RESEARCH COMMUNICATION

Hepatitis Virus B is Not a Risk Factor in Hepatoblastoma Patients

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Abstract

The prevalence of primary liver cancer varies throughout the world. Hepatoblastoma is the most common pediatric liver malignancy, comprising approximately 1% of all pediatric cancers. The exact etiology of hepatoblastoma remains undetermined. Concerning hepatitis B infection, whether there are links with hepatoblastoma is poorly documented. Here, we summarize knowledge on the prevalence of hepatitis B seropositives among the patients with hepatoblastoma. According to the literature review, six reports were recruited for metanalysis, with a total of 60 cases. The overall prevalence of seropositive cancer was 3.3 % (2/560). Further analysis revealed no correlation between prevalence rate and nationality of the studied population (P > 0.05). Therefore, hepatitis B infection does not appear to be a contributing factor for hepatoblastoma. Further studies are needed to clarify which are the risk factors for the hepatoblastoma.

Key Words: hepatitis B - hepatoblastoma - seropositive

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Introduction

The prevalence of primary liver cancer varies throughout the world. Hepatoblastoma (HB) is the most common pediatric liver malignancy, comprising approximately 1% of all pediatric cancers (Schnater et al., 2003). The disparate clinical staging systems and histologic classifications that were developed during the last decades, nevertheless, reflect the remaining difficulties and uncertainties in characterizing HB (Schnater et al., 2003). The exact etiology of this cancer remains undetermined (Schnater et al., 2003). However, it is noted for the association with Beckwith-Weidemann syndrome, familial adenomatosis polyps, and low birth weight (Herzog et al., 2000).

Cases of combined hepatitis B infection and hepatoblastoma have sporadically been reported (Inagaki et al., 2001; Pongpipat et al., 1983). All human hepatitis B viruses characterized so far express three envelope proteins, pre-S1, pre-S2, and HBs, which are believed to function as binding proteins for the cellular receptor, as targets for immune-mediated virus elimination, and in virion morphogenesis and secretion (Pongpipat et al., 1983). In addition, Fernholz et al (Fernholz et al., 1993) reported that cloned infectious HBV DNA genomes having the pre-S region substituted by the variant pre-S region were replication competent in cultured hepatoblastoma cells (Pongpipat et al., 1983). Here, we summarize knowledge on the prevalence of hepatitis B seropositives among hepatoblastoma patients using a metanalysis.

Materials and Methods

A literature review to find the previous reports about prevalence of hepatitis B seropositive among the patients with hepatoblastoma was performed. The author used the electronic search engine PubMed (www.pubmed.com) in searching for the literatures. The available reports were collected and extracted for the data about the seroprevalence of viral hepatitis B. Those primary data were used for further metanalysis study.

Concerning the metanalysis study, the overall prevalence rate was calculated. Also, the association between seroprevalence rate and nationality of the populations was assessed using the Chi square test. SPSS 11.0 for Windows was used for statistical analysis in this study.

Results

According to the literature review, 6 reports (Pongpipat et al., 1983; Ichikawa et al., 1991; Chan et al., 2002; O' Brien et al., 1989; De Potter et al., 1987; Ohaki et al., 1983) were recruited for further metanalysis (Table 1), including a total of, 60 cases were studied. The overall prevalence of seropositive cancer wa 3.3 % (2/60). Further analysis

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Table 1. Previous Reports on the Seroprevalence of Viral Hepatitis B Among the Patients with Hepatoblastoma

Items	Authors					
	Ichikawa et al, 1991	Chan et al, 2002	O'Brien et al, 1989	De Potter et al, 1987	Pongpipat et al, 1983	Ohaki et al, 1983
Nationality	Japan	Hong Kong	USA	Belgium	Thailand	Japan
Number of cases	15	11	6	9	9	10
Positives	0	0	0	0	0	0
Seroositive rate (%)	0	0	0	0	2	0

revealed no correlation between the prevalence rate and nationality of the studied population ($P > 0.05$).

Discussion

Hepatoblastoma accounts for approximately 5% of malignancies in childhood (Wittekind, 2000). Most HBs fall into epithelial or mixed epithelial and mesenchymal categories (Wittekind, 2000). Jia et al propose that hepatitis B virus X (HBx) might interfere with the molecular pathway also through binding to and altering the activities of helicases necessary, thereby, increase the mutation rate induced by chemical carcinogens during human liver carcinogenesis. They proposed the association between hepatitis B particle and primary liver cancer (Jia et al., 1999). Pongpipat et al proposed that hepatitis B infection might be an important risk for development of HB (Pongpipat et al., 1983). However, Buckley et al said that no evidence was found to support the primary study hypotheses relating to hepatitis infection, maternal estrogen exposure, alcohol consumption, smoking, or potential sources of nitrosamines (Buckley et al., 1989). Here, the author retrospective analyzed the prevalence of hepatitis B seropositive among the patients with HB.

According to this metanalysis, the summative prevalence is about 3.3 %. Of interest, this prevalence rate is similar to general population. In addition, seroprevalence rate does not correlate to nationality of the studied population. Therefore, unlike other primary hepatic cancer, hepatitis B infection might not be a contributing factor for HB. The report of the pattern of primary liver cancer in Taiwan after hepatitis B vaccination coverage program (Lee et al., 2003) also supports this finding. In that report, the significant decreasing of incidence of hepatocellular carcinoma was noted but there was no significant change in the incidence of HB(Lee et al., 2003). Further studies are needed to clarify which is the exact risk factor for the HB.

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