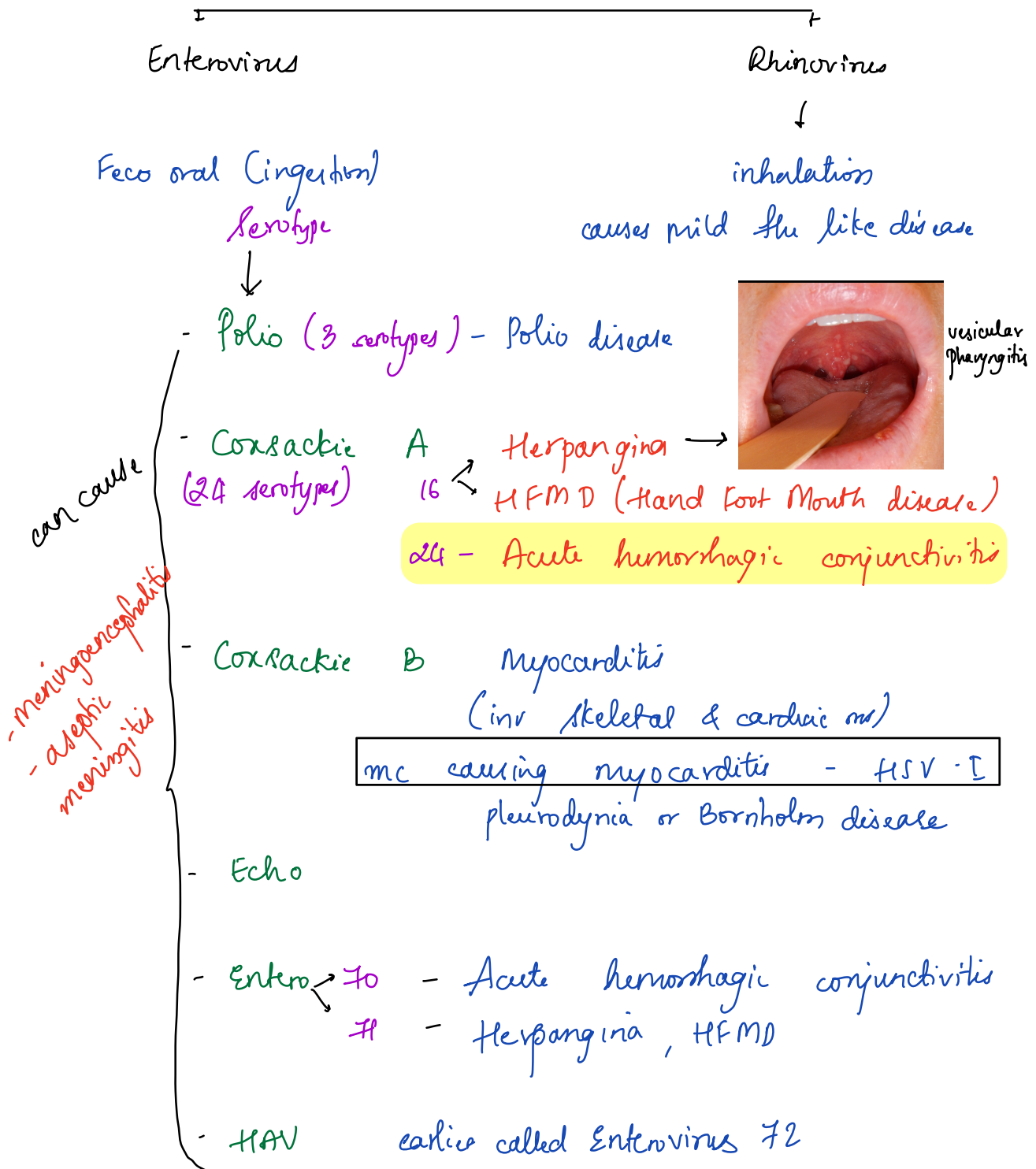
Blueberry muffin syndrome

∴ dermal
erythropoiesis



afw microphthalmos

PICORNA



mc organisms causing meningoencephalitis - Enterovirus

" sporadic encephalitis
" temporal lobe " } HSV-1
" mollaret's meningitis

" amoebic encephalitis - Acanthamoeba

" amoebic meningoencephalitis - Naegleria

" brainstem encephalitis - Rabies

Polio

Type 1 - mc to cause paralysis (wild virus)

Type 2 - in vaccine mutation VDPV - Vaccine Derived Polio virus

Type 3 - mc to cause paralysis (vaccine virus)
VAPP - Vaccine associated paralytic polio

Vaccine - Bivalent Type 1 & 3

∴ mutation

↓ no cases since 1999 due to Type 2

→ live vaccine - OPV (Sabin)

