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Effect of hepatitis C virus infection on the lymphoid population in hemophiliacs [letter; comment]

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EFFECT OF HEPATITIS C VIRUS INFECTION ON THE LYMPHOID POPULATION IN HEMOPHILIACS

To the Editor:

The recently published paper by Brettler et al[1] prompts us to make the following comments: (1) Although it is of interest to discover the prevalence of hepatitis C virus (HCV) infection in hemophiliacs treated with factor concentrates that had not been subject to viral inactivation, it is more interesting to elucidate whether factor concentrates of newer generations elaborated using virucidal methods are safe and do not create the risk of HCV contamination. (2) Much has been written about the impact of human immunodeficiency virus (HIV) infection on lymphoid populations. However, as the authors have shown in their paper,

there is a statistically significant association between HCV and HIV infection in hemophiliacs and, therefore, it is possible that some of the lymphoid alterations thought to be due to HIV might in fact be either the result of HCV infection or a combination of both viruses.

In a series of 26 hemophiliacs, 67% of the patients treated with factor concentrates that had not undergone viral inactivation displayed antibodies to HCV. By contrast, none of the three patients treated exclusively with pasteurized factor (Haemate P and Behring HS) exhibited antibodies to HCV, hepatitis B virus, or HIV.

To analyze the impact of HCV on lymphoid populations we formed three groups of patients: (1) HCV+, HIV−; (2) HCV+, HIV+; and (3) HCV−, HIV+. A decrease was observed in the CD4 population in all the subgroups (721 ± 124, 348 ± 233, and 334 ± 444, respectively; controls 1,238 ± 409), thus suggesting that this finding does not reflect specificity to HIV infection, although it was more severe in the HIV+ patients ($P = .04$). In the first group (HCV+, HIV−) the decrease in the CD4 population was only due to a decrease in the helper inducer (CD4+, Leu8−) cells, whereas in the HIV+ groups a decrease appeared in both subpopulations: helper-inducer (CD4+, Leu8−) and helper-supressor (CD4+, Leu8+). The HIV+ patients also showed an increase in their T-cell–activated lymphocytes (CD3+, Ia+) and a decrease in the CD16+ natural killer cells that was not observed in the first group. By contrast, none of these alterations was found in the three patients who had only received pasteurized factor. Such findings suggest that the lymphocyte alterations observed in the patients with HCV antibodies are not specific and would rather be related to the presence or absence of HIV infection.

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RESPONSE

To the Editor:

The comments in the letter of Gomez et al are appreciated. Our study was not intended to study the safety of newer viral inactivated concentrates. Recent studies are demonstrating that such methods do appear to lower the risk of HIV infection.[1] Although examining CD4 lymphocyte populations in the different subset of patients (HIV+, HCV−; HIV−, HCV+; HIV−, HCV−) was beyond the scope of our paper, we agree that lymphocyte alterations in the hemophilic population are related mainly to the presence of HIV infection. However, other authors have suggested that liver disease may cause alterations in lymphocyte subpopulations in HIV negative hemophilia patients.[2]

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