NAFLD, HEPATOTROPIC VIRUSES AND CARDIO-METABOLIC RISK

Authors:

Amedeo Lonardo[1], Fabio Nascimbeni[1,2*], and Stefano Ballestri[3]

Affiliations:

[1]Azienda USL di Modena, NOCSAE

[2] University of Modena and Reggio Emilia, Department of Biomedical, Metabolic and Neural Sciences

[3]Azienda USL di Modena, Pavullo Hospital

Corresponding Author: Fabio Nascimbeni, M.D., Ph.D.

Address: Division of Internal Medicine, Nuovo Ospedale Civile Sant'Agostino Estense (NOCSAE) – Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia and Azienda USL, Via Giardini 1355, 41126, Baggiovara, Modena, Italy. E-mail: fabio.nascimbeni@unimore.it. Phone: +390593961805; Fax: +390593961322

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1002/hep.29052

To the Editor:

In their large epidemiological study, Joo et al. report on HBsAg seropositivity as a factor strongly associated with a reduced risk of developing NAFLD over follow-up, probably as a result of interference of HBV with the pathogenesis of NAFLD (1). These findings also have major clinical consequences, we would like to comment on.

A previous article from our group has illustrated the link between cirrhotic and non-cirrhotic liver disease of varying etiology and cardiovascular risk (2). In short, based on published studies, we concluded that liver steatosis, due to alcohol abuse, metabolic derangements and HCV infection, is indeed associated with insulin resistance and cardiovascular risk. Conversely, HBV infection and primary biliary cholangitis, which (unless in case of concurrent host's additional metabolic risk factors) are not directly associated with liver steatosis and insulin resistance, appear to be spared from cardio-metabolic risk (2). Moreover, with regard to the replicative cycle of HBV and HCV, liver steatosis seems to exert opposite influences: while HCV is associated with steatogenesis and thrives on fatty substrates, HBV is not steatogenic and its replication seems to be inhibited by fatty liver (2).

From this general notion, and given that liver steatosis is a strong predictor of cardio-metabolic risk (3), it can be anticipated that HBV infection, being associated with lower rates of incident NAFLD, will likely be spared from cardio-metabolic risk. Confirming this prediction, recent studies have shown that individuals with HBV infection are protected from developing both the metabolic syndrome and acute ischemic stroke (4, 5).

In conclusion, the study by Joo et al. is important in promoting our understanding of the pathogenesis and risk factors of NAFLD. However, additional studies are needed to further explore the link between liver steatosis, hepatotropic viruses and cardio-metabolic risk and to strengthen the notion that fatty liver is a trigger and an amplifier of cardio-metabolic risk and a major modulator of infection with hepatotropic viruses (2).

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