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Virological Reaction in Patients with Persistent HCV

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Editorial

Chronic Hepatitis C virus (HCV) infection is one of the most common causes of chronic liver disease, cirrhosis, hepatocarcinoma and a leading indication for liver transplantation worldwide. During its replication, HCV uses and modulates lipid-related pathways in the host inducing profound changes in lipid metabolism. Rapid changes in lipid homeostasis and insulin resistance have been demonstrated in chronic HCV patients treated with IFN-free antivirals. Despite the presence of relatively low levels of circulating total cholesterol and LDL-C, HCV infection has been linked to an increased incidence of cardiovascular (CV) adverse events Consistently, a reduction in CV risk has been associated with viral clearance and eradication upon antiviral treatment.

In recent years, HDL functionality has emerged as an important factor linked to relevant CV outcomes. In addition, apolipoprotein levels and their ratios to cholesterol levels –as indicators of lipoprotein particle size- have been proposed as surrogate predictors of CV events. In our study, the changes in serum lipid levels, apolipoproteins and their ratios, and HDL functionality were evaluated in patients with chronic HCV infection undergoing treatment with direct-acting antivirals agents (DAAs). This was a single-center prospective observational study. Consecutive patients with chronic HCV infection being treated with DAA were included. Blood samples were obtained prior to the start of DAA, 4 weeks within treatment, at the end of treatment (EOT, 12 or 24 weeks), and 12 weeks after treatment completion.

Demographic, laboratory data and virological variables were evaluated at different stages of treatment. Standard lipid profile, apolipoprotein A-I and B, ApoB/ApoA-I ratio and LDL/ApoB ratio were calculated, and the antioxidant HDL function was determined using a validated dihidrorodhamine (DHR)-based fluorescent assay. Whole plasma was used for antioxidant HDL function determination as previously described. Informed consent was obtained prior to enrollment and the study was approved by the Ethical Review Board of the Facultad de Medicina, Pontificia Universidad Catolica de Chile (study protocol 14-019). For the statistical analysis, descriptive statistics (i.e. medians, IQR, frequencies with CI) were first performed to assess the characteristics of the study sample. Analyses were aimed to determine variations in lipid serological variables during and after DAA treatment. Paired-sample analyses were conducted, using Fisher's Exact Test for categorical variables and Mann–Whitney's or Student's T tests for quantitative data, according to normality tests. One-way ANOVA for paired samples with correction for multiple group comparisons was performed to assess the changes in the clinical, laboratory and virological variables during and after treatment. Stata 13.0 ® and Prism 6 Graphpad ® software were used for statistical analyses.

Twenty patients were assessed during our study. Their median age was 60.5 years (IQR 56–70), 65% (13/20) were male, 80% (16/20) had cirrhosis, and most were classified as Child-Pugh class A (81.5%). HCV genotype 1b was responsible for 100% of infections, and 45% (9/20) had a previously failed antiviral treatment with IFN-based regimens. One patient had a CV event (MI, stroke) before entering the study, and no CV event was seen in patients included during the study period. None of the patients were using statins at enrolment or during the study period. The patient's baseline characteristics are depicted in Supplementary Table 1. Treatment regimens included asunaprevir + daclatasvir (ASV-DCV) for 24 weeks, sofosbuvir + daclatasvir (SOF-DCV) for 12 weeks and sofosbuvir + ledipasvir (SOF/LDV) for 12 weeks. Upon DAA treatment 90% (18/20) of the infected patients achieved SVR12 and HCV load was undetectable at week 4 of treatment, with a rapid decrease in ALT serum levels as expected.

Increases in total cholesterol (134.1 mg/dl to 157.5 mg/dl, p = 0.0122) and LDL-C (67.0 mg/dl to 83.8 mg/dl, p = 0.0136) levels were observed in patients achieving SVR12 when baseline values were compared with the week 12 of the follow-up period. No changes in triglycerides, HDL-C, apolipoprotein A-I and B levels and apo B/A-I ratio were found during treatment or in the follow-up period. However, the LDL-C/apo B ratio, which estimates LDL particle size, increased after treatment (0.93 to 1.28, p = 0.023). Changes in the lipid panel values at each point are detailed in supplementary. HDL antioxidant capacity improved gradually from 34.4% at baseline to 42.4% at week 4 of treatment (p = 0.011), 65.9% at the end of treatment (p = 0.002) and remained stable at the 12-week follow-up period (62.2%, p = 0.001) after the end of treatment, compared to baseline values. HDL antioxidant capacity did not improve in the two patients (10% of the sample) who did not achieve SVR12. Further analysis indicated that SVR was the only variable associated with improvement of HDL function (p = 0.048)

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