→ killed vaccine - IPV (Salk)

Earliest change - degeneration of Nissl bodies

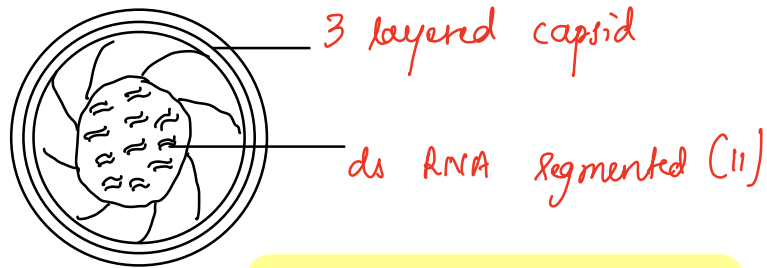
ROTA

Reoviridae

ds RNA

linear segmented (11)

non enveloped
icosahedral



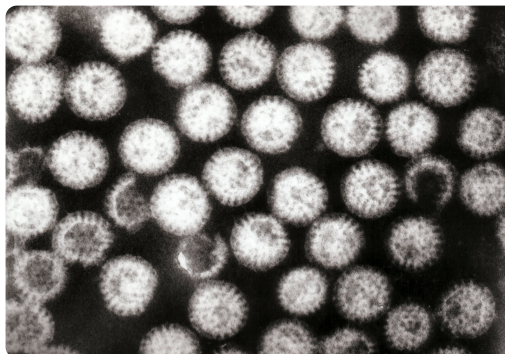
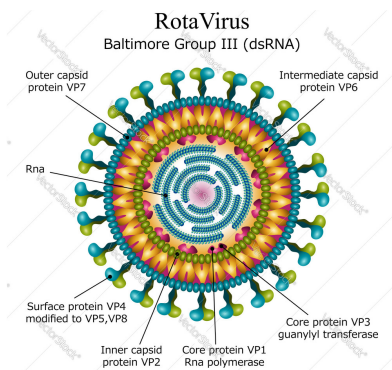
CART WHEEL APPEARANCE

mc serotype in India & world → G1P (8)

C/F : children diarrhoea

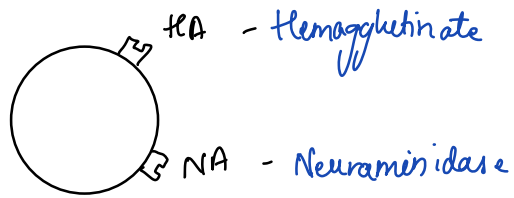
vaccine : live → oral

no complications (previously: intussusception)



ORTHOMYXOVIRIDAE

Influenza



Serotype:

Influenza A (mc) : H_1N_1 - (swine flu) pandemic flu }
 H_2N_2 - seasonal flu } PNEUMONIA → pandemic
 H_5N_1 - avian flu } → endemic

B : — PNEUMONIA → epidemic only
GASTROENTERITIS

C (no cases)

pandemic

— due to Antigenic shift
(sudden change due to genetic reassortment)
occurs in every 10-15 yrs

epidemic

— due to Antigenic drift
(gradual change due to point mutation)
occurs every 2-3 yrs

Δ^{vis} :

Ag	-	HA	Ag	-	highly specific	} ELISA
Ab	-	IgM	Ab	-	highly sensitive	
Gold std	-	Neutralization test				

Vaccine : trivalent live - nasal spray

Inf A H_1N_1
 Inf A H_3N_2
 Inf B

killed - im

Now - quadrivalent 2 Inf A 2 Inf B

Antigenic variation :

- Antigenic shift (genetic reassortment) 10-15 yrs
- Antigenic drift (point mutation) 2-3 yrs

