



Viral Hepatitis C and Impact of Direct-Acting Antivirals on Liver Transplant

Emily Richards*

Department of Hepatology, Harvard University, Boston, United States of America

*Corresponding Author: Emily Richards, Department of Hepatology, Harvard University, Boston, United States of America; E-mail: emily.richards@harvard.edu

Received date: 26 August, 2024, Manuscript No. JLDT-24-151931;

Editor assigned date: 28 August, 2024, PreQC No. JLDT-24-151931 (PQ);

Reviewed date: 11 September, 2024, QC No. JLDT-24-151931;

Revised date: 18 September, 2024, Manuscript No. JLDT-24-151931 (R);

Published date: 25 September, 2024, DOI: 10.4172/2325-9612.1000281

Description

Hepatitis C Virus (HCV) infection has long been a global health challenge, known for its ability to cause chronic liver disease, cirrhosis and Hepatocellular Carcinoma (HCC). Prior to the advent of Direct-Acting Antivirals (DAAs), the treatment landscape for HCV was limited and often ineffective, with interferon based therapies offering suboptimal cure rates and numerous side effects. However, the introduction of DAAs revolutionized the management of HCV by offering highly effective and well-tolerated treatment options. HCV is a blood borne virus that primarily affects the liver [1-3]. Chronic infection can lead to progressive liver damage, cirrhosis and in some cases, liver cancer. According to the World Health Organization (WHO), approximately 58 million people worldwide are living with chronic HCV infection. Cirrhosis, resulting from prolonged liver inflammation due to the virus, is a leading cause of liver transplants, with HCV accounting for a significant portion of the cases requiring transplantation. The prognosis for patients with chronic HCV who develop cirrhosis is poor if left untreated. The liver's ability to regenerate is overwhelmed by the continuous cycle of inflammation and fibrosis, leading to the gradual loss of function. In these patients, liver transplantation becomes the only viable option when end-stage liver failure occurs. Additionally, HCV-related liver cancer (HCC) further complicates the clinical scenario, as it is one of the most common indications for transplantation in many parts of the world [4,5].

Before the beginning of DAAs, the standard treatment for chronic HCV was a combination of interferon and ribavirin, sometimes augmented with protease inhibitors. This treatment, although capable of achieving Sustained Virologic Response (SVR), was associated with several side effects, including flu-like symptoms, fatigue, depression and hematologic abnormalities. The cure rates varied widely depending on the HCV genotype, with some genotypes being more resistant to treatment [6,7]. Despite these limitations, interferon based therapy remained the cornerstone of HCV treatment for decades. Direct-Acting Antivirals (DAAs) target specific enzymes in the HCV replication cycle, directly inhibiting the virus's ability to replicate. Unlike interferon-based therapies, DAAs are well tolerated, with minimal side effects and much shorter treatment durations (typically 8 weeks to 12 weeks). The drugs are often used in combination to prevent resistance and optimize treatment efficacy.

Some DAAs include sofosbuvir, ledipasvir, daclatasvir and glecaprevir, among others [8].

The most important benefit of DAAs is their ability to halt the progression of liver disease. Studies have shown that achieving an SVR with DAAs significantly reduces the risk of liver disease progression. Cirrhosis patients who achieve SVR exhibit improvements in liver function, a reduction in liver stiffness and even a reversal of fibrosis in some cases. Importantly, the risk of Hepatocellular Carcinoma (HCC) also decreases after sustained viral clearance. While the risk of HCC does not disappear entirely in patients with advanced cirrhosis, the reduction in viral replication and inflammation offers a much better prognosis than that associated with ongoing chronic infection [9,10]. For patients without cirrhosis, DAAs effectively prevent the development of more severe liver disease. Liver transplantation is typically required for patients with end-stage liver disease resulting from chronic HCV infection. Prior to the widespread use of DAAs, the increasing burden of HCV-related cirrhosis and HCC led to a growing demand for liver transplants, outpacing the availability of suitable donor organs. The use of DAAs has had a profound impact on the need for liver transplants in HCV infected patients. As more individuals achieve SVR and their liver function improves, the need for liver transplantation has decreased. Several studies have demonstrated that the rate of liver transplantation due to HCV has dropped significantly in regions with high DAA treatment coverage.

Conclusion

DAAs have transformed the management of HCV, providing highly effective treatment options with minimal side effects. Their ability to cure chronic HCV infection and reduce the progression of liver disease has had a deep impact on the need for liver transplants, reducing the incidence of HCV-related cirrhosis and hepatocellular carcinoma. As access to DAAs continues to increase globally, the burden of HCV-related liver transplants is expected to decrease, improving the prognosis for millions of individuals living with chronic hepatitis C.

References

1. Poynard T, Yuen MF, Ratzin V, Lai CL (2003) Viral hepatitis C. *Lancet* 362(9401):2095-2100.
2. Levrero M (2006) Viral hepatitis and liver cancer: The case of hepatitis C. *Oncogene* 25(27):3834-3847.
3. Szabo E, Lotz G, Paska C, Kiss A, Schaff Z (2003) Viral hepatitis: New data on hepatitis C infection. *Pathol Oncol Res* 9:215-221.
4. Manns MP, Wedemeyer H, Cornberg M (2006) Treating viral hepatitis C: Efficacy, side effects and complications. *Gut* 55(9):1350-1359.
5. Zein NN (2000) Clinical significance of hepatitis C virus genotypes. *Clin Microbiol Rev* 13(2):223-235.
6. Nguyen MH, Keeffe EB (2005) Prevalence and treatment of hepatitis C virus genotypes 4, 5 and 6. *Clin Gastroenterol Hepatol* 3:S97-101.
7. Alter MJ (2007) Epidemiology of hepatitis C virus infection. *World J Gastroenterol* 13(17):2436.

8. Rehermann B, Nascimbeni M (2005) Immunology of hepatitis B virus and hepatitis C virus infection. *Nat Rev Immunol* 5(3): 215-229.
9. Thomson BJ, Finch RG (2005) Hepatitis C virus infection. *Clin Microbiol Infect* 11(2):86-94.
10. Thomas DL, Astemborski J, Rai RM, Anania FA, Schaeffer M, et al. (2000) The natural history of hepatitis C virus infection: Host, viral and environmental factors. *Jama* 284(4):450-456.