

Research Article

Detection of Antibody Levels in Vaccinated Oncological Patients against Viral Hepatitis B (Post-Exposure Prophylaxis)

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Abbreviations

HBV: Hepatitis B virus; Anti-HBsAg: Antibodies to Hepatitis B surface Antigens; HBsAg: Hepatitis B surface Antigens

Introduction

Hepatitis B Virus (HBV) is a major global health issue due to its high prevalence and related mortality. It is estimated that 2 billion people, approximately 30% of the global population, show serological evidence of current or past HBV infection, and 240 million people worldwide are chronically infected with HBV [1]. Antibodies to hepatitis B surface antigens (anti-HBs) are important markers of immunity against Hepatitis B. The detection of this antibody titer is essential in evaluating the vaccine response. Antibody titer of >10 mIU/mL after one to two months of completing primary vaccination schedule is considered seroprotective [2]. Over time and age the antibody titers are known to decrease. It has also been observed that individuals with some detectable antibody levels are likely to respond better to booster dose. The immune response to vaccination is a complex process involving both the innate and the adaptive immune system and requiring multiple steps and cell interactions: antigen uptake and processing by dendritic cells, migration to lymphoid tissue, antigen presentation to T cells, T cell-B cell interaction, and finally plasma cell maturation, proliferation and antibody production. The perfect function of all these involved cells is a prerequisite to achieve a protective antibody level after vaccination [3]. Vaccination is most effective in preventing HBV infection and complications. The complete vaccine series induce protective antibody levels in more than 95% of infants, children, and young adults. Protection has been estimated to last at least 20 years and is possibly lifelong [4]. HBV vaccination is the mainstay of HBV prevention. However, vaccine failure occurs in 5–10% of recipients: individuals who cannot produce a protective level of antibodies against the hepatitis B surface antigen (anti-HBs) after a standard vaccine course [5]. Although the mechanisms that determine the different vaccine responses are not fully understood, the complex interplay between Hepatitis B surface Antigen (HBsAg) and host factors influences the different immune

Abstract

Anti-HBs response of oncological patients vaccinated after exposure during an outbreak in a general hospital was evaluated. Only nine patients could be traced back. In all nine samples only two were found with protective anti-HBs level. Our experience demonstrates the importance of vaccination prior to potential exposure.

Keywords: Anti-HBs; Hepatitis B; Hepatitis C; Hospitalization; Post-exposure prophylaxis; Onco-patients

response among individuals [6].

The aim of this study was to investigate if anti-HBs antibodies were produced in vaccinated oncological patients. They were vaccinated after exposure during the outbreak with 4 doses of vaccine contained 40 µg/ml of hepatitis B surface antigen.

Material and Methods

We analyzed samples of sera obtained from 9 exposed patients for anti-HBs antibodies. They were patients exposed to viral hepatitis B and C during an outbreak; they were vaccinated immediately after exposure six years before sampling. Only these nine patients can be traced back. Fasting plasma serum from the all nine patients were analyzed in National reference center for viral hepatitis. Detection of anti-HBs antibody was tested in samples of patient's plasma using ELISA tests (BIO RAD).

Results

In this study, we analyzed samples of patients' sera. Based on regular medical examination after exposure we can conclude that hepatitis B virus infected any of these nine patients. After six-year-period, in all nine patient's samples we found only two with protective anti-HBs level. Rest of samples were anti-HBs negative.

Discussion

The vaccination is the most effective measure in preventing HBV infection and complications. The complete vaccine series induce protective antibody levels in more than 95% of infants, children and young adults. Protection has been estimated to last at least 20 years and is possibly lifelong [7,4]. Sahana and co-authors in their work investigated decrease of anti-HBs antibodies level among medical students and health care workers. The proportion of subjects who were unprotected after 5 to 10 years were 20% only [8-10]. We can generally assume this group healthy and not immunocompromised. Comparing to our group of immunocompromised patients their response to vaccination was very poor. In our group six years after vaccination only 2 of 9 patients (22.2%) developed protective anti-

HBs antibody level. 7 patients (77.8%) were unprotected despite of vaccination in specific strengthened scheme (40µg for each dose). Although a rapid and effective, strategy for HBV immunization of patients with hematological malignancies is highly desirable, to date there is not an approved schedule for these patients. Conducting HBV vaccination trials in adult patients with hematological malignancies is troublesome [9].

