

Genotypic ARV Resistance Report

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Patient Information

Our Ref. ID: VCI-20835

Name:	♂ ๒๓	Your Ref. ID:	P18-01483
Hospital/Site:	PRIBTA	Study/Visit:	
Risk Factor:	No Information	Collection Date:	18-Sep-2021
Clinical Staging:	No Information	CDC Staging:	No Information
		Genotyping Date:	04-Oct-2021

Lab Information

Current CD4:	No Information	Current Antiretroviral:	Unknown
Current VL:	1900 copies/ml (18-Sep-2021)		

Summary Data

Subtype and % similarity to closest reference isolate:	B (94.1%)
Sequence includes RT: condons:	20 - 241

Resistance Report (RT)

RT TAMs:	-
RT NRTIs:	M184I
RT NNRTIs:	-
RT Other:	K22R, V35T, V60I, I135T, T200K, R211K, F214L

Antiretroviral	High-level resistance	Intermediate resistance	Low-level resistance	Potential low-level resistance	Susceptible
NRTI					
zidovudine (AZT)					
tenofovir (TDF)					
stavudine (D4T)					
lamivudine (3TC)					
emtricitabine (FTC)					
didanosine (DDI)					
abacavir (ABC)					
NNRTI					
rilpivirine (RPV)					
nevirapine (NVP)					
etravirine (ETR)					
efavirenz (EFV)					
doravirine (DOR)					

ConsensusR#20835RT.txt

Remark: 1. Although the mutation is not found, it does not mean that one is fully susceptible to the treatment since the resistant virus may be minor population which cannot be detected by the assay (detectable limit = viral load 1,000 copies/ml).
 2. The accumulation of TAMs (M41L, D67N, K70R, L210W, T215Y/F, K219Q/E) increases resistance to tenofovir. Mutations M41L and L210W, contribute more than others
 3. References: Stanford dBase system (<http://hivdb.stanford.edu/>)

Reported by: _____ Date: 4/10/2021

Vaccine and Cellular Immunology Laboratory

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Mutation Scoring

	NRTI						
	3TC	ABC	AZT	D4T	DDI	FTC	TDF
M184I	60	15	-10	-10	10	60	-10

RT Comments

NRTI

- M184V/I cause high-level in vitro resistance to 3TC and FTC and low-level resistance to ddI and ABC. However, M184V/I are not contraindications to continued treatment with 3TC or FTC because they increase susceptibility to AZT, TDF and d4T and are associated with clinically significant reductions in HIV-1 replication.

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