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Commentary

Genes Facilitate the Development of Hepatocellular Carcinoma

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ABOUT THE STUDY

Direct-acting antivirals (DAAs) have achieved a sustained virological response rate (SVR) of 9599% in the treatment of HCV. Several studies suggest that treatment with sofosbuvir (SOF), including DAA, may be associated with an increased risk of developing HCC. The purpose of this study is to investigate the potential mechanism of SOF in the development of HCC. In Taiwan, the prevalence of hepatitis C virus (HCV) infection is about 2-5%, and HCV is the leading cause of cirrhosis and hepatocellular carcinoma (HCC) worldwide. In patients with acute HCV infection, 60-90% develops chronic infection after 20-30 years of chronic infection, and 20-30% develops cirrhosis or HCC. Anti-HCV therapy has made great strides in recent years. Understanding the life cycle of HCV has led to the development of replicative cell culture systems as well as the discovery of many potential targets for direct-acting antiviral (DAA) agents. Several oral treatment regimens that combine DAA from different families, including NS5B nucleotide inhibitor (NI), dNS5B non-nucleoside inhibitor (NNI), NS5A replication complex inhibitor and NS3 / 4A protease inhibitor (PI) It was developed. Sofosbuvir (SOF) based regimens are commonly used in the treatment of chronic hepatitis C (CHC), with 95-97% persistent virology in compensatory cirrhosis and 85-90% in patients with advanced liver disease. It brought about the response efficiency (SVR).

Overall, several recent studies have shown that DAA treatment of HCV may induce changes in the inflammatory microenvironment associated with the development of HCC. This may partially explain the increased incidence of HCC in the clinical setting. However, few studies have aimed to identify host risk factors associated with the development of HCC after DAA treatment. We have found that HCV-induced epigenetic changes reprogram host gene expression and persist after viral eradication. These results may explain why some CHC patients progress to the development of HCC after HCV eradication. However, no studies have addressed the effects of SOF upregulation genes on the development of HCC in CHC patients. This study found that SOF increased cell proliferation and migration of HCC cells. According to the Cancer Genome Atlas (TCGA) database, several SOF upregulation genes identified by next-generation sequencing (NGS) are known to be associated with the development of HCC and among them, some genes were associated with overall survival and disease-free survival. Depletion of these genes reduced cell proliferation and migration increased by SOF in HCC cells.

In summary, SOF upregulated genes have been shown to be associated with the carcinogenesis of several types of cancer. Nonetheless, detailed mechanisms of how these genes regulate the development of HCC require further experimentation to elucidate them. The results of this study suggest an association between multiple SOF upregulation genes and increased cell proliferation and migration, which may contribute to the development of HCC.

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