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Authors' Reply

Fibrosis progression in chronic hepatitis C in patients coinfected with human immunodeficiency virus and hepatitis C virus

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Fibrosis progression is a complex event in patients with chronic hepatitis C. Fibrosis progression occurs when the components of the extracellular matrix are deposited in excess in the liver, leading to the substitution of functional hepatic tissue with non-functional fibrotic tissue. Although several studies have investigated the mechanisms of fibrosis development, fibrosis progression still constitutes a public health problem. In patients with chronic hepatitis C, fibrosis progression has been associated with several factors, including host genetic polymorphism. Like human leukocyte antigen (HLA), human platelet antigen (HPA) has also been demonstrated to influence fibrosis progression. However, these study were conducted with patients who were monoinfected with hepatitis C virus (HCV). The study by Picelli et al.[1] is the first to have evaluated the influence of HPA polymorphism in fibrosis progression using patients with human immunodeficiency virus and hepatitis C virus (HIV/HCV) coinfection. Although the study included 36 patients, they were representative of the entire population of HIV/HCV coinfected patients who were assisted at the Specialized Outpatient Service of Domingos Alves Meira and at the Department of Internal Medicine, Gastroenterology Division, Botucatu School of Medicine [São Paulo State University (UNESP), Botucatu, SP, Brazil]. This study showed that HPA-1, -3, and -5 polymorphisms do not influence fibrosis progression in HIV/HCV coinfected patients. Therefore, although there are demonstrated associations between HPA polymorphism and fibrosis progression in HCV monoinfected patients, similar associations are not observed in HIV/HCV coinfected patients. Accordingly, additional studies could be conducted to evaluate other factors that may have associations with fibrosis progression in coinfected populations.

Associations have already been reported between diseases and genetic host factors, such as HLA and other polymorphisms[2]. Similarly, previous studies have already shown that HPA system polymorphisms are associated with viral diseases[3][4] and disease progressions, including fibrosis progression in patients with chronic hepatitis C[5].

In the study of Picelli et al.[5] that was cited in this letter to the editor, the associations between HPA-1, -3, and -5 systems and fibrosis progression were investigated in patients with HIV/HCV coinfection. When comparing patients with HIV/HCV coinfection and either lower stages of fibrosis (F1/F2-Group 1) or higher stages of fibrosis (F3/F4-Group 2), Picelli et al.[1] observed no deviations from the Hardy-Weinberg equilibrium in the HPA systems that were evaluated. No differences were observed between G1 and G2 according the distributions of the allelic and genotypic frequencies of the HPA systems.

Thus, the role of HPA system polymorphisms in fibrosis progression was differed for patients with HCV monoinfection[5] and those with HIV/HCV coinfection[1].

It is important to consider that HPA is not a soluble factor, unlike platelet-derived growth factor or transforming growth factor β1. HPA systems are polymorphic antigenic determinants that reside in proteins of the platelet membranes[6]. Therefore, HPA molecules could be cited in the letter in the same manner as those receptors and membrane proteins.

Conflict of Interest

The authors declare that there is no conflict of interest.

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