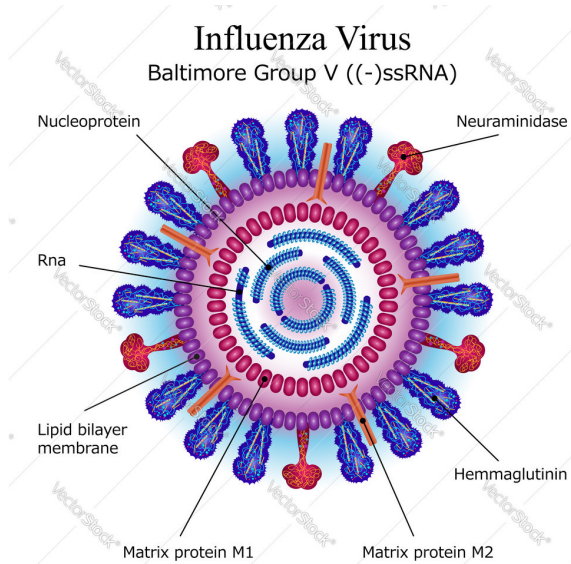
Rx: Neuraminidase inhibitors

Osetamavir (oral) 75mg Zanamavir (inhalational) 10mg	Rx 80 x 5 days prophylaxis OD x 7 days
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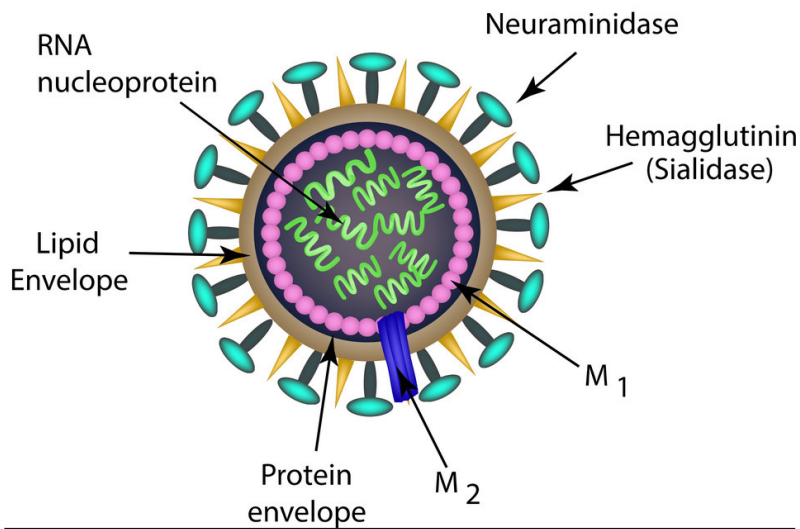
Von magnus phenomenon - error during assembly
 (daughter viruses formed w/ host cell nucleic acid)

No infection

Sample: Use in titres of Influenza virus



8 segments



- high risk <5 yrs >60 yrs
- virus shedding ⊕ before symptoms begin
- Aquatic birds act as reservoir
- 2° attack rate 5-15%

PARAMYXOVIRIDAE

- parainfluenza, mumps
- Measles RSV, Castle disease virus
- Nipah

Parainfluenza (HA, NA) → **Croup syndrome**
laryngotracheobronchitis

Mumps (HA, NA) → Salivary glands - **parotid gland (mc)** (submandibular, sublingual also)
aseptic meningitis - before, during, after parotid inv.
Gonads - oophoritis / orchitis
mc complication - meningoencephalitis

Measles (HA, G spike) (Koplik spots) → **Otitis media mc**
SSPE (late complication) - Subacute Sclerosing Panencephalitis
Giant cell pneumonia in immunocompromised (Hutchinson's pneumonia)

RSV (F, G spike) → Bronchiolitis in children < 2 yrs
Rx: inhaled ribavirin

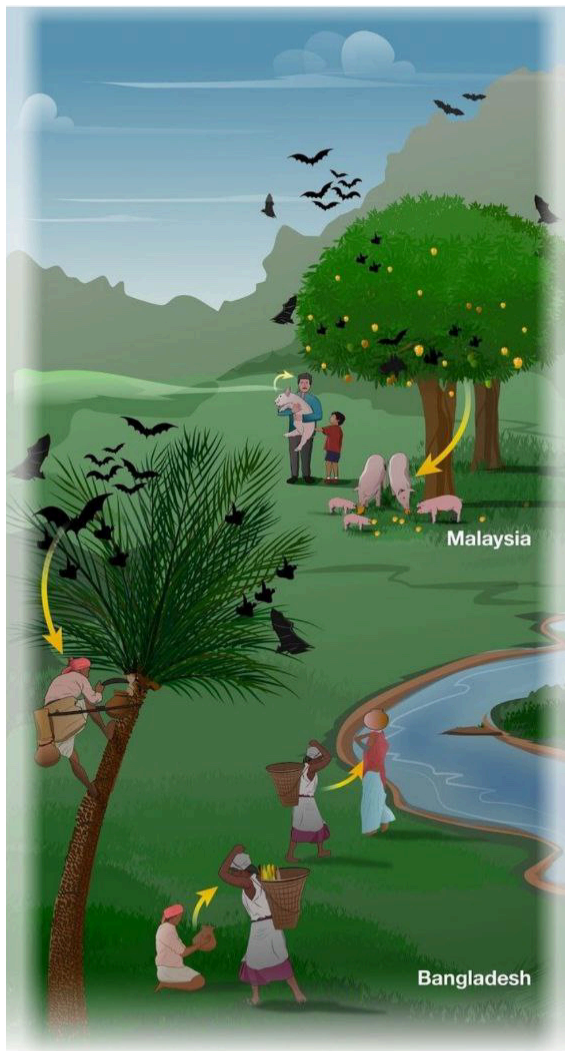
New castle disease virus → New castle disease

Nipah — Encephalitis outbreak

1st case - Malaysia

India → 2001, 2007 West Bengal
2018 in Kerala

reservoir - fruit bat [reservoir for < Nipah Ebola >]



The image depicts the life cycle of the Nipah virus.

