

Antiretroviral Therapy: Cheat Sheet

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

Oral Ulcers:

Infection

Topical

Therapy: Topical

Corticost-

eroids

-Herpes Simplex

-Cytomegalovirus

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

Infection	
-Aphthous Ulcers	-Necrotizing ulcerative gingivitis/ period- ontitis
-Histoplasmosis	-Necrotizing Stomamtitis (NS)

Aphthous Lesions Clinical Types

Intralesional:

Triamcinolone: 40 mg/ml

(0.5 ml-1.0 ml injected bid)

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.

Systemic Therapy:

Prednisone: 0.5-1.0

taper

mg/kg qd x 7-10d, then

Thalidomide: 200 mg PO

-HPV Lesiosn

-Lymphoma

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 ulcera- tions, Parotid enlarge
3	Persistent oral candidiasis, Oral hairy leukoplakia, Acute necrotizing ulcerative stomatitis, gingivitis, period- ontitis	Persistent oral candidiasis (after 8wks), Oral hairy leukoplakia, Acute necrot- izing ulcerative gingivitis or periodontitis.
4	Chronic (>1mo) orolabial HSV, Kaposi's sarcoma,	Chronic (>1mo) orolabial HSV, Karpo's Sarcoma, Non-Hodgkin's lymphoma

Antiretroviral Cancer:

NRTIs: Nucleoside OR Nucleotide Reverse Transcriptase

Inhibitors (Nukes)

NNRTIs: Non-nucleoside Reverse Transcriptase Inhibitors (non-n-

ukes)

Pls: Protase Inhibitors

Fusion Inhibitors

Chemokine Receptor Antagonists

Integrase Inhibitors

See Life cycle of HIV

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

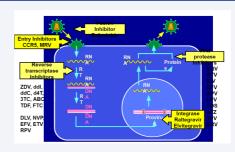
Oral Candidiasis:

Erythematous Chelitis

Pseudomembranous

Angular

Current Antiretroviral Targets





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Reverse Transcriptase Inhibitors:

Nucleoside Analogues Zidovudine (AZT, ZDV) Didanosine (ddl) 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 (FLV)		

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)

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

phosphorylated to activate compound.

does NOT need to be tri-phosphorylated only di-

Non-nucleoside Reverse Transcriptase Inhibitors:

Agents directly bind to reverse transcriptase to inhibit transcription.

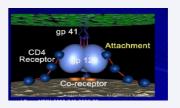
NNRTIs do not require phosphorylation to be active.

Protease Inhibitors (PIs) MOA:

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

Pls 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)



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