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**International Meeting on** 

## Clinical Case Reports April 18-20, 2016 Dubai, UAE

## HCBP6 maintaining of cholesterol homeostasis by down-regulating SREBP2/HMGCR

Jun Cheng, Zhang Y, Gao L L, Liu S A, Han M, Liang P, Lu H P and Feng S H Capital Medical University, China

Dysregulation of cholesterol homeostasis is associated with metabolic diseases including fatty liver, atherosclerosis and type-2 diabetes. Growing evidences have suggested that the nucleocapsid of HCV (core) involves in lipid droplet accumulation, changes lipogenic gene expression and/or its activity. However, as a HCV core protein interacting protein, the function of HCBP6 protein is remains unclear. Here, we found that overexpression of HCBP6 in hepatoma cells suppressed the expression of sterol response element binding protein2 (SREBP2) that is a crucial factor for maintaining cholesterol homeostasis and HMGCR expression. Consistently, we found that silence of HCBP6 expression in hepatoma cells induced SREBP2 expression and eventually increased intracellular cholesterol biosynthesis. Moreover, HCBP6 expression level was reduced in liver tissue of mice fed with a high-fat diet and this decrease correlated with an increase of total cholesterol level. Thus, HCBP6 controls cholesterol homeostasis through regulating SREBP2 and HMGCR expressions and their activities. We also found that miR-185 regulated HCBP6 expression through a complex cholesterol-responsive feedback loop. In conclusion, we provide evidence that a mir-185 mediated HCBP6 expression maintaining of cholesterol homeostasis by down-regulating SREBP2/HMGCR.

chengj0817@sina.cn

## Neonatal transient hepatitis-B surface antigenemia detected less than 24 hours post immunization

**Laila Obaid** Mafraq Hospital, UAE

**Introduction:** Perinatal hepatitis-B virus (HBV) infection occurs during delivery from an infected mother; it is associated with few or no symptoms but has a high risk of chronic subclinical disease in later childhood or adulthood. Up to 90% of infants infected perinatally will develop chronic infection. The risk of transmission is 70-90% from women seropositive for hepatitis-B surface antigen (HBsAg) and hepatitis-B e-Antigen at the time of delivery. Pregnant women are usually screened for HBV infection early at pregnancy or at delivery if the HBV status is unknown by measuring HBsAg. Neonates born to mothers with HBsAg-positive or of unknown HBV status should receive hepatitis immunoglobulin (HBIG) and HBV vaccine. The combination of HBV vaccine and HBIG is 85-95% effective in reducing HBV infection from vertical transmission when given within 12 hours of birth.

**Case Report:** A full term male infant was born to multi-gravida mother with poor antenatal care and high risk behavior. The mother and newborn were tested for HIV, Syphilis and HBV before discharge. The maternal HBsAg screen was negative but the infant screen was positive at 19 hours of age. The baby received vaccine and immunoglobulin. Repeated maternal HBsAg confirmed negative for HBsAg. On further reviewing of the mother and infant medical records it was found that the infant received the vaccine at 9 hours of age. Transient HBsAg antigenemia can occur following receipt of HBV vaccine with HBsAg being detected as early as 24 hours and most often at 2-6 days and up to 2 to 3 weeks following administration of the vaccine. We recommend that, if a newborn screen for HBsAg is required, the test should be performed prior to administration of HBV vaccine to avoid unnecessary parental concern and investigation.

laobaid@seha.ae

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