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Therapy of hepatitis C in thalassemia: the influence of iron on achieving sustained viral response

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Dear Editor,

We read with great interest the published letter by Taher et al. [1]. It was a case report of sustained viral response in retreatment of chronic hepatitis C virus (HCV) infection with pegylated interferon and ribavirin in a thalassemic case with previous combination treatment failure after switching to another iron chelating agent that dramatically had decreased his serum and liver iron concentration. The author also in another study had concluded that ribavirin in thalassemic patients was safe and effective. Herein, we want to add some points that merit paying attention.

In a meta-analysis that we have already conducted, we reviewed 15 prospective clinical trials of anti-HCV therapy of HCV-infected thalassemic patients. The evidence regarding pegylated interferon and ribavirin are really scarce; however, base even on these evidence, we could conclude that ribavirin is safe turn back after cession of therapy. Although information for genotype 1 was too few however, we could statistically determine that genotype 1-infected individuals are the most group of patients who take benefit from administering ribavirin. Because of lack of data, we could not draw conclusion about other genotypes, particularly 4.

In chronic HCV-infected subjects, dysmetabolism of iron and both serum and hepatic iron overloading and their mutual

association with stage of liver fibrosis and grade of necroinflammatory activity are determined to be an undeniable fact [2]; however, we think that the considered role of this iron status in viral response of both thalassemic and non-thalassemic subjects is being overemphasized. Desai et al. in a meta-analysis of six RCTs, including non-thalassemic subjects, concluded that phlebotomy had OR of 2.6 (95% CI 1.6–4.5); however, just one of these six trials reported significant results [3]. Five of them failed to determine significant improvement in sustained viral response of subjects who received phlebotomy as long as the therapeutic regimen [4, 5]. Despite of sustained viral response, they consistently reported that end-of-treatment biochemical and histological responses significantly influenced hepatic iron concentration [4, 5]. Also, in our meta-analysis on thalassemic subjects, most of the trials that compared liver iron content or serum ferritin of sustained viral responders and non-responders did not report significant differences in liver iron content or serum ferritin [6–11]. It is noteworthy that Fargion et al. have reported that biochemical response to 1-year IFN therapy is more dependent on liver iron content than viral markers [12].

To summarize, we think that both biochemical and histological responses and liver and serum iron content are heavily dependent on each other; however, the role of iron on sustained viral response in both thalassemic and non-thalassemic patients is still uncertain, and we should look after other approaches to improve rate of sustained viral response in these patients.

Conflict of interest The authors declare that they have no conflicts of interest relevant to the study.

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