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Lack of Resistance Mutations to the Novel HIV-1 Capsid Inhibitor Lenacapavir Among People Living with HIV in Guangdong, China

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Background: The capsid inhibitor (CAI) lenacapavir (LEN) was approved for use in 2022, yet there are few reports about its drug resistance mutations (DRMs) and sensitivity.

Purpose: To delineate the prevalence of CAI DRMs and drug susceptibility among HIV-1 infected individuals living in Guangdong, China.

Patients and Methods: A total of 1035 individuals with HIV-1 infection, including 660 highly Active Anti-Retroviral Therapy (HAART) naive individuals and 375 hAART experienced individuals whose protease (PR)/ reverse transcriptase (RT) fragments were amplified successfully during drug resistance surveillance between October 2021 and December 2023, were randomly included in this study. The entire HIV-1 *gag* gene was amplified from plasma in LEN-naive individuals with or without antiretroviral therapy. The epidemiological and demographic information of the enrolled individuals were collected. The Stanford HIV Drug Resistance Database HIVdb program for Capsid was used to interpret the CAI DRMs and the LEN susceptibility.

Results: Among 1035 samples, 805 *gag* sequences were amplified, sequenced and assembled successfully from 518 hAART drugs naive individuals and 287 hAART drugs experienced individuals. Among them, 0.50% (4/805) carried at least one CAI DRM, of which 0.19% (1/518) from HAART naive individuals and 1.05% (3/287) from HAART experienced individuals. Among the individuals with CAI DRMs, two patients carried CAI major mutations (Q67H) conferring intermediate resistance to LEN and two patients carried CAI accessory mutation (T107A) conferring low level resistance to LEN.

Conclusion: Extremely low prevalence of CAI DRMs was detected among people living with HIV (PLWH) in Guangdong, China. Our observations indicate that LEN application may be promising when used in clinical practice in China. Before the administration of LEN, there is no need to consider detecting CAI mutations in PLWH through DRM examination for the time being.

Keywords: HIV-1, capsid inhibitor, lenacapavir, drug resistance mutation, Guangdong

Introduction

Human immunodeficiency virus (HIV) infection attracts great public health concern, due to its easy transmission across regions and association with numerous deaths worldwide.[1] The HIV-1 genome encodes three major polyproteins (Gag, Pol and Env) and six accessory proteins (Tat, Rev, Nef, Vif, Vpr and Vpu).[2] Among them, Gag proteins (including P17, P24, P7, P6 et al) play vital roles during HIV-1 life cycle, being involved in assembly, virion maturation after particle release and early postentry steps in virus replication.[3] The most important Gag protein is the capsid protein P24, which is produced by the protease enzyme cleavage of the Gag polyprotein.[4]

Lenacapavir (LEN) is a long-acting capsid inhibitor (CAI) of HIV-1 with the suggested application of subcutaneous injection once every six months.[5] Compared with other CAIs, such as PF-74, GS-CA1 and GSK878,[6–8] which are still in the research stage,

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LEN is more active and has better efficacy.[9] LEN was first approved in the European Union for use in combination with other antiretroviral therapy drugs in adults with multidrug-resistant HIV infection in August 2022,[10] and LEN was approved by the US Food and Drug Administration for the treatment of HIV-1 as part of an HAART regimen in heavily treatment-experienced adults in December 2022.[11] In September 2023, the application for the listing of LEN was accepted by the China Centre for Drug Evaluation (No. JXHS2300088), which means that LEN will soon be available in China.[12] Therefore, it is necessary to understand the background of CAI drug resistance mutations (DRMs) among LEN-naive individuals with HIV-1 infection in China.

Resistance associated mutations in close proximity to the LEN binding site on the capsid protein had been identified through cell culture-based viral breakthrough assays.[13,14] However, there are scarce data available about the prevalence of CAI resistance in PLWH in general. Therefore, the aim of this study was to investigate the prevalence of CAI mutations and resistance among people living with HIV in Guangdong, a highly economic competitive province of China in the market, and assess the necessity of drug resistance testing before the administration of LEN in China.