Salient points on Nipah virus disease:

- It is an RNA virus which belongs to family **Paramyxoviridae**, genus henipavirus.
- It is a zoonotic virus.
- Natural host: **Fruit bats** of the Pteropodidae family
- Transmission:
 - Animals (bats, pigs) to humans
 - Human-to-human
- Clinical features: Asymptomatic infection (subclinical), acute respiratory infection, fatal encephalitis.
- There is no treatment or vaccine available for either people or animals. The primary treatment for humans is supportive care.
- Nipah virus is on the WHO list of Blueprint priority diseases.

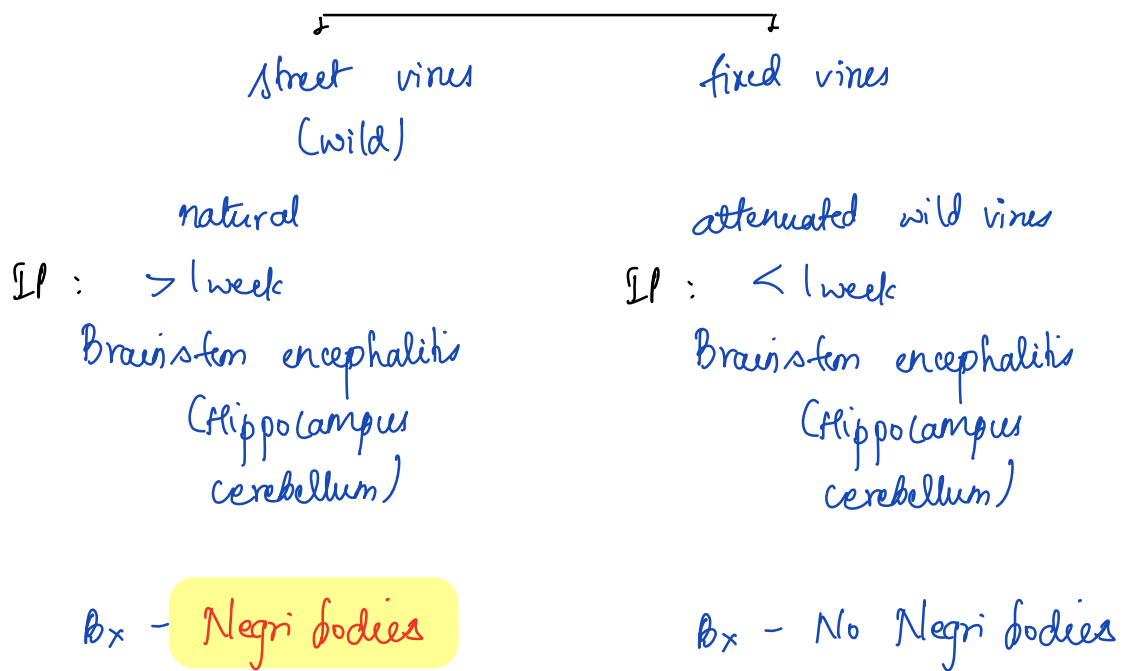
RHABDOVIRIDAE

Rabies → No viremia

mc mode of transmission in India - Dog bite
world - Bat

Rabies vaccine not recommended for rat bite

Rabies viruses



CF :

- Encephalitis rabies - anxiety, hyperexcitability, hydrophobia
- Paralytic rabies - paralysis
seen in partial vaccination

Δ^{ris} • Antemortem - Saliva , CSF , Corneal smear
 Skin from nape of neck (best)
 ($\because$ high nerve endings present here
 in rabies - no viremia , travel through nerves)

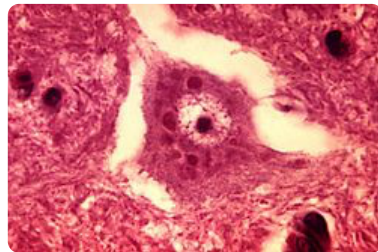


Direct immunofluorescence Assay > PCR

or

Viral culture from corneal scrapings used to confirm Dx

• Postmortem - brain bx Negri bodies
 (80% cases)



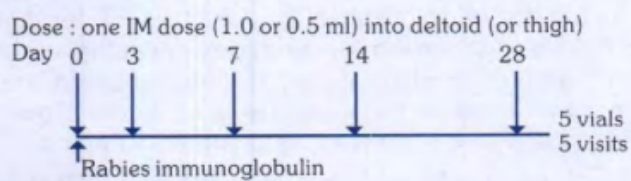
Treatment : Local - • wash $\bar{c}$ running tap water (15 mins)
 • apply antiseptic
 • Immunoglobulin $\begin{cases} \text{ERIG (mc used)} \\ \text{HRIG} \end{cases}$
 • Do not suture the wound

The dose of human rabies immunoglobulin is 20 IU/kg body weight; for equine immunoglobulin and F(ab')₂ products, it is 40 IU/kg body weight.

