Antiretroviral Therapy: Cheat Sheet

by Carm (Carmilaa) via cheatography.com/49544/cs/16182/

CDC Stage of HIV Disease

-Stage I: Acute HIV infection
-Stage II: Asymptomatic HIV
-Stage III: Early Symptomatic HIV
-Stage IV: Late Symptomatic HIV
= A Constitutional Disease
= B Neurological Disease
= C Secondary Infections
-C1 AIDS defining
-C2 Other infections
= D Secondary Cancers
= E Other Conditions

Clinical Staging of Oral Manifestations of HIV

Stage:	Adults and Adolescents (>15yo)	Children (<15yo):
1	No disease	No disease
2	Angular Chellitis, Recurrent oral ulcerations	Angular Chellitis, Linear gingival erythema, extensive warts, Recurrent oral ulcerations, Parotid enlarge
3	Persistent oral candidiasis, Oral hairy leukoplakia, Acute necrotizing ulcerative stomatitis, gingivitis, periodontitis	Persistent oral candidiasis (after 8wks), Oral hairy leukoplakia, Acute necrotizing ulcerative gingivitis or periodontitis.
4	Chronic (>1mo) orolabial HSV, Kaposi's sarcoma,	Chronic (>1mo) orolabial HSV, Karpo's Sarcoma, Non-Hodgkin's lymphoma

HIV-related Oral Lesions:

Infections:	- Fungal, Viral, Bacteria
Neoplams:	- Kaposi's Sarcoma, Non-Hodgkin's Lymphoma
Other:	- Aphthous-like Ulcers, Lichenoid or drug reactions, Salivary Gland Disease

Oral Candidiasis:

Erythematous Chelitis

Pseudomembranous

Angular

Oral Ulcers:

-Herpes Simplex Infection	-HPV Lesiosn
-Cytomegalovirus Infection	-Lymphoma
-Aphthous Ulcers	-Necrotizing ulcerative gingivitis/ period-ontitis
-Histoplasmosis	-Necrotizing Stomamtitis (NS)

There are many different causes of oral ulceration in patients with HIV infection = Herpes simplex infection, Varicella zoster infection. Accurate diagnosis and appropriate management of oral ulceration in patients with HIV infection generally result in complete healing of the ulceration.

Aphthous Lesions Clinical Types

Topical Therapy:	Intralesional:	Systemic Therapy:
Topical Corticost-eroids	Triamcinolone: 40 mg /ml (0.5 ml-1.0 ml injected bid)	Prednisone: 0.5-1.0 mg/kg qd x 7-10d, then taper
		Thalidomide: 200 mg PO qd

Antiretroviral Cancer:

NRTIs:	Nucleoside OR Nucleotide Reverse Transcriptase Inhibitors (Nukes)
NNRTIs:	Non-nucleoside Reverse Transcriptase Inhibitors (non-n-ukes)
PIs:	Protase Inhibitors

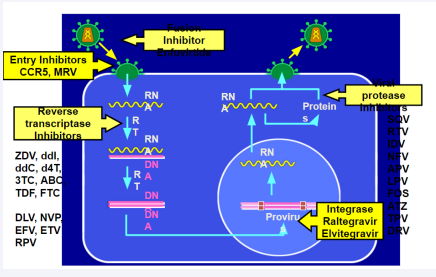
Fusion Inhibitors

Chemokine Receptor Antagonists

Integrase Inhibitors

See Life cycle of HIV

Current Antiretroviral Targets



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Reverse Transcriptase Inhibitors:

Nucleoside Analogues Zidovudine (AZT, ZDV) Didanosine (ddI) Zalcitabine (ddC) Stavudine (d4T) Lamivudine (3TC) Abacavir (ABC) Emtricitabine (FTC)

Non-nucleoside analogues: Nevirapine (NVP) delavirdine (DLV) Efavirenz (EFV) etravirine (ETV) rilpivirine (RPV)

Nucleotide analogue
Tenofovir (TFV)

Protease Inhibitors:

saquinavir (SQV)
ritonavir (RTV)
indinavir (IDV)
nelfinavir (NFV)
amprenavir (APV)
lopinavir (LPV)
fosamprenavir (FPV)
atazanavir (ATV)
tipranavir (TPV)
darunavir (DRV)
dolutegravir (DTG)

Reverse Transcriptase Inhibitors:

Integrase Inhibitors	Fusion Inhibitor:	Entry Inhibitors:
raltegravir (RAL)	fuzeon (T20)	maraviroc (MVC)
elvitegravir (ELV)		

NRTIs Mechanism of Action:

Nucleoside Analogs (like AZT):	Analog of thymidine, cytosine or guanine
	Triphosphorylated inside lymphocytes to active compound.
	Incorporate into growing HIV viral DNA strand by reverse transcriptase.
Nucleotide Analogs:	tenofovir (TDF)
	does NOT need to be tri-phosphorylated only di-phosphorylated to activate compound.

After incorporation of NRTIs, viral DNA synthesis will be terminated.

Non-nucleoside Reverse Transcriptase Inhibitors:

Agents directly bind to reverse transcriptase to inhibit transcription.

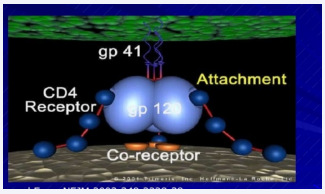
NNRTIs do not require phosphorylation to be active.

Protease Inhibitors (PIs) MOA:

Protease enzyme cleaves HIV precursor proteins into active proteins that are needed to assemble a new, mature HIV virus.

PIs bind to protease preventing the cleavage and inhibiting the assembly of new HIV viruses.

Fusion Inhibitor:



Chemokine Receptor Antagonists:

Marviroc (Selzentry)

CCR5 or CXCR4 receptors on cell surface

Virus will bind to one of the 2 receptors (some pt virus will bind to either receptors)

Marviroc blocks viral entry at CCR5

Dosed 300mg BID= 150mg BID with P450 inhibitors. = 600mg BID with P450 inducers

Integrase Inhibitors

Raltegravir (Isentress)

Dosed = 400mg BID (1tab BID)

No induction or inhibition on CYP450 enzymes or Pgp

Metabolized by UGT1A1 (glucuronidation) = Only affected by drugs that inhibit or induce UGTs (ie. rifampin)