Retrovirology

Poster presentation

P16-39. T cell recognition of autologous and non-autologous HIV-1 protease peptides by HIV-1 infected patients undergoing PI therapy

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Background

HIV-1 protease is an important virus protein that is a target of both antiretroviral therapy and cellular immune response, which can both act as inducers of mutation. Here, we investigated the T cell recognition of autologous HIV-1 protease epitopes.

Methods

Seventy-two HIV-1+ patients undergoing protease inhibitor (PI) therapy were recruited. We synthesized three peptides from HXB2 strain protease (wild-type) and 32 additional peptides from the same regions, which encompassed the most frequent PI-induced mutations in Brazil. PBMC from those patients were analyzed by the CFSE dilution assay to detect proliferating CD4+ and/or CD8+ T cell responses against each of the 35 protease peptides. Pol protease HIV-1 genomic regions were sequenced using internal primers with the ABI Prism Dye Terminator Cycle Sequencing Reaction Kit in an automated sequencer.

Results

Variable profiles of recognition of wild-type and corresponding mutant epitopes were found. To analyse these profiles, we took into consideration only those patients whose autologous protease sequence included a 100% match with one of the tested peptides. Only 21% such patients recognized a peptide identical to its own autologous protease sequence. Paradoxically, the most frequent pattern of recognition (49%) was the recognition of peptides dissimilar to autologous protease sequences, with failure to recognize peptides identical to autologous sequences. This indicated that the absence of recognition of autologous sequences was not due to spurious sequence variations not included in peptides, but rather by the actual absence of recognition of such matching autologous peptides. Bona fide crossreactivities were a relatively rare event, occurring in 18% of the patients.

Conclusion

Recognition of sequences dissimilar to the autologous sequence occurred in the majority of patients. It is possible that the observed immune responses were developed to an antigenic sequence which was common in the past, and which then underwent an escape or drug-induced mutation.

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