Hepatitis C virus (HCV) is the leading cause of chronic liver disease, including cirrhosis and cancer, and liver transplantation. Undoubtedly, effective antiviral therapy is highly essential for achieving sustained virological response (SVR) in HCV-infected patients [1,2]. Simeprevir (SMV) is a macrocyclic NS3/4A HCV protease inhibitor with a potent activity against several HCV genotypes [3]. Combining SMV/sofosbuvir (SOF), a polymerase inhibitor +/- ribavirin (RBV) is widely used as a first line HCV treatment regimen, especially in the developing countries, but is it justifiable enough?

In a recent meta-analysis evaluating the effectiveness of SMV/SOF +/- RBV in HCV-1 patients; pooled SVR12 was 85.6% (CI 81.3% to 89.0%) [4]. Further, in a randomized trial using peg-INF, RBV with SMV in 151 patients with HCV-1b, the SVR24 was achieved in an average of 75.9% patients and adverse events leading to the discontinuation of treatment were reported in an average of 9% of participants [5]. Additionally, in a study of 120 patients with cirrhosis and contraindications to peg-INF/ RBV who were treated with SMV/SOF, the SVR12 in patients with Child class A, B, and C was 87, 77, and 67%, respectively. 11% of the patients developed severe adverse events, including sepsis, variceal bleeding, hepatocellular carcinoma, and hyperbilirubinemia [6]. Interestingly, in a large prospective observational cohort study including 836 patients receiving a 12-week regimen of SMV/SOF +/- RBV, patients with cirrhosis, prior decompensation, and previous protease inhibitor treatments were less likely to achieve an SVR regardless of RBV addition [7]. However, SMV/SOF +/- RBV combination appears to be safe and effective in treatment-naive and prior null-responder HCV genotype 1-infected patients without cirrhosis treated for 12 weeks [2]. Keeping patients’ best interests in mind, I recommend sparing this regimen to nonstructural protein 5A relapsers [8] considering it as first line only in treatment-naive non cirrhotic patients.

References

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