Acute Hepatitis B treated with Lamivudine

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Sir,

Lamivudine is a nucleoside analogue with potent inhibitory effect on hepatitis B virus (HBV) replication. It has been approved for the treatment of chronic hepatitis B and several studies confirmed the efficacy and safety of the drug.¹ It has also been administered to treat acute exacerbations of chronic hepatitis B following either liver transplantation² or use of immunosuppressive drugs.³ Only a few case reports have been published regarding lamivudine treatment in acute hepatitis B.⁴ We describe a patient with severe acute hepatitis B who has been treated with lamivudine and made an uneventful recovery.

A 29 year-old-man, in previously good health, presented with a 7-day history of jaundice and fatigue. Physical examination revealed a well-nourished, conscious person with deep jaundice and hepatomegaly but no splenomegaly, palmar erythema or spider nevi. Laboratory tests demonstrated elevated aminotransferase levels (ALT 4030 IU/L and AST 1670 IU/L), hyperbilirubinemia (18.9 mg/dl) predominantly direct, prolonged prothrombin time (17.5 seconds with International Normalised Ratio/INR 1.5) and normal ammonia and albumin values. Serological tests were positive for hepatitis B surface antigen (HBsAg), IgM antibody to hepatitis B core antigen and hepatitis B e antigen (HBeAg). IgM antibody to hepatitis A virus, antibody to hepatitis C vi-

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Dr. Spyros P. Dourakis, 28 Achaias str., 115 23 Athens, Greece, Tel.: +210-6918464, Fax: +210-6993693, e-mail: spiros@ath.forthnet.gr rus and antibodies to hepatitis D virus were all negative. The serum HBV DNA concentration was 1,320,000 copies/ml (Amplicor, HBV Monitor[™], Roche Diagnostic Systems Inc., Branchburg, NJ, USA). Acute hepatitis B was diagnosed.

His condition deteriorated and on the seventh hospital day he developed signs of hepatic encephalopathy and aggravation of his laboratory findings that were as follows: ALT, 6840 IU/L/ AST, 3880 IU/L; total bilirubin, 26.4 mg/dl; prothrombin time, 23.3 seconds; INR 2.07; ammonia, 105 μ g/dl/ and albumin, 3.2 g/dl. Mild ascites was shown in abdominal ultrasonography.

At this juncture, because of the progressive clinical and biochemical deterioration and the threat of hepatic failure, we started lamivudine at a dosage of 100 mg daily after informed consent had been obtained. His condition improved rapidly and after one week of treatment, the hepatic encephalopathy was resolved and the prothrombin time and the aminotransferase values decreased. Treatment with lamivudine was continued and four months later the patient had normal liver-function tests and HBV infection was serologically resolved with evidence of seroconversion from HBsAg to antibody to hepatitis B surface antigen (anti-HBs). Lamivudine was discontinued.

Our reason for lamivudine administration in this patient with severe acute hepatitis B and viraemia was to eliminate viral replication in the effort to modify the clinical course of the disease. This report illustrates that the drug can be used safely in acute liver failure. The resolution of acute hepatitis might be induced by lamivudine treatment but the spontaneous resolution of the disease could not be excluded. However, the rapid improvement in the condition of our patient after administration of the drug suggested that lamivudine treatment should be considered in selected cases of severe icteric acute hepatitis B. Further investigation with randomized prospective trials for the optimal use of the drug in this group of patients is needed.

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