Materials and Methods

Study Population and Data Collection

A total of 1035 HIV-1 infected individuals, including 660 hAART naive individuals and 375 hAART experienced individuals whose PR and partial RT fragments were amplified successfully in drug resistance surveillance between October 2021 and December 2023, were randomly included in this study. All of the HAART experienced individuals were considered to have virological failure (plasma viral load ≥200 copies/mL after six months of treatment), and 225 of them (60.00%) had at least one protease inhibitor (PI), nucleoside reverse transcriptase inhibitor (NRTI), or nonnucleoside reverse transcriptase inhibitor (NNRTI) DRM. The epidemiological and demographic information of all the enrolled individuals, including age, sex, risk group, baseline plasma viral load, and $CD4^+$ T cell count, were collected from the Chinese national free HAART Database.

Sample Processing and HIV-1 Genotyping

Plasma was separated from peripheral blood samples after centrifuging at 3000 r/min for 10 minutes. HIV-1 genotype was used for comparative analysis based on PR and partial RT sequences: firstly, the online tool COMET (https://comet.lih.lu/index.php) was used to determine the HIV-1 genotypes preliminarily, and then IQ-TREE (https://www.iqtree.org), a fast and effective stochastic algorithm online tool to infer phylogenetic trees by maximum likelihood, was used to confirm the subtyping.[15]

RNA Extraction, Amplification, Recovery and Sequencing

The automatic magnetic bead-based Virus RNA Extraction Kit (Daan, China) was used to extract RNA from the plasma samples of patients. The HIV *gag* gene (about 1500 bp, corresponding HXB2 790–2292) was first used for reverse transcription using the one-step PrimeScript III RT–PCR Kit (TaKaRa, China) and then amplified by nested PCR using Ex Taq (TaKaRa, China). The cycling conditions for reverse transcription and the first round of PCR amplification were as follows: 30 min at 50 °C (cDNA synthesis) and initial denaturation for 2 min at 94 °C; 3 cycles of 30s at 94 °C, 1 min at 55 °C and 2 min at 72 °C; and 32 cycles of 15s at 94 °C, 30s at 55 °C and 110 s at 72 °C, followed by a final extension at 10 min at 72 °C. And the cycling conditions for the second round of PCR amplification were as follows: initial denaturation for 2 min at 94 °C; 3 cycles of 30s at 94 °C, 1 min at 55 °C and 2 min at 72 °C; and 32 cycles of 15s at 94 °C, 30s at 55 °C and 100s at 72 °C; followed by a final extension at 10 min at 72 °C. The primers for amplification and sequencing were provided by Prof. Li's group, State Key Laboratory of Pathogen and Biosecurity, Beijing Institute of Microbiology and Epidemiology, China (shown in Supplementary Table 1). Possible contamination was monitored by using the negative controls.

After identification of the amplified PCR products using 1% gel electrophoresis, the positive products were purified and sent to a commercial company (Tianyi Huiyuan, China) for Sanger sequencing.

Drug Resistance Testing and Interpretation

The sequences obtained from sanger sequencing were assembled and cleaned using Sequencher software Version 5.4 and then aligned with BioEdit software Version 7.2. The Stanford HIV Drug Resistance Database HIVdb program for Capsid

(https://hivdb.stanford.edu/hivdb-capsid/by-sequences/) was employed to identify CAI major or accessory mutations and LEN susceptibility. The sequences with high, intermediate, or low level resistance profiles to LEN were all defined as LEN resistance.

Statistical Analysis

The statistical analyses were performed using IBM SPSS Statistics software 25. The quantitative data were described using the median and range. Chi-squared test was used to compare two groups, and $p<0.05$ was considered statistically significant.

Results

Demographic Information of the Study Participants

Totally, 805 *gag* sequences were successfully obtained from 1035 hIV-1 patients whose *gag* sequences were amplified, with a success PCR rate of 77.78%. Among them, 287 sequences from HAART experienced individuals (35.65%) and 518 sequences from HAART naive individuals (64.34%).