Systemic - • Immunoglobulin im
 • vaccine

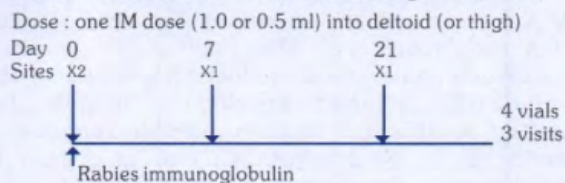
Categories of contact with suspect rabid animal	Post-exposure prophylaxis measures
Category I – touching or feeding animals, licks on intact skin	None
Category II – nibbling of uncovered skin, minor scratches or abrasions without bleeding	Immediate vaccination and local treatment of the wound
Category III – single or multiple transdermal bites or scratches, licks on broken skin; contamination of mucous membrane with saliva from licks, contacts with bats.	Immediate vaccination and administration of rabies immunoglobulin; local treatment of the wound

- (i) *Essen regimen*: the 5-dose regimen prescribes 1 dose on each of days 0, 3, 7, 14, and 28; as shown below



- (ii) *Zareb regimen*: the 4-dose abbreviated multisite regimen prescribes 2 doses on day 0 (1 in each of the 2 deltoid or thigh sites) followed by 1 dose on each of days 7 and 21, as shown below.

Abbreviated multisite intramuscular regimen (2-1-1)

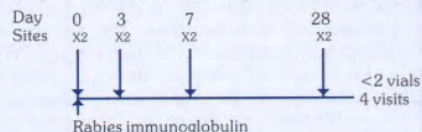


Intradermal administration for post-exposure prophylaxis

The 2-site regimen prescribes injection of 0.1 ml at 2 sites (deltoid or thigh) on days 0, 3, 7 and 28. The day 14 dose is missed. This regimen may be used for people with category II and III exposures in countries where the intradermal route has been endorsed by national health authorities. The regimen is as shown below:

2-site intradermal regimen (2+2+2+0+2)

Dose : one ID dose = one fifth of IM dose (0.1 ml) ID per site



prophylaxis :

Post exposure prophylaxis

	im	0	3	7	14	28
Esken regimen		1	1	1	1	1
	id					
Redcross regimen		2	2	2	-	2

Pre exposure prophylaxis (im)

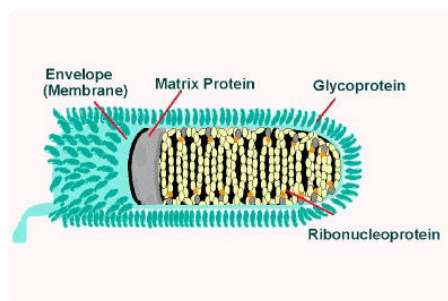
1 - 1 - 1

post exposure prophylaxis in a
pre vaccinated (im)

1 1

According to WHO latest - only 4 doses -
remove 28th day dose

Milwaukee protocol - Rx - Therapeutic coma



Bullet shaped virus

FILOVIRIDAE → Ebola
Marburg

not seen in India

Ebola

Subtypes (5)

- | | |
|-------------------------|------------------------------|
| 1) Zaire - 90% fatality | } causes disease
in human |
| 2) Sudan | |
| 3) Bundibugyo | |
| 4) Taïforest | |
| 5) Reston | |



