LETTER TO THE EDITOR

Readers are encouraged to write letters to the editor concerning articles that have been published in Clinical Gastroenterology and Hepatology. Short, general comments are also considered, but use of the Letters to the Editor section for publication of original data in preliminary form is not encouraged. Letters should be typewritten and submitted electronically to http://www.editorialmanager.com/cgh.

Occult Hepatitis B and Risk of Reactivation After Hepatitis C Treatment With Direct-Acting Antivirals

Dear Editor:

In their observational study Wang et al[1] determined the reactivation of hepatitis B virus (HBV) in coinfected patients treated with direct-acting antivirals (DAAs). They studied hepatitis B surface antigen (HBsAg)-positive patients and occult HBV infections (OBIs) and reported 3 reactivations in 10 HBsAg-positive patients and none in 124 OBI cases.

Reactivation of HBV after hepatitis C virus treatment with DAA is an emerging issue in coinfected patients. There have been at least 6 cases in the medical literature.[2–6] Among these patients, 2 developed liver failure and underwent liver transplantation. Another patient without flare showed an increase in HBV DNA level (from 3 log copies/mL to 4.3 log copies/mL) after peginterferon + ribavirin + simeprevir therapy and was given entecavir treatment.[7] Recently, U.S. Food and Drug Administration Adverse Events Reporting System database reported 29 cases (which may include the published cases) of HBV reactivation between November 2013 and October 2016.[8] Among these patients, 3 developed hepatic decompensation; 1 required liver transplantation, and 2 died.

In the current study, no reactivation was reported in OBI group. However, OBI status has not been clearly defined. In an international workshop, OBI has been defined as the detection of HBV DNA in the liver (with or without HBV DNA in serum) without HBsAg.[9] In clinical practice, it refers to the presence of HBV DNA in the absence of HBsAg.[10] The diagnosis of OBI clearly depends on HBV DNA analysis; however, HBV DNA level in OBI group was not provided in the study. The characteristics of reactivation including the change in HBV DNA levels, any HBV treatment to control the reactivation, and the outcomes were not provided, which makes it complicated to draw conclusions about reactivation risk in OBI group.

RESAT OZARAS, MD
BILGUL METE, MD
FEHMI TABAK, MD
Infectious Diseases Department
Cerrahpasa Medical School
Istanbul University
Istanbul, Turkey

References

1. Wang C, et al. Clin Gastroenterol Hepatol 2017;15:132–136.
2. Takayama H, et al. Hepatology Res 2016;45:489–491.
3. Collins JM, et al. Clin Infect Dis 2015;61:1304–1306.
4. Ende AR, et al. J Med Case Rep 2015;9:164.
5. De Monte A, et al. J Clin Virol 2016;78:27–30.
6. Hayashi K, et al. Clin J Gastroenterol 2016;9:252–256.
7. Kimura H, et al. Kanzo 2015;56:422–427.
8. Bersoff-Matcha SJ, et al. Program and abstracts of the 2016 Annual Meeting of the American Association for the Study of Liver Diseases; November 11–15, 2016; Boston, Massachusetts. Abstract LB-17.
9. Raimondo G, et al. J Hepatol 2008;49:652–657.
10. Kwak MS, et al. World J Hepatol 2014;6:860–869.

Conflicts of interest
The authors disclose no conflicts.

http://dx.doi.org/10.1016/j.cgh.2016.11.030