The demographic and viral characteristics of 805 study participants are shown in Table 1. Among them, 8.70% (70/805) were recruited in 2021, 46.46% (374/805) were recruited in 2022 and 44.84% (361/805) in 2023. Most of the subjects (83.35%, 671/805) were male. The median age was 42 years old. The most prevailing risk group was heterosexual contact (HET, 59.13%, 476/805), followed by the men who have sex with men (MSM) group (31.06%, 250/805) and intravenous drug users (IDU) group (5.34%, 43/805). At the time of the drug resistance test, the median HIV-1 RNA viral load was 4.43 (log 10, copies/mL) and the CD4+ T-cell count at baseline was 242 (cells/μL), respectively. Among the 287 hAART experienced individuals, approximately 48.43% (139/287) had confirmed infections before 2019. Most of them (82.23%, 236/287) used treatment with two NRTIs plus one PI or NNRTI regimens during the whole antiretroviral process; 17.77% (51/287) of them had ever used regimens containing INSTI.

Table 1 Demographic and Viral Characteristics of 805 Study Participants

All Individuals with Successful *gag* Gene Amplification	**Individuals (%)**	**Cases Related to CAI Mutations**	
		N	**%**
Patients, n	805	4	0.50
Sex, n (%)			
Male	671 (83.35)	4	0.60
Female	134 (16.65)	0	0.00
Year of samples, n (%)			
2021	70 (8.70)	1	1.43
2022	374 (46.46)	2	0.53
2023	361 (44.84)	1	0.28
Treatment status, n (%)			
HAART naïve	518 (64.34)	1	0.19
HAART experienced*	287 (35.65)	3	1.05
Geographical region, n (%)			
Pearl River Delta	517 (64.22)	2	0.39
Northern	97 (12.05)	1	1.03
Eastern	39 (4.84)	0	0.00
Western	152 (18.88)	1	0.66
Transmission Route			
Heterosexual	476 (59.13)	2	0.42
Men who have sex with men	250 (31.06)	1	0.40
Intravenous drug users	43 (5.34)	1	2.33
Unknown	36 (4.47)	0	0.00

(*Continued*)

Table 1 (Continued).

All Individuals with Successful *gag* Gene Amplification	Individuals (%)	Cases Related to CAI Mutations	
		N	%
HIV-1 subtype**, n (%)			
CRF01_AE	313 (38.88)	4	1.28
CRF07_BC	238 (29.57)	0	0.00
CRF08_BC	79 (9.81)	0	0.00
CRF55_01B	56 (6.96)	0	0.00
Other**	119 (14.78)	0	0.00
$CD4^+$ T-cell counts (cells/μL) based on 468 individuals, median (Range)	242 (1–1067)	/	/
Viral loads (Log10, copies/mL) based on 551 individuals, median (Range)	4.43 (2.32–7.39)	/	/
PI DRMs, n (%)	36 (4.47)	0	0.00
NRTI DRMs, n (%)	132 (16.40)	2	1.52
NNRTI DRMs, n (%)	253 (31.43)	2	0.79

Notes: HAART experienced *All treatment failure (plasma viral load ≥ 200 copies/mL after six months treatment), 59.86% of them (176/294) with at least one PI, NRTI, or NNRTI drug resistant mutations. Other **Including B, C, CRF67_01B, CRF68_01B, CRF109_0107, CRF118_BC, CRF125_0107, CRF145_0755, CRF154_0755 and unique recombination form. HIV-1 subtype **Based on PR/RT sequences.
Abbreviations: PR, protease; RT, reverse transcriptase; PI, protease inhibitors; NRTI, nucleoside reverse transcriptase inhibitors; NNRTI, non-nucleoside reverse transcriptase inhibitors; DRMs, drug resistance mutations.