IP : 2 to 21 days

mode of transmission - Fruit bat

- reservoir is **fruit bat**

- Transmission : contact & secretions

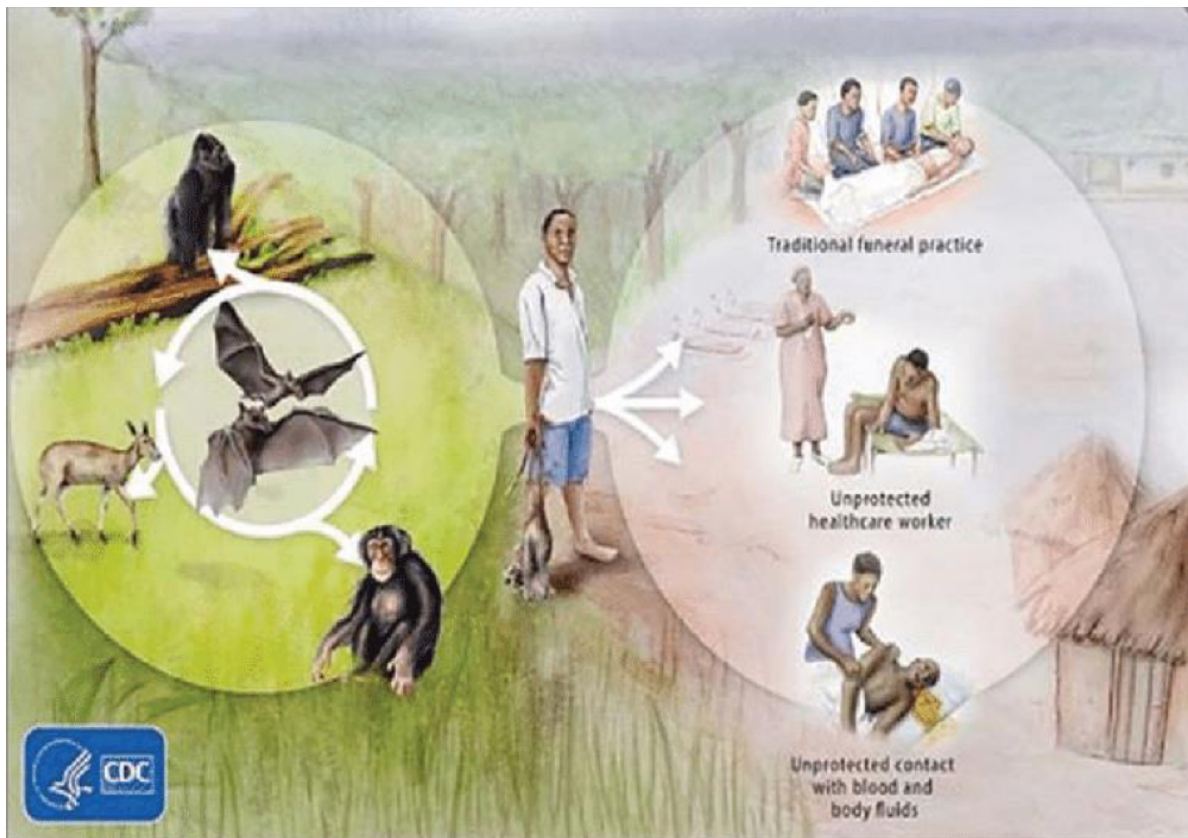
consumption of infected animal meat
touching objects

vaccine : rVSV - ZEVOR (under trial)

Note: Hemorrhagic diseases:

