

Familial Screening A Must In HBV

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Abstract

Introduction: Chronic hepatitis B Virus (HBV) infection is an important cause of cirrhosis of liver for which effective treatment is available but still significant number of patients require liver transplantation when it reaches end stage liver disease. There are various routes of transmission for HBV like parenteral, vertical, nosocomial and through close contact among family members of HBV patient. Familial prevalence in HBV is an important aspect and merits strict screening of all family members which can prove to be lifesaving for many.

Case Report: - We report a thirty-six-year-old male, not a known case of any chronic illness presented with bilateral pedal oedema for last one-month, abdominal distension and breathlessness on exertion for last two weeks. On evaluation he was diagnosed to be suffering from chronic hepatitis B related chronic liver disease (CLD) with decompensation in form of ascites with hepatocellular carcinoma (H.C.C) with portal vein thrombosis. On family evaluation he told that his on elder brother had died of HBV related H.C.C few years back and mother also died of HBV related CLD in past. His five brother and one sister were found to be HBV Positive and only one brother was HBV negative. on regular antiviral treatment for last five years.

Conclusion: The family members of every Hepatitis B patient should be screened for HBV infection. The positive members should be evaluated in detail for stage of disease and if needed treatment should be started on priority. In case of family history of HBV related hepatocellular carcinoma (H.C.C), other HBV positive patients with low viral load should also be treated with antiviral therapy. The members who are found to be HbsAg negative should be vaccinated with complete course of three doses of HBV vaccine.

Introduction

HBV transmission has many routes; most predominant one includes percutaneous or per mucosal exposure to HBV-containing body fluids. The most important source of infection is blood [1]. HBV transmission occurs through different kind of human contact, including vertical transmission from mother to newborn, sexual contact, close household contact, needle sharing, and occupational exposure (horizontal transmission) [2,3]. HBV is efficiently transmitted by sexual contact [2,3]. HBV has a higher prevalence within families due to household transmission. Studies show that family members of individuals with HBV have a significantly increased risk of infection, up to four times greater than in the general population. This increased risk is largely attributed to close contact and various modes of transmission within the household. The transmission within families occurs through blood, sexual contact, and from mother to child (vertical transmission). Within families, horizontal transmission (e.g., between spouses or children) is also common. The HBV status of the index case significantly impacts transmission within the family. Mothers with HBV have a higher risk of transmitting to their children. Larger families may have a higher risk due to increased potential for close contact. Early identification of infected individuals and their family members through screening programs, followed by vaccination, is crucial for preventing further transmission. In India, Hepatitis B is considered a public

health problem, with a prevalence rate of 2-4% in the general population. However, the prevalence among household contacts of HBV-positive individuals is significantly higher. Intra-familial transmission is a major factor in maintaining HBV endemicity in India. Studies in India have reported familial transmission rates ranging from 11.9% to 19.2%, much higher than the general population. It is recommended to screen all family members of HBV-positive individuals and provide vaccination to susceptible individuals, especially children, to control transmission. Care providers should take precautions like not sharing items like razorblades, toothbrushes, nail clippers and properly covering open cuts or wounds.

Case Report

We report a thirty-six-year-old male, not a known case of any chronic illness presented with bilateral pedal oedema for last one-month, abdominal distension and breathlessness on exertion for last two weeks. On evaluation, complete hemogram showed microcytic hypochromic anaemia and thrombocytopenia (80,000) with increased AST and ALT levels (86 & 74 I.U. respectively), mild hyperbilirubinemia (2.3 mg%) with low total serum protein & albumin levels (5 gm and 2.6 gm respectively). The renal function test, thyroid profile, blood sugar, serum electrolytes, ECG, Chest X-ray, Urine complete examination anti HCV & anti-HIV antibody were negative but HbsAg was positive with HBV DNA Quantitative of 2.1×10^5 I.U/ml. His ultrasonogram showed altered echotexture of liver, splenomegaly, ascites with multiple hypo and hyper nodules in both lobes of liver? regenerative. The triple phase computed tomography scan showed features of CLD, ascites with multiple small heterogenous hypodense lesions seen scattered in both lobes of liver with 22 x 19 mm lesion in segment seven of liver. These lesions showed arterial enhancement and wash out in portal and delayed venous phase. Portal vein was dilated with diameter of 16 mm with heterogenous hypodense content which enhanced on arterial phase suggestive of malignant thrombus which extended into left and right branch. The right branch of portal vein measured 15.1mm and left branch measured 19 mm. Multiple collaterals were seen in peri gastric, peripancreatic, splenic hilum, mesentery and anterior abdominal wall with recanalization of umbilical vein. Multiple subcentimetric lymph nodes were seen in peri gastric region, porta, right and para-aortic region, root of mesentery, measuring 8mm in SAD. The intrahepatic and suprahepatic part was also dilated with thrombus with extension into right atrium. Spleen was enlarged to 12.3 cm. The right lobe of liver measured 14.7 cm with irregular nodular outline. There was evidence of volume redistribution with relative hypertrophy of caudate lobe and left lobe of liver. There was evidence of bilateral pleural effusion. The alpha feto protein levels were significantly raised to 2050. The surgical oncologist opinion was taken who ruled out any surgical intervention or liver transplantation and advised for conservative and supportive management only. On family evaluation he told that his on elder brother had died of HBV related H.C.C few years back and mother also died of HBV related CLD in past. His five brother and one sister were found to be HBV positive and only one brother was HBV negative. All the family members who were found to be HbsAg positive were evaluated in detail including HBV quantitative viral load and were started on antiviral treatment, even at low viral load, as per scientific rationale. The patient and family members on their request were referred to higher center for further expert opinion but in view of ascites, Trans arterial chemoembolization (TACE) was ruled out. He was started on antiviral treatment.



