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Alcohol Induced Hepatic Degeneration of HCV-Tg Mouse

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Introduction

Hepatitis C virus (HCV) has become a major public health issue and is prevalent in most countries. HCV infection starts frequently without clinical symptoms, and progresses in the majority of patients (70 to 85%) to persistent viremia and chronic hepatitis including cirrhosis and hepatocellularcarcinoma (HCC) [1]. Alcohol is one of the independent cofactors accelerating the development of HCC in chronic hepatitis C patients. This is of great interest because a synergy between excessive alcohol intake and HCV infection has been documented in the development of HCC in chronic hepatitis C patients [2]. The aim of this study is to investigate liver changes in ethanol feeding HCV-transgenic (Tg) mouse and to establish an animal model system.

Materials and Methods

10 HCV-Tg male mice were divided into 4 groups : Non-Tg (n=2); NTG, Tg 116-60 (n=3); TG1, Tg K27 (n=3); TG2 and Tg 99 (n=2); TG3. For 13 weeks, all mice were given 10% ethanol mixed 5% sucrose added to the drinking water until simultaneously sacrificing. For observing liver pathological changes, we performed histo-pathological and immunohisto- chemical method.

Results

In histopathological observations, NTG shown moderate centrilobular necrosis, while hepatic cord destruction and moderate centrilobular necrosis were evident in the TG1 with numerous homogeneous cytoplasm of hepatocyte and mild hepatocyte dissociation. Severe centrilobular necrosis and moderate hepatocyte dissociation was demonstrated in the TG3 with piecemeal necrosis and increasing lymphocyte. All groups, collagen fibers were detected as normal. In immunohistochemical observations, expressions of TGFβ1 and p-Smad2/3 were obviously detected in dissociated and nectotic hepatocytes of TG3.

Discussion

As shown in result, TGFβ1, p21 and p-Smad2/3 expression in hepatocyte is a useful marker in coexistence with alcohol and HCV [3]. In addition, it is considered that this transgenic mouse system would be a useful animal model for the study of pathogenesis in related to alcohol intake and HCV infection, concurrently [4].

References

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