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Ligase chain reaction as a modality for the detection of point mutation in the precore region of HBV related HCC cases from Northern India

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Background & Aim: Mutant Hepatitis B with precore stop codon has been reported to be associated with severe liver damage in HBeAg negative patients with hepatocellular carcinoma. Clinically, the biological importance of pre-core G1896A mutation is not well established. The purpose of the present study was to determine hepatitis B virus genotypes and also to elucidate the association of G1896A mutation of precore gene and the severity of liver damage in HBV related HCC cases.

Methods: Detection of HBV DNA sequences was carried out by polymerase chain reaction (PCR) using primers derived from the precore region of HBV genome. Ligase Chain Reaction (LCR) assay was performed to screen the presence or absence of G1896A mutation. Direct nucleotide sequencing was done to confirm the results of LCR. A total of 116 HBV related cases who attended the medical Out Patients Department and wards of Lok Nayak Hospital, New Delhi, India were screened over the period of 3 years. Patients having super-infection with HDV/HCV/HIV and past history of interferon therapy were excluded.

Results: Sequence analysis of viral DNA established that the G1896A mutation was observed in 32 cases in HCC cases. Phylogenetic analysis revealed 60% isolates belonged to genotype A, while 20% belonged to genotype D and 20% belonged to genotype E.

Conclusion: The present data suggests that precore G1896A mutations is responsible for 27.2% of the patients of Asian Indian origin suffering from HBV related HCC cases and these cases are more symptomatic and aggressive in patients with the mutant form of the virus as compared with the wild form.

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IL30: A biological cytokine provides hepatic protection in Chronic inflammation mediated liver fibrosis

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Chronic hepatic diseases such as cirrhosis, hepatocellular carcinoma and virus mediated immunopathogenic infections are affecting billions of people worldwide. These diseases commonly initiate with fibrosis. Owing to the various side effects of anti-fibrotic therapy and the difficulty of diagnosing asymptomatic patients, suitable medication remains a major concern. To overcome this drawback, the use of cytokine-based sustained therapy might be a suitable alternative with minimal side effects. Here, we studied the therapeutic efficacy and potential mechanisms of IL30 as anti-fibrosis therapy in murine liver fibrosis models. Carbon tetrachloride (CCl4) or 3,5-diethoxycarbonyl-1,4-dihydrocollidine (DDC) 0.1% (wt/wt) Purnima 5015 Chow was fed for 3 weeks to induce liver fibrosis. Either control vector (pCtr) or pIL30 was injected hydro-dynamically once per week. A significant decrease in collagen deposition and reduced expression of α-smooth muscle Actin (αSMA) protein indicated that IL30–based gene therapy dramatically reduced bridging fibrosis that was induced by CCl4 or DDC. Immuno phenotyping and knockout studies showed that IL30 recruits NKT cells to the liver to remove activated hepatic stellate cells (HSCs) significantly and ameliorate liver fibrosis. Both flow cytometric and antibody mediated neutralization studies showed liver NKT cells up-regulates NKG2D ligand and it binds with the NKG2D ligand Rae1 positive activated HSCs to ameliorate liver fibrosis. Furthermore, adoptive transfer of liver NKT cells in T cells deficient mice showed reduction of fibrosis upon IL30 administration. Taken together, our results show that highly target specific liver NKT cells selectively remove activated HSCs via an NKG2D-Rae1 interaction to ameliorate liver fibrosis after IL30 treatment.

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