FIGURE 1- Triple phase CECT Scan abdomen showing multiple lesions scattered in both lobes of liver



FIGURE 2- Triple phase CECT Scan abdomen showing enhancement of lesions in arterial phase with splenomegaly

Discussion

HBV infections are dependent on the country of birth, as in HBV-endemic countries, most HBV transmission occurs perinatally or during childhood. In countries with intermediate and high HBV endemicity, HBV transmission mainly occurs during infancy and early childhood through vertical or horizontal transmission. In one estimate, approximately 90% of infections occur before 10 years of age, leaving many adults immune from infection later in life [4]. HBV infection is a major cause of hepatocellular carcinoma (HCC), the most common type of primary liver cancer. Chronic HBV infection can lead to liver inflammation, fibrosis, and cirrhosis, which are all significant risk factors for developing HCC. HBV can directly contribute to HCC development through DNA integration into the host genome and the expression of viral proteins that disrupt cellular processes. Cirrhosis is a major risk factor for HCC, but even without cirrhosis, chronic HBV infection significantly increases the risk of developing liver cancer. The HBV protein HBx plays a role in oncogenesis by interfering with cell growth regulation and DNA damage repair. Age, male gender, higher HBV viral load and other factors like obesity and diabetes can also influence HCC risk. Due to the persistent risk of HCC even after viral suppression, regular HCC screening is crucial for early detection, especially in high-risk individuals. In 2023, the CDC recommended screening for HBV infection in all adults aged 18 years or older at least once in their lifetime. A triple panel test including HBsAg, anti-HBs (anti-surface antibody), and anti-HBc (anti-core antibody) should be employed regardless of risk factor disclosure [5]. Genotype C has been shown to be associated with an increased risk of HCC as an independent risk factor [6]. Antiviral therapy can reduce the risk of HCC by lowering HBV viral load and inflammation, but the risk isn't eliminated completely. Long-term HBV treatment in HCC has been shown to improve overall survival. A meta-analysis by Yuan et al. demonstrated that overall survival was higher in those who received antiviral treatment with nucleoside analogues (NA), by 11% at 1 year, 28% at 3 years, and 40% at 5 years [7] compared to those who were untreated. HBV vaccination is highly effective in preventing HBV infection and reducing the risk of HCC. In our department due to implementation of Jeevan Rekha

Project & National Viral Hepatitis Control Program (NVHCP) through which there is provision of total free treatment including viral load and other routine tests, drugs, endoscopy, fibroscan, indoor admission in wards etc. Moreover, as a well-planned policy, hepatitis B patients are given free consultation and treatment on daily basis without any waiting period. Our model treatment centre is high flow centre and there is lot of thrust on screening especially of the spouses and family members of HBV patients. This team effort has led to good social bonding with the patients who developed full faith in the treating team. This familial bonding led to overcome the hurdle of illiteracy and rural background in majority of patients who were treated for HBV. Thus, we were able to convince majority of patients for getting tested their family members for HBV infection.

Conclusion

The family members of every Hepatitis B patient should be screened for HBV infection. The positive members should be evaluated in detail for stage of disease and if needed treatment should be started on priority. The members who are found to be HbsAg negative should be vaccinated with complete course of three doses of HBV vaccine.

Conflict of Interest

The authors declare that there was no conflict of interest and no funding was taken from any source to conduct this research.

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