

EV0013

ePoster Viewing

HIV/AIDS (incl anti-retroviral drugs, treatment & susceptibility/resistance, diagnostics & epidemiology)

Molecular epidemiology of BK virus in Iranian HIV-infected patients

Amitis Ramezani*¹, Minoos Mohraz², Kayhan Azadmanesh³, Rouhollah Vahabpour⁴, Nasim Chitsaz⁵, Monireh Kazemimanesh³, Mohammad Banifazl⁶, Shahla Akhgari⁷, Anahita Bavand¹, Arezoo Aghakhani¹

¹*Clinical Research Dept., Pasteur Institute of Iran, Tehran, Iran*

²*Iranian Research Center for Hiv/Aids, Tehran, Iran*

³*Pasteur Institute of Iran, Virology Dept., Tehran, Iran*

⁴*Pasteur Institute of Iran, Hepatitis and Aids Dept., Tehran, Iran*

⁵*Franchise Manager at Behestandarou, Msd Products Agency, Tehran, Iran*

⁶*Iranian Society for Support of Patients With Infectious Disease, Tehran, Iran*

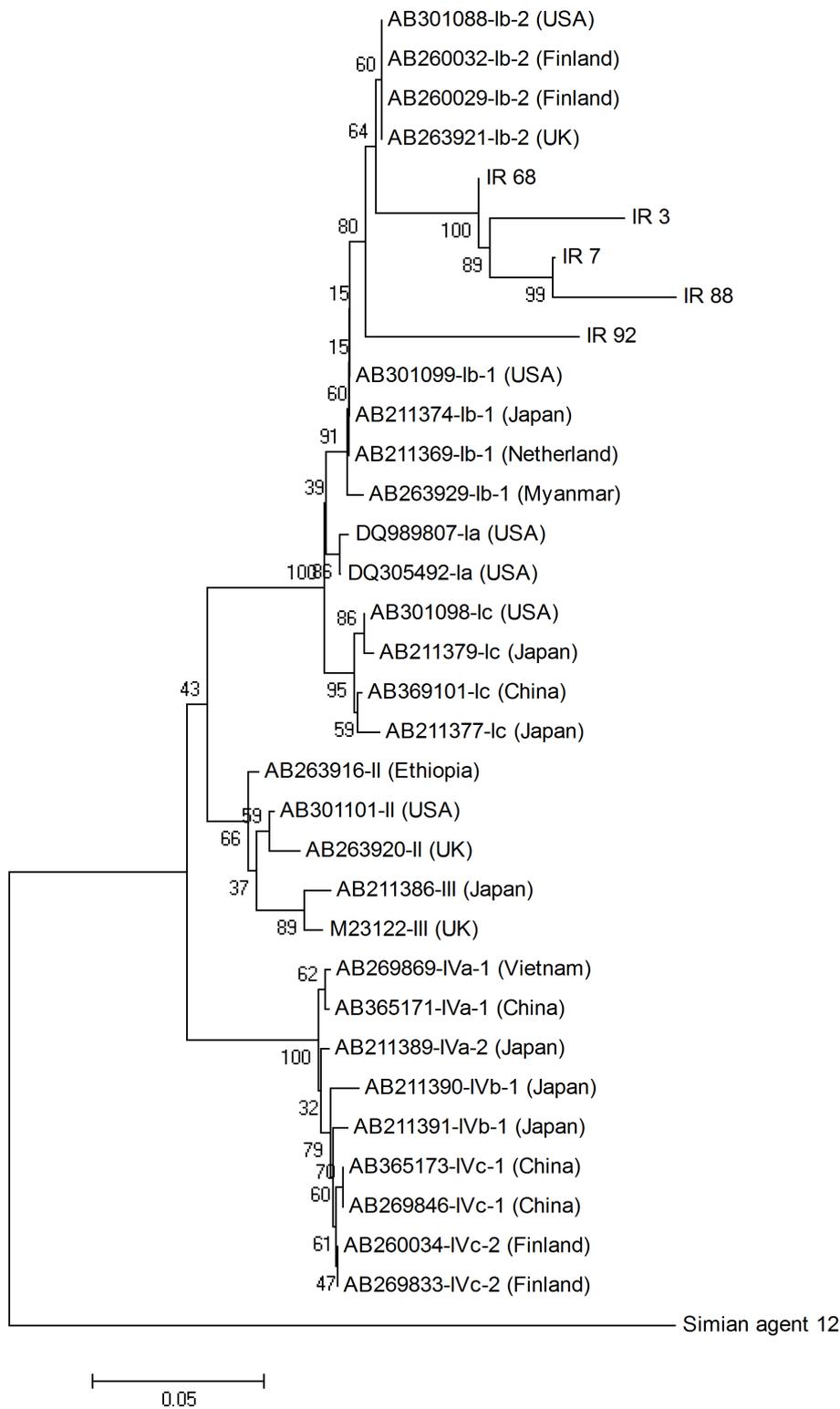
⁷*Payame Noor University of Tehran Shargh, Tehran, Iran*

