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Frequent genetic defects in long-term survivors for more than 26 years in the absence of antiretroviral therapy in Korea: its association with ginseng treatment

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Background

The association between long-term nonprogressors (LTNPs) and genetic defects in HIV-1 is not clear because of paucity of genetic defects in LTNPs. To date, there is only a report on gross deletion in 2 genes of HIV-1. Ginseng has been used as the premier medicinal plant for a thousand years [1]. Korean red ginseng (KRG) has been applied to HIV-1 infected patients since November 1991 [2]. We have found that most HIV-1 infected patients treated with KRG for a significant period slowly progressed to AIDS and revealed a high frequency of genetic defects.

Materials and methods

A total of 250 individuals were diagnosed with HIV-1 infection in Korea before 1993. Among the 250 individuals, we selected all who remain healthy for >18 years in the absence of antiretroviral therapy. They were four individuals (26, 25, 21, and 18 years from diagnosis). One patient who was diagnosed with HIV-1 in 1992 took a little KRG – less than 1,000 g. To investigate whether LTNPs are associated with genetic defects, we amplified near full-length HIV-1 sequences in 10 overlapping fragments (0.8 to 1.4 kb) using nested PCR with peripheral blood mononuclear cells. We compared the data with that in 4 control patients. Amount of KRG supplied was significantly higher in LTNPs (21,767-8,866 g) than 1,250-1,304 g in controls ($P < 0.01$).

Results

We obtained 1,339 and 386 PCR amplicons over 20 years in LTNPs and over 10 years in controls, respectively. All LTNPs revealed at least one genetic defect in 4 genes, whereas controls revealed it in both 5' LTR/*gag* and *nef* genes. Overall genetic defects were significantly higher in LTNPs (11.4%) than 4.9% in controls ($P < 0.001$). Among the 4 LTNPs, one patient who had taken the least amount of KRG revealed significantly higher proportion of premature stop codons (5.6%) than 1.6% in 3 LTNPs ($P < 0.01$). There was a significant difference in the frequency of genetic defects between on KRG (10.9%) and baseline prior to KRG intake (4.3%) ($P < 0.01$). There was a significant correlation between amount of KRG supplied and proportion of genetic defects (r=0.80, $P < 0.05$). At single gene level, only the *nef* gene showed a significantly higher frequency of genetic defects in LTNPs (11.5%) than 3.6% in controls ($P < 0.01$).

Conclusion

These data show the possibility that frequent genetic defects that might be caused by KRG treatment are associated with long-term slow progression.

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