Regression of Fibrosis After Direct-acting Antivirals Treatment of Patients with Hepatitis C Virus Cirrhosis: Transposability to Nondecompensated Patients

Dear Sir,

We read with much interest the study by Garg et al.¹ recently published in the *Journal of Clinical and Experimental Hepatology* reporting how, in a real-life study carried out in northern India in patients with liver cirrhosis related to chronic hepatitis C virus (HCV) infection, successful antiviral treatment with direct acting antivirals, with or without ribavirin, is able to determine a decrease of more than 2 points in the Child-Pugh score in approximately 80% of patients with decompensated liver disease and by 1 point in approximately 30% of patients with compensated cirrhosis.

Indeed, we feel that Garg et al should be commended for performing such a thorough evaluation, both before and after antiviral therapy, using all the noninvasive (transient elastography and biochemical indexes) armamentarium currently available to stage disease and also other parameters able to assess prognosis of patients with liver disease such as the Model for End-stage Liver Disease and the Child-Pugh scores.² Noteworthy, they also showed, in the subset of patients with compensated liver disease at the baseline, that liver stiffness significantly decreased after HCV cure and that 66.7% of these patients presented an elastographic decrease that was consistent with a fibrosis stage decrease.

We recently assessed the outcome of sofosbuvir-based antiviral treatment in a consecutive series of 27 patients with advanced, compensated chronic liver disease due to HCV infection who had a median follow-up after the end of successful antiviral therapy of 60 weeks (minimum follow-up: 52 weeks) and found that median liver stiffness significantly ($P < 0.001$) decreased from 20.9 kPa before treatment to 15.9 kPa at follow-up, with a regression of at least one fibrosis stage in 44.4% of patients, according to elastography-assessed METAVIR stages. Moreover, during follow-up, we observed a median stiffness decrease of 44.1%, with 16 patients (59.3%) showing an elastographic response to treatment and no episodes of liver decompensation.

Despite our results were not corroborated in patients with decompensated disease, they are strikingly in keeping with those obtained by Garg et al in a population with different ethnic and demographic background, and therefore, we feel that what has been observed in the study carried out in northern India can also easily be transferred to

other settings.³ Finally, we feel that, taking into account durability of response to antiviral treatment, even longer periods of time will be required so as to assess the fate of those cirrhotic patients—who represent approximately 50% of the treated population—where there is no apparent improvement in a relatively shorter follow-up and to identify outcome predictors.⁴

CONFLICTS OF INTEREST

The authors have no conflict of interest to disclose.

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