Background: Human polyomavirus BK virus (BKV) belonging to the Polyomaviridae family, a double-stranded DNA virus that infects approximately 90% of the general population as a subclinical or mild infection. Under immunosuppressive conditions such as kidney transplantation or HIV infection, BKV may be reactivated resulting in hemorrhagic cystitis and tubulointerstitial nephritis. Due to insufficient data on the prevalence and molecular epidemiology of BKV viremia in HIV patients we aimed to determine the prevalence and molecular epidemiology of BKV in Iranian HIV infected patients.

Material/methods: In this cross-sectional study, 99 patients with HIV infection without any neurological or renal problems in Tehran, Iran from January to April of 2014 were consecutively enrolled. Presence of BKV DNA in plasma was evaluated by nested PCR. PCR products were purified and then sequenced directly for both directions and phylogenetic tree was constructed.

Results: A total of 99 HIV infected patients with mean age of 37.9 ± 10 years were enrolled in the study. The mean CD4 count was 410.3 ± 211.4 cells/mm³. More than half of the patient (50.5%) presented CD4 counts 200-499 cells/mm³, 30.3% with CD4 counts ≥ 500 and 19.2% had CD4 counts less than 200 cells/mm³. 74.7% of patients were under HAART treatment as lamivudine 74.7%, efavirenz 60.6%, zidovudine 51.5%, tenofovir 21.2%, and the rest with lopinavir, nevirapin in different combinations. BKV DNA was detected in 8.08% of HIV patients. There was no significant difference in respect to BKV viremia and age, sex, possible route of HIV transmission, CD4 count and specific antiretroviral treatment in our study cohort. Characteristics of HIV infected patients with BKV viremia will be presented in congress. BKV viremia presented in 4 out of 25 patients (16%) not receiving antiretroviral therapy in comparison to 4 out of 74 of HAART-treated patients (5.4%) ($P=0.023$). In patients with CD4 counts ≥ 200 cells/mm³ viremia was found more frequently ($7/80 = 8.8\%$) than in those with lower counts ($1/19 = 5.2\%$) (Not significant). All sequenced BKV isolates belonged to subtype Ib-2.

Fig.1: Phylogenetic tree was constructed by the neighbour-joining method of five BKV isolates detected from Iranian HIV patients. The numbers represent the percentages of bootstrap values (1000 replicates) for the node. Our sequences assigned in this study are revealed by the IR prefix.



Conclusions: Our findings indicated that BKV viremia is relatively prevalent in HIV infected patients and significantly higher in naïve than HAART-treated cases. Therefore HAART can eliminate BKV infection from plasma and reduce viremia although the actual implication of BKV viremia in HIV patients is not clear. Moreover, BKV Ib-2 was the only detectable genotype in our study cohort from Iran.