

regulated by wild and mutant HBx, demonstrated a lower level in the LO2/HBx-d382 cell. Further characterization of miR-338-3p revealed that it negatively regulated cellular proliferation. Cell cycle analysis showed that miR-338-3p induced cell cycle arrest at the G1/S phase. A dual-luciferase reporter assay demonstrated that the 3'UTR of CyclinD1 were directly bound to miR-338-3p and western blotting analysis further indicated that miR-338-3p down-regulated the expression of CyclinD1.

Conclusion: This study demonstrates that HBx can influence cellular miRNA expression. The deregulation of the expression of miR-338-3p by HBx may represent a potential novel pathway which HBx acts to deregulate cell proliferation leading to hepatocarcinogenesis.

PP-106 Relationship between HBsAg, HBeAg expression and serum HBV DNA level in 140 patients with chronic hepatitis B

Y. Yan^{1*}, L. Mai¹, G.L. Lin¹, J. Liu¹, J.Y. Zhu¹. ¹The Third Affiliated Hospital of Sun Yat-sen University, China

Objective: The aim of this study was to investigate the relationship between HBsAg, HBeAg expression and serum HBV DNA level.

Methods: The expression of HBsAg, HBeAg in the livers of 140 patients with chronic hepatitis B was detected by immunohistochemistry. And the level of serum hepatitis B virus DNA (HBV DNA) was tested. Statistical significance was assessed using One-Way analysis of variance (ANOVA).

Results: Serum HBV DNA level in 13 patients with HBsAg (–±), 108 patients with (+–+) and 19 patients with (+++–) was 5.313±1.874 copies log₁₀/ml, 6.010±2.016 copies log₁₀/ml and 5.664±1.548 copies log₁₀/ml respectively (P=0.408). Serum HBV DNA level in 42 patients with HBsAg (–±), 79 patients with (+–+) and 19 patients with (+++–) was 5.886±1.997 copies log₁₀/ml, 5.968±2.020 copies log₁₀/ml and 5.634±1.551 copies log₁₀/ml respectively (P=0.800).

Conclusions: The expression of HBsAg, HBeAg in the liver does not correlate with serum HBV DNA level.

PP-107 Cause analysis of chronic HBV infected patients without antiviral therapy in the Pearl River Delta region

G.L. Lin^{1*}, X.J. Zhang¹, Y. Yan¹, Y.K. Wu¹, X.Y. Li¹, L.Q. Yang¹, Y.T. Chong¹. ¹The third affiliated hospital of Sun Yat-sen University, China

Objectives: To analyze the causes of the patients with chronic Hepatitis B without taking antiviral therapy and the strategies of dealing with it.

Method: We make long-termed observation on the patients with chronic Hepatitis B Virus (HBV) infection, who were voluntarily to be followed up in our clinic department, and analyze statistically the objective and subjective causes of patient without taking antiviral therapy.

Results: In total eligible 951 cases, 424 cases didn't receive the antiviral therapy. 105 out of 424 cases had the indications of the antiviral therapy (105/424, 24.8%), the other 319 cases had no indications of the therapy (319/424, 75.2%). The ratio of female (124/202, 61.4%) who didn't get the antiviral therapy was significant higher than that of male (300/749, 40.0%). 49 out of 105 cases who had the indications of the antiviral therapy worried about the unhealthful effect on their fertility by the antiviral drugs and put off antiviral therapy (49/105, 46.7%); 31 out of 105 cases could not pay for the antiviral therapy (31/105, 29.5%); 19 out of 105 cases queried the safety of the antiviral drugs and uncertainty of course of the treatment

(19/105, 18.1%). 6 out of 105 cases were because of poor compliance (6/105, 5.7%).

Conclusions: No antiviral indications was the main cause of the untreated group. The causes of that patients with indications didn't receive antiviral therapy were that worrying about their fertility, fees of the treatment hard to bear, querying the safety of the antiviral drugs and uncertainty of course of the treatment, and poor compliance. Formal long-termed follow up by the clinicians, good communications between clinicians and patients and health education might improve the effects of anti-HBV treatment.

PP-108 Investigation on serum HBV viral loads and the changes of liver pathological features in 158 patients with chronic hepatitis B

Y. Yan^{1*}, L. Mai¹, J. Liu¹, J.Y. Zhu¹, Y. Zhang¹. ¹The Third Affiliated Hospital of Sun Yat-sen University, China

Objective: To investigate the relationship between serum HBV DNA loads and liver pathological changes in the patients with chronic hepatitis B.

Methods: The relationship among HBV DNA loads, live histological inflammation grades and fibrosis stages of 158 cases was analyzed.

Results: The serum HBV DNA loads in HBeAg-positive group with inflammation grades G₀₋₁ (6 patients), G₂ (74 patients) and G₃₋₄ (25 patients) were 5.580±1.098 copies log₁₀/ml, 6.520±2.004 copies log₁₀/ml and 6.950±1.467 copies log₁₀/ml respectively. There was no significant difference in patients of three inflammation grades (P=0.250). The serum HBV DNA loads in HBeAg-positive group with liver tissues fibrosis stages of S₀₋₁ (23 patients), S₂ (56 patients), S₃₋₄ (26 patients) were 6.599±1.832 copies log₁₀/ml, 6.559±2.012 copies log₁₀/ml, 6.562±1.601 copies log₁₀/ml respectively, the difference was not significant (P=0.996). The serum HBV DNA loads in HBeAg-negative group with inflammation grades G₀₋₁ (8 patients), G₂ (17 patients) and G₃₋₄ (28 patients) were 2.132±1.875 copies log₁₀/ml, 4.745±2.250 copies log₁₀/ml and 5.581±2.305 copies log₁₀/ml respectively. The serum HBV DNA level in patients with G₂ and G₃₋₄ inflammation grades was significant higher than in patients with G₀₋₁ inflammation grades (P=0.001). The serum HBV DNA loads in HBeAg-negative group with liver tissues fibrosis stages of S₀₋₁ (10 patients), S₂ (45 patients), S₃₋₄ (18 patients) were 2.689±3.225 copies log₁₀/ml, 5.127±1.833 copies log₁₀/ml, 5.375±2.410 copies log₁₀/ml respectively. The serum HBV DNA level in patients with fibrosis stages of S₂ and S₃₋₄ was significant higher than in patients with fibrosis stages of S₀₋₁ (P=0.005).

Conclusions: The serum HBV DNA level does not correlate with the inflammation grades and fibrosis stages of liver tissues in HBeAg-positive patients. The serum HBV DNA loads display a positive correlation with the inflammation grades and fibrosis stages of liver tissues in HBeAg-negative patients.

PP-109 Association of IL-6 gene polymorphism and its levels in HBV related hepatocellular carcinoma progression in India

R. Saxena¹, Y.K. Chawla², J. Kaur^{1*}. ¹Department of Biochemistry, Postgraduate Institute of Medical Education and Research, Chandigarh, 160 012, India, ²Department of hepatology, Postgraduate Institute of Medical Education and Research, Chandigarh, 160 012, India

Objectives: Hepatitis B Virus (HBV) infection is a primary risk factor for hepatocellular carcinoma (HCC), the fifth most frequent cancer, worldwide. The present study was undertaken to analyze the association of IL-6 (–572) and