

Factors Associated with Lack of Serological Responses to HIV and HCV in Coinfected Patients with Profound Immunosuppression

1065

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I. Background

A limited number of seronegative HIV-infected individuals with AIDS and very late disease stages have been reported in the literature. In contrast, HCV seronegativity does not seem to be rare in HIV patients. It is unclear if seronegativity for any of these infections could occur as result of loss of pre-existing antibodies (seroreversion) or to a primary lack of antibody production. Herein, we report HIV and HCV antibody responses in a large series of HIV-infected patients with profound immunosuppression.

II. Methods

From 2001 to 2006, all patients diagnosed with HIV-1 infection (EIA reactive and positive WB) who presented with CD4 counts <50 cells/ μ L at our institution were identified.

Sera were screened by two different EIAs, a third generation and a second-generation EIA. The latter assay has been proven to be particularly useful to detect HIV-1 seroreversion (CID 2006;42:700-8). In subjects with CD4 counts <10 cells/ μ L, an HIV-1 WB assay was additionally performed. All HIV-infected patients with a history of intravenous drug use were screened for HCV antibodies using commercial EIA. A sensitive HCV-RNA PCR assay, was performed in subjects with HCV seronegativity.

III. Results

Of 417 subjects fulfilling the requested criteria, 331 had samples available. Data of this group are shown in Table 1. All were reactive for HIV using both EIAs. Low-level optical density values using the second-generation EIA (which could be associated with seroreversion) were not observed.

All 86 subjects with CD4 counts <10 cells/ μ L had a positive WB pattern (reactivity to at least two *env* bands), although a few had weak or lack of reactivity to *gag* or *pol* products.

Twenty subjects maintained severe immunodeficiency for long periods (median of 13.3 months; IQR: 6.9-26.5) but did not show changes in EIA reactivities for HIV over time.

In the 255 IDUs that were screened for HCV antibodies, only 17 (6.7%) were seronegative. However, 6 of them were serum HCV-RNA+. Interestingly, all showed low-level HCV-RNA (<106 IU/mL) and 4 of them could be genotyped (3 HCV-1 and 1 HCV-4). Data are shown in Table 2.

Table 1. HIV-1 positive patients with CD4<50 cells/ μ L

Nº subjects	331
Male	297 (89.7%)
Age (years) [mean]	38.9 (SD 7.4)
Risk factors	
IDU	255 (77%)
Heterosexual	22 (6%)
Homosexual	39 (11.8%)
Unknown	15 (4.5%)
CD4 (cells/ μ L) [median]	23 (IQR 29)
HIV-RNA (log cop/mL) [mean]	4.6 (SD 1.2)
EIA 2 nd generation, reactivity	100%
EIA 3 rd generation, reactivity	100%

Table 2. HIV-1 positive patients HCV-EIA negative HCV-RNA positive

Patients	CD4 (cells/ μ L)	HIV-RNA (cop/mL)	HCV-RNA (cop/mL)	HCV Genotype
1	35	27254	876000	3A
2	12	210522	36600	1A
3	7	43000	4200	1A
4	30	<50	266000	1B
5	8	36000	102	-
6	48	550000	107	-

IV. Conclusions

Complete HIV seroreversion in subjects with profound immunosuppression seems to be a very rare event. Our results indirectly suggest that seronegativity in HIV infection may be more the result from lack of antibody formation. In contrast, HCV seronegativity is relatively frequent in immunosuppressed HIV patients and might be the result from poor HCV replication and/or severe immunosuppression.