

ABSTRACTS

Abstracts of the 27th Annual Conference of APASL,
March 14–18, 2018, New Delhi, India

© Asian Pacific Association for the Study of the Liver 2018

Plenary Clinical

16 March 2018

PO-C-01

Post-treatment wisteria floribunda agglutinin-positive Mac-2-binding protein combined with platelet predict hepatocellular carcinoma development in chronic hepatitis C patients

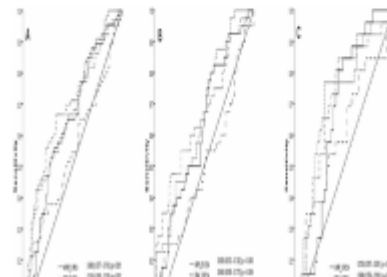
Ming-Lan Yeh¹, Chung-Feng Huang¹, Joe-Fu Huang¹, Chia-Yen Dai¹, Ming-Lang Yu¹, Wan-Long Chung¹

¹Kaohsiung Medical University Hospital, Taiwan

Background: Wisteria floribunda agglutinin-positive Mac-2-binding protein (WFA+M2BP) is a novel marker for liver fibrosis assessment. We aimed to predict the hepatocellular carcinoma (HCC) occurrence after antiviral therapy in Taiwanese patients with chronic hepatitis C (CHC) using WFA+M2BP.

Method: Seventy patients with HCC and another 140 age, gender,

Conclusion: Post-treatment WFA+M2BP, especially combined with platelet, predict HCC development in Taiwanese CHC patients after antiviral therapy.



caused by different virus genotypes. The aim of this study was to test the proposed hypothesis by the study of the SNP genes IFNL4 (rs368234815), IFNL3 (rs12979860 and rs8099917), CD209 (rs4804803), TLR3 rs3775291 and rs13126816 in cohorts of Mongolian patients with hepatitis C virus and in the ethnically similar Buryat group, and also in patients with Hepatitis C caused by different virus genotypes.

Method: A total of 400 patients with chronic HCV were examined, including 200 from the Republic of Buryatia and 200 from Mongolia. The compared groups of patients completely matched in clinical-laboratory and sex-age indices.

Result: There were no associations of polymorphic variants of the genes CD209, IFNL3, and ethnicity of patients, as well as genotypes of the virus in the Buryat population. Obviously, the internalization of different genotypes of the virus into the cell is universal, and, at least, does not depend on the polymorphism of the CD209 gene. In contrast, as a result of the work performed, two SNPs in the candidate genes TLR3 (rs3775291) and IFNL4 (rs368234815) were detected, polymorphic variants of which occur with different frequency in patients with 1 and not 1 (2/3) genotypes of the virus. Carriers of G-allele rs3775291 TLR3 are 3.1 times more resistant to infection with 2/3 virus genotypes ($p < 0.0001$), and carriers of AG-allele rs368234815 IFNL4 - 2.0 times ($p < 0.02$). Consequently, the higher the proportion of human carriers of these alleles and their haplotypes in a population, the higher the tolerance for the spread of 2/3 genotypes of the virus in it. Under these conditions, the first genotype of the virus will receive genetically determined selective advantages, displacing the 2nd and the 3rd from circulation.

Conclusion: Further studies at the level of practically healthy people in Mongolia and Buryatia, as well as the inclusion of other polymorphisms in the analysis will help establish the role of congenital immunity genes in the selective selection of individual genotypes of the virus.

HCV-4

Expansion of intrahepatic lymphocytes expressing PD-1 and bcl-2 in chronic hepatitis C: correlation with disease progression

Eman Abdelhameza¹, Nermine Elhassan², Ahmed Elrefaay², Mervat Sultan¹, Ayaf Abdallah², Maha Elshahawy²

¹Assistant Professor, Egypt; ²National Liver Institute, Menoufia University, Egypt

Background: Programmed Death-1 (PD-1) plays an important role in balancing antiviral immunity. Recently, PD-1 has been shown to induce immune exhaustion of T cells in chronic hepatitis C (CHC). Immune exhaustion and apoptosis are responsible for viral persistence. Little information is available regarding the correlation between PD-1 and anti-hepatocellular carcinoma (HCC) risk.

significant correlation with either steatosis or Ki67. There was a strong significant negative correlation between PD-1 and bcl-2 ($r = -0.723$, $P = < 0.000$).

Conclusion: Intrahepatic PD-1 are conversely interrelated to bcl-2 with a pivotal role in disease severity and progression.

HCV-5

Investigation of the relationship between gene polymorphisms of TNF- α (-1031) and chronic hepatitis C infection

Ozlem Kandemir¹, Nurcan Aras², Gulay Borekeci³, Ozlem Kandemir⁴, Aysegül Cetinkaya⁵, Irem Bekalp Yilmaz⁶, Guhan Oreckci Temel⁶

¹Mersin University, Turkey; ²Mersin University Faculty of Medicine, Department of Clinical Microbiology and Infections, Turkey; ³Mersin University Faculty of Medicine, Department of Medical Biology, Mersin, Turkey; ⁴Turkey; ⁵Mersin University School of Health, Mersin, Turkey; ⁶Mersin University, Faculty of Medicine, Department of Biostatistics, Turkey

Background: In this study we were aimed to determine the relationship between TNF- α (-1031 T/C) gene polymorphism and chronic HCV infections

Method: A total of 99 patients with chronic hepatitis C and 99 healthy subjects were included in the study. Approximately 2 ml of blood from the subjects were taken into tubes containing EDTA, and genomic DNA was isolated using DNA isolation kit (Roche, Switzerland). After the isolation of genomic DNA from blood samples of the patient and control groups, TNF- α (-1031 T/C, rs1799964) polymorphism was investigated by using the Real-Time Polymerase Chain Reaction (RT-PCR) from the DNAs. The data obtained were analyzed using the SPSS package program.

Result: Among chronic hepatitis C group, TNF- α (-1031) TT, TC, CC genotypes were detected in 6 (6,1%), 27 (27,3%) and 66 (66,7%) of the patients, respectively, while these numbers were 5 (5%), 34 (34,3%) and 60 (60,6%) in control group, respectively. When the distribution of allele frequencies of TNF- α (-1031) TC polymorphism was evaluated in the patients and control groups, frequencies of T and C alleles were found to be 39 (39,7%) and 159 (80,3%) in the patient cases and 44 (22,2%) and 154 (77,8%) in the controls. However, there was no statistically significant difference between two groups regarding genotypes and alleles distributions for TNF-alpha ($p > 0,05$).

Conclusion: Our results showed that TNF-alpha (-1031 T/C) polymorphism was not effective in chronic HCV infection. Further studies on this subject can contribute to the elucidation of the molecular mechanisms of chronic hepatitis C.