CAI DRMs in HAART Naive and HAART Experienced Participants

In about 0.50% (4/805) of the 805 individuals, at least one CAI major or CAI accessory DRM was detected, and all mutations were detected among CRF01_AE strain. Among the 805 individuals, 0.19% (1/518) was derived from HAART naive individuals, whereas 1.05% (3/287) came from HAART experienced individuals (Table 1 and Figure 1). About 0.25% (2/805) harboured only CAI major DRMs (all HAART experienced, 0.70% (2/287)), and 0.25% (2/805) harboured only CAI accessory DRMs (HAART experienced, 0.35% (1/287); HAART naive, 0.19% (1/518)). CAI major mutation Q67H and CAI accessory mutation T107A (both 0.25%, 2/805) were detected in this study, while no CAI major DRMs were detected among HAART naive individuals.

Sensitivity to LEN in HAART Naive and HAART Experienced Participants

All of the individuals carried CAI major or CAI accessory DRMs associated with intermediate or low level resistance to LEN according to the HIVdb program for Capsid (Figure 1). The characteristics of the patients with CAI DRMs and the corresponding drug resistance levels are shown in Table 2. All of the four patients carrying CAI DRMs showed at least low-level resistance to LEN. Among the 805 individuals, 0.19% (1/518) was derived from HAART naive individuals,

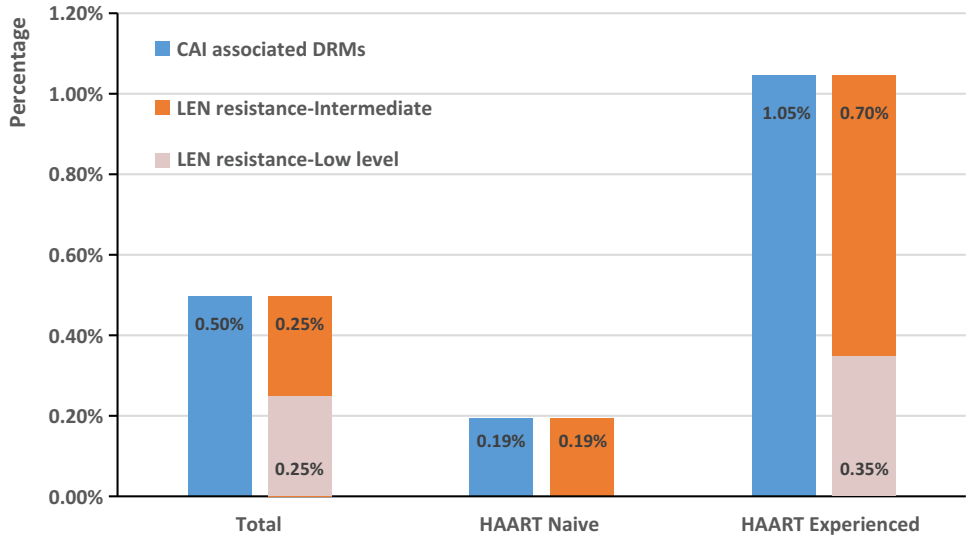

Figure 1 Percentages of CAI associated DRMs and LEN resistance among people living with HIV in Guangdong, China.

Table 2 Characteristics of the Individuals with CAI Drug Resistance Mutations

Sample ID	Treatment status	Geographical Region	Sex	Age	Sampling Time	Transmission Route	HIV-1 Subtype	CAI DRMs		Resistance to LEN	Other Class DRMs
								Major	Accessory		
ZK02074	Experienced	Northern	Male	45	2021/10/26	IDU	CRF01_AE	Q67H	/	M	No
ZK02840	Experienced	PRD	Male	80	2022/3/25	HET	CRF59_01B	Q67H	/	M	NRTI (K65R, V75M, M184V); NNRTI (V106M, G190A)
ZK02880	Experienced	Western	Male	52	2022/3/18	HET	CRF01_AE	/	T107A	L	NRTI (M41L, K65R, Y115F, M184V); NNRTI (V106I)
230861	Naïve	PRD	Male	20	2023/11/15	MSM	CRF01_AE	/	T107A	L	No

Abbreviations: PRD, Pearl River Delta; IDU, Intravenous drug users; HET, Heterosexual; MSM, men who have sex with men; M, intermediate resistance; L, low level resistance; DRMs, drug resistance mutations.

