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PP-095 Dynamics of serum HBV DNA levels and MELD scores in fatal acute-on-chronic hepatitis B liver failure

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Background and Aims: Until now, it is still not very clear what impact the progression and result in fatal outcomes of acute-on-chronic hepatitis B liver failure (HB-ACLF). Model for end-stage liver disease (MELD) scores system is an internationally recognized to predict the pathogenetic condition of the end-stage liver disease. The study is to investigate the dynamics and clinical significances of serum HBV DNA levels and MELD scores in the terminal phase of HB-ACLF.
Methods: 106 fatal patients with HB-ACLF were enrolled, who had never accepted antiviral therapy or liver transplantation. At three phases of 29–90 d, 8–28 d and 1–7 d before death, they were detected serum HBV DNA levels by Realtime-PCR in order meanwhile MELD scores were calculated.
Results: In 35 patients with HBeAg positive, HBV DNA levels were 6.16±1.59 log10, 5.63±1.52 log10 and 5.12±2.13 log10 copies/ml while MELD scores were 19.03±7.51, 24.79±6.40 and 33.06±7.64 respectively. In 71 patients with anti-HBe positive, HBV DNA levels were 4.63±1.82 log10, 5.77±1.74 log10 and 4.69±1.68 log10 copies/ml while MELD scores were 24.01±6.32, 28.34±7.77 and 33.25±10.13 in sequence. HBV DNA levels of HBeAg positive group were significantly higher than that of anti-HBe positive group at the phase of 29–90 d ($P < 0.05$). But in the same group, HBV DNA levels were no significant difference among three phases, regardless of HBVe system status. With the exacerbation of the ill, MELD scores increased steadily and significant difference was found in all patients ($P < 0.05$).
Conclusions: To initiate HB-ACLF occurrence, HBeAg positive patients need higher serum HBV DNA levels than that of anti-HBe positive ones. Regardless of HBVe system status, sustained high HBV DNA levels may promote the disease worsened and be fatal to patients.

PP-096 Different clinical features of spontaneous decrease of HBV DNA level between HBeAg-positive and HBeAg-negative chronic hepatitis B patients

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Background: To compare the clinical features of spontaneous decrease of HBV DNA level between Hepatitis B e antigen (HBeAg)-positive and HBeAg-negative chronic hepatitis B patients (CHB).
Methods: Dynamic change of HBV DNA level was observed for 12 weeks in 315 cases with admission HBV DNA level above 10,000 copies/mL. All patients had never been treated with antiviral or immunoregulatory drugs during this period. In the first observation week, HBV DNA, HBeAg, ALT, AST, total bilirubin (TBIL), prothrombin time (PT) were detected. HBV DNA was redetected at the end of observation period. These cases were divided into subgroups according to HBeAg and HBV DNA decrease (HBeAg-negative group, HBeAg-positive group, and each was subgrouped into decreased subgroup and non-decreased subgroup).
Results: Among the 315 CHB patients, 171 patients (54.3%) underwent spontaneous decrease of HBV DNA level within 12 weeks. 83 (55.0%) of 151 HBeAg-positive patients underwent spontaneous decrease of HBV DNA level, while 88 (53.7%) of 164 HBeAg-negative patients underwent that. The difference between the above two groups was not significant ($\chi^2 = 0.054$, $P = 0.816$). Among HBeAg-positive patients, AST, TBIL, PT were higher in patients with spontaneous decrease of HBV DNA level than in those without ($t = -3.485$–-4.156, $P = 0.000$–0.001). Among HBeAg-negative patients, HBV DNA, ALT, AST, TBIL, PT were higher in patients with spontaneous decrease of HBV DNA level than in those withoutv ($t = -5.224$–2.476, $P = 0.000$–0.014).
Conclusion: Half of the observed CHB patients underwent spontaneous decrease of HBV DNA level, and the worse the hepatic function was, the higher incidence of spontaneous decrease of HBV DNA level would be whenever HBeAg were positive or not.

PP-097 A time delay anti-HBV infection treatment mathematical model with adefovir

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Background: The time delay mathematical models are often used to describe the process of HBV infection and treatment. Based on the basic virus infection model introduced by Nowak et al. and Park Soo's data of adefovir, we set up a time delay model to simulate the dynamic behavior of free viral, here the time delay stands for the time between viral entry into a target cell and production of new virus particles.
Methods: The model is described by three variables: *x, y, v* represent the numbers of susceptible cells, infected cells, and free virus. The model include 12 parameters: *l, d, c, h, b, a, n, k, u, xmax, t, e-mt*, where *c* is the maximum proliferation rate of target cells, *xmax* is the *x* population density at which proliferation shuts off, *h* and *n* represent the efficacy of the therapy, *e-mt* accounts for cells that are infected at time *t* but die before becoming productively infected *t* time units later, the meanings of *l, d, b, a, k, u* are the same as those given in Nowak's model. During treatment, we chose $\{ l, d, c, h, b, a, n, k, u, xmax, t, e\text{-}mt\} = \{2.525\times10^2, 3.79\text{e-}004, 3\times10^{-6}, 0.5, 40, 1.644\text{e-}003, 0.9999, 0.0749, 0.1679, 6.6623\text{e+}005, 1.8, e\text{-}0.00002\times1.8\}$.
Results: The simulation data of the free viral in our model is consistent with the clinical data perfectly.
Conclusion: The result shows that our time delay anti-HBV model may be used as a reference for comparing the efficiency of different drugs, and other HBV infection models.
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PP-098 The factors related to creatine kinase (CK) elevations in patients treated with telbivudine

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Background: Asymptomatic CK elevations were observed in patients who received the telbivudine treatment. This study is to analyze the factors related to this phenomenon.