The Impact of Hepatitis C Microelimination Strategies in HCV/HIC Co-Infected Individuals: A Current State of Art
Ibraheem M Alkhawaldeh¹, Mostafa Hossam El din Moawad ²,³, Mohammad Al-Jafari¹,⁴*

Dear Editor,

Some fights are rising above others in our effort to achieve better public health outcomes, and one of them is undoubtedly the fight against viral hepatitis. This complex, multidisciplinary challenge has drawn attention worldwide and prompted the World Health Organization (WHO) to establish the ambitious aim of eliminating viral hepatitis as a serious public health hazard by 2030. This attempt is complicated, and one of its most important aspects is the interaction of two formidable viruses: hepatitis C virus (HCV) and human immunodeficiency virus (HIV).

People living with HIV and chronic HCV infection suffer from a significant serious illness. Globally, an estimated 2.3 million people are co-infected with both HIV and HCV; 37 million people who are HIV positive are more at risk of getting HCV than those who do not have HIV. HIV infection is associated with a significantly increased incidence of liver fibrosis and cirrhosis, as well as a rapid progression to HCV-related liver disease (1,2).

The WHO set the aim of eliminating viral hepatitis as a major public health hazard by 2030. The objective was announced in June 2016 and included a 90% reduction in new cases of viral hepatitis and a 65% reduction in hepatitis-related fatalities. In 2016, a worldwide strategy to restrict the spread of the hepatitis C virus (HCV) was formed, which includes screening donated blood products, harm reduction initiatives aimed at persons who inject drugs (PWID), and increasing the availability of direct-acting antiviral (DAA) drugs(3).

HIGH-RISK GROUPS FOR HCV AND HIV COINFECTION

Since 2000, there has been an increase in reports of HCV infections in Europe, the United States, Australia, and other Asian-Pacific countries. During this emerging pandemic, however, HCV infection has been proven to spread through sexual encounters, notably among men who have sex with men (MSM), rather than parenteral exposures to infected blood via injectable drug use, use of contaminated medical equipment or transfusion. High quantities of HCV have been identified in the nasal, sperm, and rectal secretions of HCV-viremic people, particularly those who are also HIV-infected. These findings imply that violent, unprotected sex experiences may increase the risk of developing HCV.(4).
According to multiple studies done on people living with HIV (PLWH) who were MSM with HCV coinfection, there is a strong relationship between the diagnosis of other sexually transmitted infections (STIs), such as syphilis, and the recent acquisition of HCV.(4).

MICROELIMINATION STRATEGIES

Microelimination of HCV/HIV coinfection is a viable strategy for reaching HCV targets globally. Other towns or jurisdictions dealing with comparable HCV/HIV epidemics have similar goals(1). The microelimination program, which began in February 2016, had screening and treatment components, both of which were designed in line with French guidelines for the treatment of HIV co-infections (5).

HCV serology was performed during the screening phase on individuals who had a previous negative result for more than 12 months or who had an unavailable test; in cases of positive serology, HCV RNA was tested. When a patient’s prior HCV RNA quantification was more than six months old, or there were risk factors for HCV reinfection, HCV RNA was systematically controlled after HCV treatment. In patients who spontaneously recovered from HCV infection, HCV RNA was performed if the previous test was more than 12 months old(5).

As part of the program’s therapeutic component, all patients with positive HCV-RNA quantification obtained information and a treatment plan. Since DAA for HCV treatment has a shorter half-life, our goal was to treat every patient for one year. To accomplish this goal, a particular plan was put in place that included: (i) a hepatologist visit, a nurse educator consultation, blood tests comprising of HCV-RNA quantification, liver ultrasound and elastometry to detect liver fibrosis;(ii) a clinical visit schedule, nurse education sessions, and laboratory measures agreed upon with the patient at the start of therapy and performed monthly during treatment and then at week (W) 4, W12, W24, and W48 after treatment was completed. In determining treatment methods (DAA and treatment duration), present French criteria were followed, which were then evaluated by multidisciplinary specialists. The goal was to assess the pharmacological interactions between anti-HCV drugs, antiretrovirals, and/or comorbidity management(5).

After DAAs were implemented in NHS Tayside, there was a rise in the percentage of coinfected patients who started and finished their HCV treatment effectively. The region’s attempts to microeliminate coinfection were significantly supported by the availability of DAAs and the co-location of blood-borne virus (BBV) services as part of a multi-stakeholder approach. These strategies should be taken into consideration elsewhere(6).

OUTCOMES OF MICROELIMINATION STRATEGIES

According to Chen et al., the implementation of DAA treatment in Taiwan resulted in a 53.2 % and a 66.4 % decline in the incidence and prevalence of HCV viremia, respectively, as compared to the epidemiological maximum. However, maintaining the efficiency of HCV microelimination would require addressing HCV reinfections in high-risk subpopulations(3).

According to Santos et al., even after HCV microelimination, PLWH in southern Spain continue to have a high burden of severe liver damage (SLD), defined as liver stiffness (LS) ≥7.2 KPa. Furthermore, levels of LS consistent with advanced liver disease are seen in more than 25% of SLD patients. In this situation, SLD is caused by persistent liver damage from a recovered HCV infection. However, one in every three individuals with this condition has a non-viral cause. The majority of these individuals with nonviral liver damage had data consistent with metabolically associated fatty liver disease or alcoholic liver disease (7).

With HCV cure, the frequency of liver-related outcomes and extrahepatic effects drops rapidly; nonetheless, some PLWH still have residual liver damage, which presents as abnormal liver function. This shows that this is currently the most prevalent cause of liver damage among PLWH. Due to remaining liver damage, the risk of developing cirrhosis decompensations or hepatocellular carcinoma is not removed after a sustained viral response. This implies that complications must be monitored(7).

AUTHORS’ CONTRIBUTION

REFERENCES


