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 studies were conducted with patients who were monoinfected with hepatitis C virus (HCV). The study by Picelli et al. 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 coinfection. 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.


