

Risk of Reactivation of Latent HBV Hepatitis in Patients Under Neurosurgical Treatment

Yoshikazu Ogawa^{1*} and Teiji Tominaga²

¹Department of Neurosurgery, Kohnan Hospital, Japan

²Departments of Pathology and Neurosurgery, Tohoku University Graduate School of Medicine, Japan

*Corresponding author: Yoshikazu Ogawa, Department of Neurosurgery, Kohnan Hospital, 4-20-1 Nagamachiminami, Taihaku-ku, Sendai, Miyagi 982-8523, Japan, Tel: +81222482131; Fax: +81223041641; E-mail: yogawa@kohnan-sendai.or.jp

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Abstract

Reactivation of hepatitis B virus (HBV) is a risk in the 350 million HBV carriers worldwide. HBV reactivation may cause hepatocellular carcinoma, cirrhosis, and fulminant hepatitis, and HBV reactivation accompanied with malignant tumor and/or chemotherapy is a critical problem for the patients with chronic HBV infection. In addition, multiple risk factors causing immunosuppressive state can also induce HBV reactivation, which includes a few cases of intrinsic cortisol over secretion as Cushing's syndrome or very low-dose steroid treatment for hypopituitarism after pituitary surgeries.

Appropriate screening methods and the discussion for preventive supplementation of antiviral drugs for HBV reactivation are required. For patients with pituitary tumors precise operative procedures and careful treatment planning are essentially required to avoid HBV reactivation.

Short Communication

Today HBV reactivation and hepatitis flare induced by cytotoxic chemotherapy has a keen interest in cancer patients with chronic HBV infection, especially in patients with hematological malignancies. Immunosuppressive state caused by immunosuppressive agents and anticancer drugs could precipitate an increase in HBV replication followed by a flare of hepatitis B, which may become severe or even fatal [1-9]. Inhibition of epigenetic regulator for the carcinoma therapy might directly lead the reactivation of latent HBV hepatitis or even HIV-1 [10]. Most patients who develop HBV reactivation are urged to suspend or postpone treatment of the underlying diseases, so establishment of preventive strategy in high-risk patients is extremely important.

One patient with Cushing's syndrome and adrenal adenoma died of hepatic failure due to reactivation of HBV [5]. The immunosuppressive state in this patient resulted from intrinsic cortisol over secretion, which was considered to be the cause of reactivation of HBV. Immediate treatment is needed for HBV reactivation with both antiviral therapy and simultaneous remission from the immunosuppressive state. So early surgery after normalization of hepatic function is the best treatment strategy. This principle should be available for possible occurrence of brain tumors especially pituitary diseases. However great care is needed, as hypopituitarism is one of the serious complications after trans sphenoidal surgery and/or gamma knife therapy [11,12], and very low-dose steroid treatment can induce HBV reactivation [13]. Because postoperative hypopituitarism can result in HBV re-activation, avoidance of treatment-induced hypopituitarism is essentially required. The extreme care is needed for prevention of damage of pituitary gland, as delicate manipulation in surgery and making sufficient distance from residual tumor for the possible gamma knife therapy thereafter. Compatibility of this

treatment dilemma could bring minimization of steroid supplementation and avoidance of HBV re-activation.

The strategy to prevent HBV reactivation is not well established. Strict monitoring or universal prophylaxis for hepatitis B core antibody-positive hepatitis is more appropriate and cost-effective with non-Hodgkin lymphoma [14]. Indications of the patients, the choice of antiviral drugs, and the duration of antiviral drug supplementation have to be established, and further investigations are needed to determine the appropriate screening methods and preventive supplementation of antiviral drugs for HBV reactivation.

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