Heterogeneity of both the underlying hematological conditions and chemotherapy regimens, maintenance therapies, relapse of the disease, and salvage regimens including high-dose chemotherapy with stem cell support make the situation more complex. Therefore, recruiting a sufficient number of patients for randomized trials and multivariate analysis requires the active collaboration of centers, especially in developing countries. Because of these difficulties, the number of HBV vaccination trials in patients with hematological malignancies is very limited and mostly confined to pediatric patients with acute leukemia [8].

The data supporting HBV vaccination almost completely come from general vaccination strategies and no evidence-based recommendations for the dose, frequency, and timing of HBV vaccination in adult hematological patients are available [9]. Özkurt et al mentioned in their paper that there is a risk of HBV transmission just after the diagnosis or during the chemotherapy of patients, is high due to frequent transfusions and interventions. Immediate vaccination is highly desirable; however, disease and chemotherapy may compromise antibody response. If the vaccination is postponed until after the chemotherapy, disease and chemotherapy-related immunosuppression are lessened and probability of response increases, but active protection from HBV during the high-risk period is missed.

Conclusion

Our work shows that in this small group of immunocompromised patients vaccinated against viral hepatitis B after exposure level of anti-HBs was tested. This parameter was detected in samples of 2 exposed patients only (22.2%), 7 patients (77.8%) remain unprotected. Comparing to health population the response of these

patients to vaccination is very poor. Our experience demonstrates the importance of vaccination prior to potential exposure, similarly as it is usual in hemodialysis patients.

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References

1. Trepo C, Chan HL, Lok A. Hepatitis B virus infection. *Lancet*. 2014; 384: 2053-2063.
2. Center for disease control and prevention. Atlanta (US): CDC guidance for evaluating health-care personnel for Hepatitis B virus protection and for administering postexposure management. Center for disease control and prevention, U.S. department of health and human services. Report no 10, 2013; 62; 1-24.
3. Barraclough KA, Playford EG. Hepatitis B virus infection in hemodialysis populations: progress toward prevention. *Kidney Int*. 2010; 77: 177-180.
4. WHO. Hepatitis B. Fact Sheet No. 204. Geneva. WHO, 2014.
5. Zuckerman JN. Nonresponse to hepatitis B vaccines and the kinetics of anti-HBs production. *J Med Virol*. 1996; 50: 283-288.
6. Sakai A, Noguchi E, Fukushima T, Tagawa M, Iwabuchi A, Kita M, et al. Identification of amino acids in antigen-binding site of class II HLA proteins independently associated with hepatitis B vaccine response. *Vaccine*. 2017; 35: 703-710.
7. Kim YJ, Li P, Hong JM, Ryu KH, Nam E, Chang MS. A Single Center Analysis of the Positivity of Hepatitis B Antibody after Neonatal Vaccination Program in Korea. *J Korean Med Sci*. 2017; 32: 810-816.
8. Yetgin S, Tavil B, Aytac S, Kuskonmaz B, Kanra G. Unexpected protection from infection by two booster hepatitis B virus vaccination in children with acute lymphoblastic leukemia. *Leuk Res*. 2007; 31: 493-496.
9. Özkurt ZN, Suyarı E, Haznedar R, Yağcı M. A randomized study comparing the efficacy of three hepatitis B vaccine induction regimens in adult patients with hematological malignancies. *Turk J Hematol*. 2016; 33: 231-235.
10. Sahana HV, Sarala N, Prasad SR. Decrease in anti-HBs antibodies over time in medical students and healthcare workers after hepatitis B vaccination. *Biomed Res Int*. 2017; 1327492.