Case Report

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Hepatitis B and Hepatitis C Co-infection: Treatment Approaches and A Case Report

Hepatit B ve Hepatit C Ko-enfeksiyonu: Tedavi Yaklaşımları ve Bir Olgu Sunumu

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ABSTRACT

Hepatitis B virus (HBV) and hepatitis C virus (HCV) co-infection is a significant health issue worldwide and can accelerate liver damage when they occur together. This study presents the treatment process of a 52-year-old female patient with HCV and HBV co-infection. The patient was initiated on direct-acting antiviral therapy (glecaprevir and pibrentasvir) for HCV infection and entecavir for HBV reactivation. As a result of treatment, both HCV-RNA and HBV-DNA became negative, and a sustained virologic response was achieved for HCV. Our study highlights the effectiveness of modern treatment approaches for patients with HBV and HCV co-infection.

Keywords: Chronic hepatitis C, chronic hepatitis B, chronic hepatitis B and C, direct-acting antiviral

ÖZ

Hepatit B virüs (HBV) ve hepatit C virüs (HCV) ko-enfeksiyonu, dünya genelinde önemli sağlık sorunları arasındadır ve birlikte oldukları durumlarda karaciğer hasarının ilerlemesini hızlandırabilir. Bu çalışmada, 52 yaşındaki bir kadın hastanın HCV ve HBV ko-enfeksiyonu ile tedavi süreci sunulmuştur. Hastaya, HCV için doğrudan etkili antiviral tedavi (glekaprevir ve pibrentasvir) ve HBV reaktivasyonu için entekavir başlanmıştır. Tedavi sonucunda hem HCV-RNA hem de HBV-DNA negatifleşmiş ve HCV için kalıcı viral yanıt elde edilmiştir. Çalışmamız, HBV ve HCV ko-enfeksiyonu olan hastalarda modern tedavi yaklaşımlarının etkinliğini vurgulamaktadır.

Anahtar Kelimeler: Kronik hepatit C, kronik hepatit B, kronik hepatit B ve kronik hepatit C, direkt etkili antiviral

Introduction

Hepatitis B virus (HBV) and hepatitis C virus (HCV) are important causes of morbidity and mortality worldwide. Chronic HBV and HCV co-infection occurs when both viruses cause infection simultaneously, with a prevalence of approximately 1-15% globally (1). The presence of both HBV and HCV can accelerate the progression of liver disease and increase the risk of complications such as cirrhosis and hepatocellular carcinoma (HCC) (2). The treatment of HBV and HCV co-infections is an important clinical experience for physicians. For this reason, we considered sharing our case as a practical contribution. Informed consent for this case report was obtained from the patient.

Case Report

A 52-year-old female patient with no known comorbidities was referred to our clinic after routine tests conducted prior to surgery for benign breast disease revealed positive anti-HCV and hepatitis B surface antigen (HBsAg) results. The patient's family history did not include hepatitis or cirrhosis, and she had no history of intravenous drug use or previous surgical interventions. It was noted that she had multiple regular sexual partners.

Laboratory tests showed negative anti-human immunodeficiency virus, anti-hepatitis B core immunoglobulin M, anti-hepatitis B e antigen (anti-HBeAG), anti-HBe, negative hepatitis delta virus Ag (HDV Ag), and negative HDV-RNA. Liver function tests indicated aspartate aminotransferase (AST): 23 IU/L, alanine aminotransferase (ALT): 18 IU/L; total bilirubin: 0.3 mg/dL, international normalized ratio: 1.2, platelet count: 280,000/mm^3, and hemoglobin: 13.8 g/dL. Thyroid function tests and autoantibodies were within normal limits. The patient's HCV-RNA and HBV-DNA levels were 4,270,000 and 7,950 IU/mL, respectively. HCV genotype analysis revealed genotype 1B. Abdominal ultrasound did not reveal any pathological findings

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except for "mild granulation in the liver parenchyma". Liver biopsy results showed fibrosis stage 2/6 and HAI score of 8/18.