Dengue
Ebola
CCF
Yellow fever
Hanta virus

EBOLA

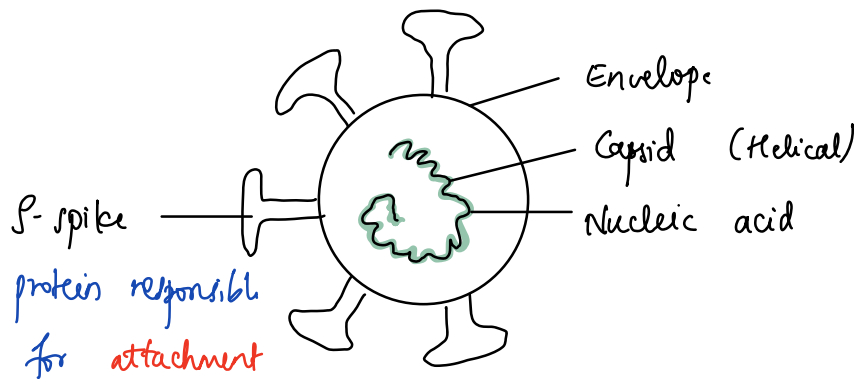


CORONAVIRIDAE

SARS - 2003

MERS - 2012

SARS COV-2 - 2019

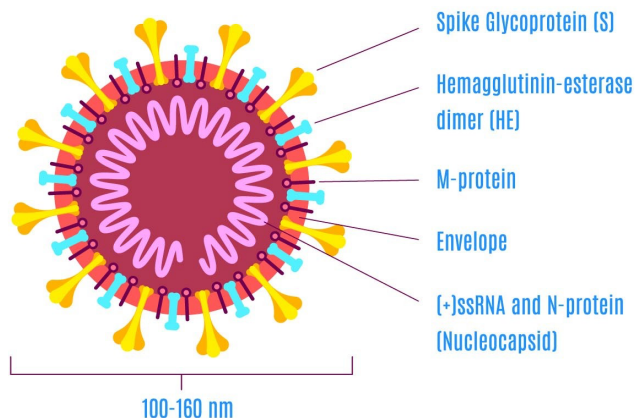


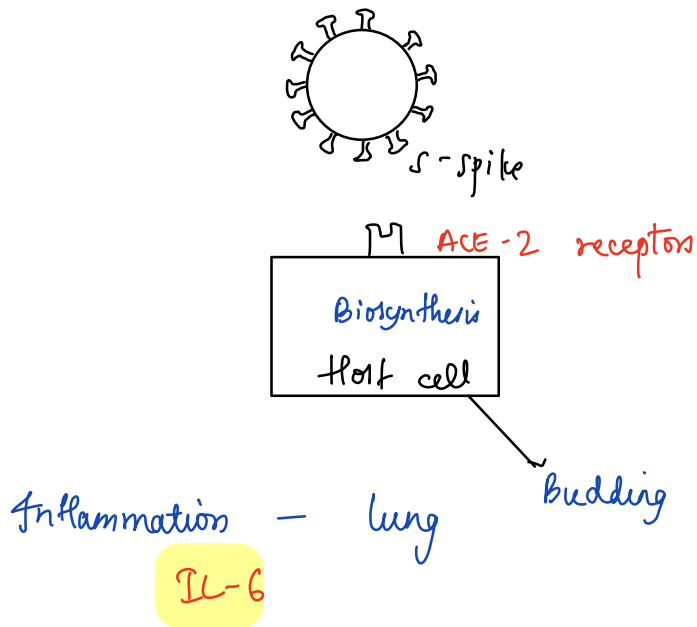
Other receptors

- Envelope proteins
- Matrix proteins
- Hemagglutinin esterase

Corona - shape due to S spike
(flower)

