Comment on: "Effect of sustained virologic response on the incidence of hepatocellular carcinoma in patients with HCV cirrhosis"

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We read with great interest the study by Cheinquer et al.¹ in a recent issue of the *Brazilian Journal of Infectious Diseases* where they suggest that patients with cirrhosis due to hepatitis C virus (HCV) who achieved sustained virological response (SVR) had significantly lower incidence of hepatocellular carcinoma (HCC) when compared to those without treatment response. Also, interferon treatment after curative resection or ablation of HCC in HCV related cirrhotics prevents HCC recurrence and improves survival, especially for patients with SVR, had been reported in a recent meta-analysis.²

However, a large-scale randomized controlled trial (the HALT-C study) showed no significant difference in the incidence of HCC between patients on maintenance interferon therapy and those without.³ The reason for the discrepant results in studies needs further clarification, together with analysis of the difference in incidence rates of HCC among cirrhotic patients.

A prospective study was carried out to investigate the efficacy and safety of standard interferon with a low progressive dosage regimen in combination with ribavirin in patients with HCV-related decompensated cirrhosis in our center since 2008. Two patients with HCV-related decompensated cirrhosis developed HCC after successful antiviral therapy. Both of them had been treated with standard interferon $\alpha\text{-}2b$ (3 Mu, every other day) and ribavirin (800 mg/d) for 48 weeks. The first case (62-yearold, female, Child-Pugh score 8 and genotype 1b) developed a hepatocellular carcinoma at month 6 after achieving SVR. The second case (53-year-old, male, Child-Pugh score 9 and genotype 2a) achieved early virologic response and end of treatment response, a single 2 cm tumor nodule was detected by enhanced CT liver scan three months after end of treatment. Both patients underwent transcatheter arterial chemoembolization due to expansion and shortage of organs.

Although SVR is associated with long term good prognosis for patients with HCV infection, 4,5 two out of 28 cirrhotic patients developed HCC compared to none of 146 patients without cirrhosis during follow-up for a mean of 47 months after SVR.5 It therefore remains uncertain if persistent virologic suppression to undetectable levels of HCV RNA impacts the risk of developing HCC in patients with severe hepatic fibrosis. Since cirrhosis is an independent risk factor for HCC, a vigilant monitoring of patients with HCV-related cirrhosis (compensated and decompensated) should be mandatory, even if SVR has been achieved after receiving interferon-based therapy.

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