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whereas 1.05% (3/287) came from HAART experienced individuals. It appears that LEN associated DRMs were prone to be induced among HAART experienced participants.

Discussion

Through resistance selection experiments in vitro, the potential for resistance emergence with the use of LEN was characterized. The experiments identified seven variants (including L56I, M66I, Q67H, K70N, N74D/S, and T107N) in the capsid portion of *gag*, associated with reduced susceptibility to LEN and reduced viral fitness.[13] Another in vitro study revealed that, despite the existence of naturally occurring polymorphisms mutations or cleavage site mutations in *gag* gene region, there is no effect in the antiviral activity of LEN, and that LEN shows no cross resistance to other classes of drugs such as PI, NRTI, NNRTI, or INSTIs.[16]

HIV-1 is one of the most rapidly evolving viruses known to date.[17] The selective pressures imposed during drug treatment and the host immune response drive the evolutionary dynamics of HIV in the process of chronic infection stage.[18] Meanwhile, because of the generous incidence of gene recombination and the lack of gene proofreading function of reverse transcriptase, there are multitudinous natural variabilities in HIV-1.[17]

In our current study, extremely low prevalence of CAI DRMs was detected in Guangdong, China. LEN related high level resistance mutations were not detected. A major CAI mutation Q67H (0.25%, 2/805) was detected among HAART experienced individuals. Q67H is associated with about five-fold reduced LEN susceptibility, and confers intermediate resistance to LEN. Another CAI mutation found in this study was a CAI accessory mutation, isolated T107A (0.25%, 2/805) was detected among one HAART naive individual and one HAART experienced individual, causing low level resistance to LEN. T107A has been reported in combination with M66I in one patient receiving LEN.[19] However, its efficacy based on susceptibility alone has not been reported. In this study, the proportion of CAI related DRMs in HAART experienced individuals (1.05%, 3/287) was greater than that in HAART naive individuals (0.19%, 1/518), and the difference was not statistically significant ($\chi 2 = 1.263$, $P=0.261>0.05$).

The rare emergence of CAI mutations is also in accordance with similar analysis done in other geographical regions. One work posted at the 17th European AIDS Conference (PE13/15) revealed that none of seven CAI mutations were detected in 1500 samples from individuals with multiple subtypes of HIV-1 infection (treatment-naive or treatment-experienced individuals with or without prior usage of PIs) from hospitals in Paris, France.[20] Additionally, a sequence-based analysis (calculating more than ten thousand sequences downloaded from the Los Alamos National Laboratory HIV database) result also showed the rare occurrence of LEN related DRMs.[21]

We found that all mutations were detected among CRF01_AE strain (Table 1) in this study, and 2/4 of the cases which carried CAI DRMs also harboured other class DRMs such as NRTIs- or NNRTIs- DRMs (Table 2), this may be due to selective pressures exerted accompanied with acquired drug resistance. CAI resistance mechanisms and the extent to which this resistance impacts the clinical effectiveness of CAIs need to be investigated in further studies.

Conclusion

In conclusion, a comparatively low prevalence of CAI DRMs was detected in this large scale queue, indicating that either HAART naive individuals or HAART experienced individuals were sensitive to LEN. Our observations indicate that LEN application may be promising when used in clinical practice in China, especially for the population with resistant INSTIs. Before the administration of LEN, there is no need to consider detecting CAI mutations in PLWH through DRM examination for the time being.

Ethics Approval and Informed Consent

The study was approved by the Institutional Review Board of Guangzhou Eighth People's Hospital (No. 202353290) and was conducted in accordance with the principles of the Declaration of Helsinki. All individuals provided written informed consent.

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Disclosure

The authors report no conflicts of interest in this work.

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