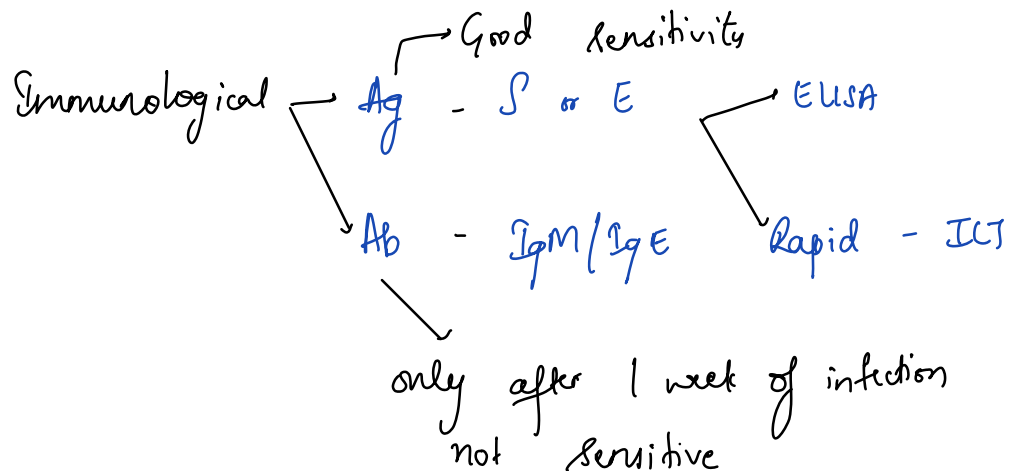
Coronavirus structure





C/F: Pneumonia $\rightarrow$ ARDS $\rightarrow$ Death

Δ^{si} : Molecular RT-PCR Gene $\leftarrow$ S spike
(good sensitivity) E protein



Rx: Entry inhibitor - HCQ

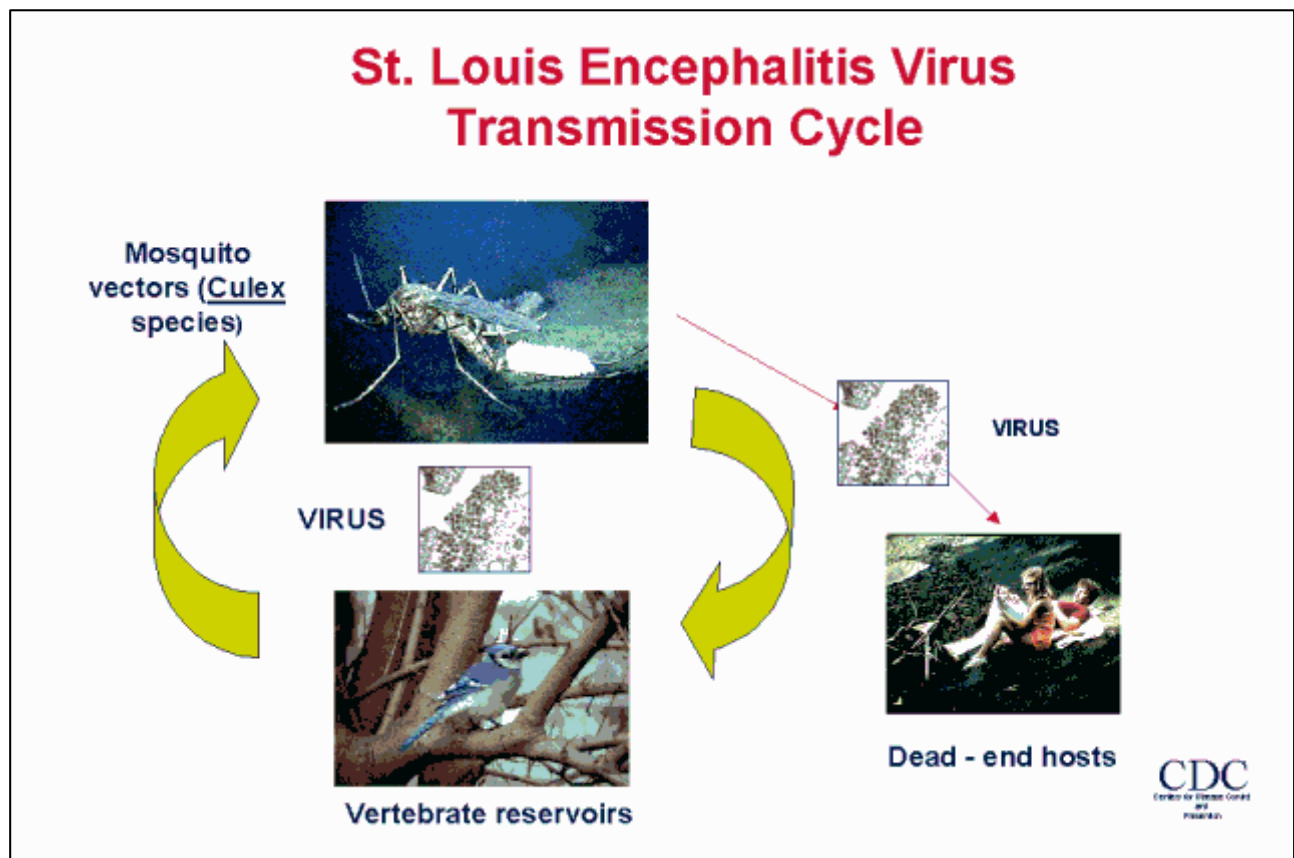
RNA
dependent
RNA pol
inhibitor { polymerase inhibitor (biogenesis) - Favipiravir
Adenosine analogue - Remdesivir

Guanosine analogue - Ribavirin

Immunomodulators - Corticosteroid
HCQ

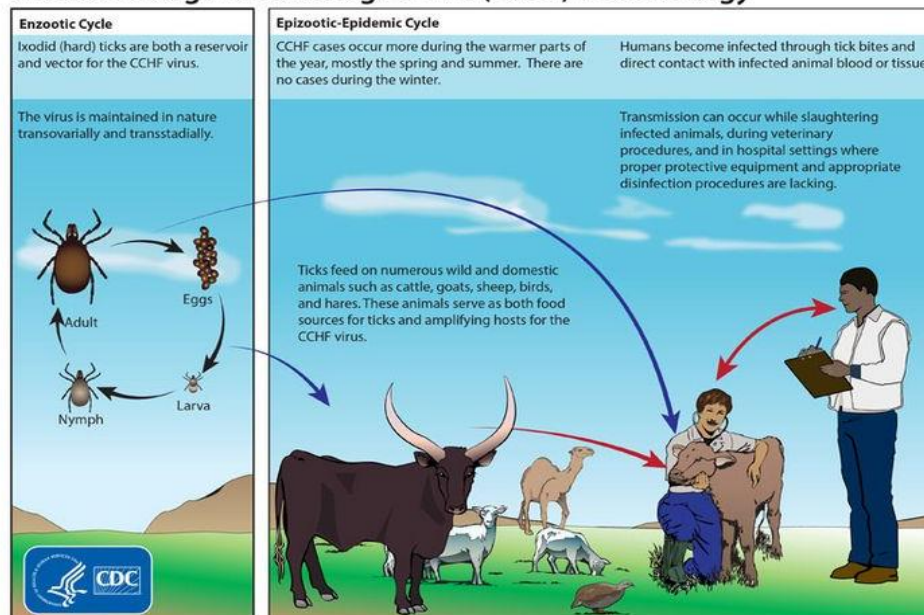
Vaccine - targeting S-protein

5.SLEV- **Vector-Culex spp.;** **Reservoir- house sparrow, pigeon, blue jay, and robin**



6.CCHF- **Vector-Hard tick (Ixodid spp.); Reservoir- cattle, goats, sheep and hares.**

Crimean-Congo Hemorrhagic Fever (CCHF) Virus Ecology



Microbiology Prep
by Dr. Shivika