

HCV Infection in Thalassemia Syndromes and Hemoglobinopathies

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ABSTRACT

Until 1990, hepatitis C virus (HCV) infection was one of the most severe complications of transfusion therapy in the thalassemia and sickle cell disease (SCD) population; in reality, serological tests to detect infection in blood donors have been available since 1990. Iron chelation therapy has extended the lives of these patients, resulting in a reduction in heart disease-related deaths as well as a rise in liver disease caused by iron overload and HCV infection, which leads to liver fibrosis, cirrhosis, and hepatocellular carcinoma. Until recently, the recommended treatment for HCV was pegylated-interferon alpha plus ribavirin, a treatment.

Keywords: Hepatitis C virus; Sickle cell disease; Carcinoma

INTRODUCTION

Hepatitis C virus (HCV) infection is one of the most severe side effects of transfusion therapy in people with thalassemia and Sickle Cell Disease (SCD) who received transfusions before 1990; in reality [1], serological tests to identify infections in blood donors have been available since 1990. Iron chelation therapy has improved the life expectancy of thalassemia patients, with a reduction in heart disease-related deaths and an improvement in liver disease-related deaths [2].

Iron overload and HCV infection have been linked to the development of liver fibrosis, cirrhosis, and hepatocellular carcinoma in thalassemia patients. Just 15–25% of infected people will get rid of the infection on their own. The remaining 75–85 percent of people experience cirrhosis, which can lead to liver failure and hepatocellular carcinoma. Cirrhosis prevalence ranged from 10% to 20% in many studies from different populations of thalassemic patients (the United States, Italy, and Greece), and the incidence of HCC in thalassemia patients was steadily increasing [3].

The significance of eradicating HCV infection has increased as a result of these findings. Around 10–20 percent of patients with Sickle Cell Anemia have chronic HCV infection. Furthermore, since blood transfusions are needed to treat the sickling crisis and anemia, patients with HCV infection are at a higher risk of iron overload and hemosiderosis, which can contribute to liverrelated morbidity and mortality [4]. Hepatic involvement can vary in severity from liver dysfunction to liver failure, and it can be caused by a variety of factors, including Acute hepatic vaso occlusion, hepatic sickle cell cholestasis, liver ischemia and reperfusion damage, hemolysis, and cholelithiasis are all symptoms of the sickling phase. HCV is a heterogeneous virus that has been described as having at least seven genotypes. Each genotype contains several subtypes that vary by 31 to 33 percent across the entire viral genome. This genetic heterogeneity has an effect on the effectiveness of antiviral therapy in terms of response rate, as it must be adapted to various viral strains in terms of treatment type and length. The distribution of genotypes and subtypes affects the effectiveness of antiviral therapies, so getting a thorough understanding of HCV genotype has a number of clinical implications. The experience of patients with thalassemia and sickle cell disease treated with DAAs therapy at the Thalassemia Unit of Rome's S [5].

Eugenio Hospital is presented in this review. The analysis was carried out in compliance with GCP principles, the Declaration of Helsinki, and all applicable local regulations. Treatments with DAAs were carried out in accordance with national guidelines and were approved by the Italian Medicines Agency [6]. To participate in this study, all patients signed a written informed consent form. Irritation at the injection site, febrile influenza-like symptoms, psychiatric illnesses, thyropathy, neutropenia, and hemolytic anemia were all common side effects of this procedure. Due to the presence of subjects who did not respond to therapy or experienced recurrences, the infection was not eradicated as predicted. When there is a sustained virological response (SVR), which is characterized as the absence of HCV RNA in serum by a sensitive test performed 24 weeks after the end of antiviral therapy; infection is believed to be eradicated. In patients with thalassemia and HCV infection, PEG-IFN plus RBV resulted in SVR rates of 25-64 percent.

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However, since ribavirin (RBV) induced hemolytic anemia and increased blood transfusions, as well as interferon side effects including influenza-like symptoms, depression, and cytopenias, this medication was not well tolerated by thalassemic significant side effects. Due to the hemolytic anemia caused by ribavirin, which increased the number of blood transfusions, this therapy has been restricted to thalassemic and SCD patients and SCD patients, and its use has been severely reduced [7]. All of the treatment regimens were well-tolerated, and there were no recorded side effects. The ongoing iron-chelation therapy for no patient has improved, and the number of blood transfusions has remained unchanged [8].

CONCLUSION

According to the data currently available in the literature, the latest DAAs are very close to eradicating HVC infection. Furthermore, the findings obtained on patients with thalassemia and sickle cell disease have consistently yielded high SVR values, often greater than 90%. There are still some non-responder subjects to DAAs therapy, and one of the reasons may be that some of them were already very compromised with severe comorbidities when they were treated, as in our non-responder subjects with infection relapse after 1 month. Furthermore, the care of patients did not initially include any of the new forms of DAAs that are currently available. The analysis was carried out in compliance with GCP principles, the Declaration of Helsinki, and all applicable local regulations.

Treatments with Direct-acting Antiviral Agents are carried out in compliance with national guidelines and have been approved by the Italian Medicines Agency (AIFA).

REFERENCES

- 1. Heit JA. Epidemiology of venous thromboembolism. Nat Rev Cardiol. 2015;12:464-474.
- Donadini MP, Ageno W. Unusual site thrombosis. Semin Hematol. 2011;48:264-270.
- 3. Hanson PO, Welin L, Tibblin G, Eriksson H. Deep vein thrombosis and pulmonary embolism in the general population. Arch Intern Med. 1997;157:1665-1670.
- 4. Ribeiro DD, Lijfering WM, Barreto SM, Rosendaal FR, Rezende SM. Epidemiology of recurrent venous thrombosis. Braz J Med Biol Res. 2012;45:1-7.
- Poort SR, Rosendaal FR, Reitsma PH, Bertina RM. A common genetic variation in the 3'-untranslated region of the prothrombin gene is associated with elevated plasma prothrombin levels and an increase in venous thrombosis. Blood. 1996;88:3698–3703.
- 6. Martinelli I, Bucciarelli P, Mannucci PM. Thrombotic risk factors: basic pathophysiology. Crit Care Med. 2010;38:S3-9.
- 7. Zoller B, de Frutos PG, Hillarp A, Dahlback B. Thrombophilia as a multigenic disease. Haematologica. 1999;84:59-70.
- Anderson FA, Wheeler HB, Goldberg RJ, Hosmer DW, Patwardhan NA, Jovanovic B, et al. A population-based perspective of the hospital incidence and case-fatality rates of deep vein thrombosis and pulmonary embolism: the Worcester DVT Study. Archives of internal medicine. 1991;151:933-938.