Serum HBV DNA and ALT Kinetics in The Trials of Antiviral Agents in Patients with Chronic Hepatitis B (Appropriate sampling time for Response Assessment of Antiviral Therapy in Chronic Hepatitis B Patients)

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Hepatitis B virus (HBV) infection is a widespread disease that affects about 350 million people worldwide, contributing to global public health problems and 25-40% of these will die from liver cirrhosis or primary hepatocellular carcinoma [1-3]. Therapeutic tools for patients with chronic HB include the nucleoside analogue lamivudine and cytokine interferon α (IFN-α) based on several credible clinical trials [3-5]. In the study by Dienstag et al. [3] and Jonas et al. [4] provide the reliable results about antiviral agent, lamivudine that was well tolerated and suppressed serum levels of HBV DNA, viral replication marker, profoundly in patients with chronic HB in children and adult population. They conducted same sampling time for assessment of efficacy of antiviral agents about virological markers HBV DNA and hepatitis B antigen (HBeAg) and biochemical markers, serum alanine aminotransferase (ALT) in patients with HBV infection.

Consistently, because clinical observations and animal studies clearly show that abolish of HBV DNA by HBV-specific cytotoxic T lymphocytes always precedes changes of ALT level as a marker of hepatocellular inflammation and nercosis in chronic hepatitis B [4-7], we suggest to consider different profiles of serum ALT and viral HBV DNA kinetics after administration of antiviral agents in chronic HB patients to monitor responses about antiviral therapy against HBV infection. Therefore, we need to get samples with different time for assessment of viral and biochemical response after treatment with antiviral agents in patients with chronic HB.

Generally speaking, responses to antiviral agents in treatment of patients with chronic HB are usually no detection of viral concentrations such as HBV DNA using non-amplified assays, sustained loss of HBeAg with or without detection of anti-HBe. HBeAg is a viral protein produced by infected cell. It is not that its production is not directly inhibited by lamivudine, but that it is inhibited by INF-α and this response thought to represent the lysis of infected hepatic cell by the host’s immune response and changes in the serum HBeAg concentration can be reflects changes in infected hepatic cell mass [6-8]. ALT is the hepatic cytoplasmic enzyme that is released from damaged cells and a reliable indicator of the level of cell damage and death [6].

Therefore, the strategies of antiviral regimen that primary endpoint is HBeAg seroconversion (undetectable HBeAg, detectable anti-HBe, and undetectable HBV DNA) has danger of viral mutation dependent on duration of treatment and demerits of high cost. Therefore, based on difference of serum HBV clearance and ALT kinetics in patients with chronic HB, we may consider that switching regimen with potent antiviral agents as a primary drugs to reduce the viral DNA concentrations to undetectable level and agents with anti-inflammatory effect to reduce serum ALT level below upper limit of normal range of ALT rather than combined regimen with antiviral agents and INF-α or adefovir to escape the viral mutation and high cost to control patients with chronic HB.

References
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