Conclusion: The data suggest that the HLA-G 14-pb Ins/Del polymorphism may not play a relevant role in the susceptibility to hepatitis B infection. However, the association between this polymorphism and infection severity suggests an evidence of HLA-G implication in hepatitis B infection progression.

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Session: Virology and Viral Infections (Non-HIV) I

Date: Friday, April 4, 2014 Time: 12:45-14:15

Room: Ballroom

Hepatitis B core IgM antibodies (anti HBcIgM), a serological marker for eradicating transfussion associated hepatitis B virus (TAHBV) infection in low income countries



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Background: The safety of blood products is one of the major problems concerned with the transfusion medicine. A definite hazard of transmission of hepatitis B to recipients of blood screen for Hepatitis B surface Antigen (HBsAg) when such donor is at the 'window phase' exist. The possibility of achieving zero risk of Transfusion Associated Hepatitis B Virus (TAHBV) infection is highly dependent on DNA testing of all collected units of blood before transfusion. This is not feasible in low income countries including Nigeria due to cost. At present, HBsAg detection is the only diagnostic screening test for HBV infection identification in the blood transfusion centers of Nigeria. Hepatitis B core IgM antibody (anti-HBc IgM) is gaining prominence as a useful alternative in reducing TAHBV infection. The present study was aimed at evaluating adoption of anti-HBc IgM an additional screening assay for blood donation since anti-HBc detection is not mandatory in Nigeria.

Methods & Materials: Two hundred and seventy-three (Male = 246, F=27; Age range:18-50 Mean:24.9years) apparently healthy blood donors in Ibadan were enrolled in the study between October and December 2012; their demographic data was obtained. The frequency of HBsAg and its corresponding antibody (HBsAb), as well as Hepatitis B envelope antigen and its corresponding antibody (HBeAg and Ab) were assessed using standard enzyme link Immunosorbent assay EELISA) technique. Anti-HBcIgM was also assessed in donors negative or positive for HBsAg using same method.

Results: The prevalence of anti-HBc IgM was 4.4%. Twelve (11 Males and 1 female) had anti-HBc IgM as the only marker of HBV infection. Prevalence of HBsAg, HBsAb, HBeAg and HBeAb were 14(5.1%), 95(34.8%), 2(14.3%) and 3(21.4%) respectively.

Conclusion: The findings of the current study recommends that all blood units should be tested for anti -HBc IgM to understand the infectivity status of the blood donors in the window period and to discard blood if zero risk of Transfusion Associated Hepatitis B Virus (TAHBV) infection will be achieved in Nigeria.

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Hepatocellular carcinoma in a long-term sustained virological responder following pegylated-interferon plus ribavirin combination therapy for chronic hepatitis C



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Background: It is well known that long-term complications of hepatitis C virus (HCV) infection including hepatocellular carcinoma (HCC) and cirrhosis are eliminated or decrease in sustained virological responders after treatment. We report a case from Turkey who developed HCC 4 years after sustained and complete response to pegylated interferon plus ribavirin combination theraphy.

Methods & Materials: A 60-year-old man with HCV genotype 1b was treated with pegylated interferon alpha-2b in combination with ribavirin for a total of 52 weeks. Initially, his liver histology was consistent with a mild activity and 1\6 fibrosis stage due to chronic hepatitis C. After 28 weeks of treatment, aminotransferase levels were in the normal range and HCV RNA (polymerase chain reaction) was undetectable in serum. Sustained and complete response were obtained with normalization of aminotransferases and disappearance of HCV RNA in serum continuously. HCV-RNA was also not detected in the liver tissue after treatment, but histopathological examination was the same as before. He followed up for HCC based on biochemical and ultrasound evaluation every 6 months.

Results: HCC was detected 48 months after cessation of therapy with the elevation of serum aminotransferases and alphafetoprotein for the first time, then splenomegaly and acid were revealed by ultrasonography. HCC was diagnosed by computed tomography and angiography, and then treated through transarterial embolization but patient died of liver failure within 2 months.

Conclusion: Successful treatment in our case didn't prevent development of HCC even in non-cirrhotic liver. Our case indicates the importance of not underestimating risk of HCC development even many years after sustained and complete response to HCV treatment. Long-term follow up are always mandatory and should include more carefully and closely surveillance for HCC.

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