In the treatment planning, direct-acting antiviral (DAA) therapy was initiated for HCV, along with a nucleoside/nucleotide inhibitor for HBV prophylaxis. The treatment regimen for the treatment-nave, noncirrhotic genotype 1B patients included a combination of glecaprevir (100 mg) and pibrentasvir (40 mg) administered 1x3/day for 8 weeks. Additionally, entecavir 0.5 mg/day was started for HBV. At the 4th week of treatment, follow-up tests showed HBV-RNA-negative, HBV-DNA at 484 IU/mL, AST at 32 IU/L, and ALT at 24 IU/L. Twelve weeks after initiating treatment, both HCV-RNA and HBV-DNA were negative, indicating that no viral genome was detected. The 24th week of follow-up showed a sustained virologic response.

Discussion

HBV and HCV co-infection can lead to a more complex clinical picture and faster progression of liver diseases compared with mono-infection. Co-infection often results in a more aggressive disease course and increased risk of complications. The presence of both HBV and HCV elevates the risk of liver fibrosis and cirrhosis and increases the risk of developing HCC. In particular, the co-existence of both viruses complicates treatment and monitoring processes, necessitating a multidisciplinary approach (3).

Co-infections typically manifest in three ways: co-dominant, HBV-dominant, and HCV-dominant. In our case, the HCV-RNA copy number was significantly higher than that of HBV-DNA, indicating HCV-dominant coinfection, and treatment was conducted according to protocols recommended for HCV mono-infected patients. This approach has shown successful results when HCV is more dominant than HBV (4,5).

In recent years, interferon-based treatments have been replaced by DAA drugs, and these drugs have provided a revolutionary development in the treatment of HCV. DAAs such as glecaprevir and pibrentasvir have high efficacy rates and provide high treatment success in coinfected patients. As observed in this case, the HCV-RNA of the patient treated with the combination of glecaprevir and pibrentasvir turned negative as of the 4th week, and a sustained viral response was achieved at the end of the treatment. The low side effects and high effectiveness of these treatment protocols have made them the primary choice for HCV treatment (6).

However, the risk of HBV reactivation increases with HCV treatment. Therefore, before initiating HCV treatment, prophylactic treatment should be initiated against the risk of HBV reactivation. Prophylaxis for HBV should be administered during DAA treatment for at least 12 more weeks after treatment ceased (7). In this case, entecavir treatment was initiated successfully to prevent HBV reactivation. At the end of the treatment period, HBV reactivation was not observed.

This case demonstrates the effectiveness and safety of DAA treatment in patients with HBV-HCV co-infection and highlights the points that should be taken into consideration for the management of the risk of HBV reactivation. In patients with HBV and HCV co-infection, it is vital to carefully monitor the viral load and to take precautions against possible complications during treatment.

Conclusion

Chronic HBV and HCV co-infection leads to a more complex clinical picture and a more aggressive disease course compared with mono-infections. DAAs have replaced traditional interferon-based therapies, particularly in HCV-dominant HBV/HCV co-infected patients, due to their lower side effects and high treatment success rates. However, the risk of HBV reactivation after DAA treatment should be considered, and patients should be monitored with appropriate prophylaxis. Treatment selection in co-infected patients should be based on the dominant virus type and side effect profile.

Ethics

Informed Consent: Informed consent was obtained from all participants.

Acknowledgments

This case report presented as a poster in Infectious Diseases and Clinical Microbiology Specialty Society of Turkey 2022 congress, Antalya.

Footnotes

Autorship Contributions

Surgical and Medical Practices: M.B., T.D., Concept: M.B., T.D., Design: M.B., T.D., Data Collection or Processing: M.B., T.D., Analysis or Interpretation: M.B., T.D., Literature Search: M.B., T.D., Writing: M.B., T.D.

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