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Case Report

A Rare Clinical Course of Spontaneous Loss of Surface Antigen of Chronic Hepatitis B by Vertical Transmission: A Case Report

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1. Abstract

The spontaneous loss of surface antigen (SLS) with or without antibodies is considered to be the manifestation of functional cure for chronic hepatitis B virus (CHB) infection. The probability of SLS is very low and its mechanism is unknown. There are many studies on the natural history of SLS, but few studies on its occurrence process. A 39-year-old male suffered from hepatitis B virus (HBV) transmitted vertically from mother to child. The first CHB activity occurred on Nov 12, 2015 and spontaneous HBV DNA clearance occurred. During the three-year follow-up, the HBV remained inactive and stable. On Mar 17, 2019, hepatitis B virology rebound and hepatitis activity occurred. No antiviral treatment was given and SLS appeared.

2. Keywords: Surface antigen; Functional cure; Hepatitis B virus

3. Introduction

The spontaneous loss of surface antigen (SLS) with or without antibodies is considered to be the manifestation of functional cure for chronic hepatitis B virus (CHB) infection. This state of infection is generally considered to be the most ideal state in the natural history of HBV because of the cessation of viral replication, the cessation of hepatic inflammation and fibrosis, and the minimal risk of cirrhosis and hepatocellular carcinoma. The probability of SLS is very low and its mechanism is unknown. There are many studies on the natural history of SLS [1, 2], but few studies on its occurrence process. We observed a case of HBV by vertical transmission and the whole process of SLS, and found its unique natural history of HBV. No similar studies have been reported.

4. Case Report

A 39-year-old male patient was complained of fatigue, urinary yellow, skin yellow staining for one week and hospitalized to our department on Nov 12, 2015. The liver function was abnormal, HBV surface antigen (HBsAg) was positive, E antigen (HBeAg) was negative, E antibody was positive, and HBV DNA was positive see (Table 1). He was diagnosed as CHB. The patient was HBsAg positive when he was young considered infection by vertical transmission during to his mother was CHB in the family. Every 1-2 years, the liver function and serum markers of HBV were examined, and liver function remained normal. Considered HBV carriers were not given antiviral treatment. During hospitalization, vitamins and other supportive treatment were given, but no antiviral treatment was given because of significantly decreasing of HBsAg and HBV DNA levels. By Dec 22, 2015, HBV DNA is undetectable. Later, the routine examinations such as liver function, hepatitis B serum markers, HBV DNA and abdominal ultrasonography were performed every 6-12 months. On Mar 17, 2019, symptoms such as fatigue, decreased appetite, yellow urine and skin were occurrened and re-hospitalized. HBsAg and HBV DNA levels were re-activated. As well as previous results. During hospitalization, we continued to give supplementary treatment, and found that virological replication index decreased spontaneously without antiviral treatment. About 2 months later, both HBsAg and HBV DNA spontaneously turned negative. The patient has no narcotic drugs use and

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no alcohol use history and no history of immunosuppressive drugs use such as corticosteroids.

HBV genotype belongs to B type, Routine blood tests showed normal, antinuclear antibody were negative, viral hepatitis (A, C, D and E), and Epstein - Barr virus, cytomegalovirus and human immunodeficiency virus infections were ruled out. Abdominal ultrasonography revealed no nonalcoholic fatty liver disease (NAFLD), no cirrhosis, no splenomegaly, and no ascites.

Table 1: Changes of main indexes of liver function and viral indexes.

Time	TBIL (1.71-21μmol/L)	DBIL (0-7.32 μmol/L)	ALT (4-40 U/L)	AST (4-40 U/L)	INR (0.7-1.5)	HBsAg (<0.05IU/L)	HBV DNA (<20IU/ml)
11/12/2015	55.4	75.3	553	359	1.57	5427	2.45×10^7
11/18/2015	44.2	37.3	226	176	1.36	4327	4.12×10^4
11/29/2015	32.3	16.4	53	44	1.04	3675	5.15×10^2
12/12/2015	16.3	6.3	32	34	1.02	2254	< 20
12/22/2015	11.3	5.5	36	26	1.11	136	< 20
3/16/2019	47.4	26.3	975	665	1.63	3256	1.25×10^6
3/25/2019	34.4	16.6	226	321	1.24	2267	6.36×10^4
4/12/2019	22.4	11.5	65	47	1.12	537	1.54×10^3
5/10/2019	15.2	6.2	33	37	1.12	25	< 20
5/27/2019	16.2	7.2	27	21	1.07	<0.05	< 20

TBIL：total bilirubin , ALT:alanine aminotransferase, AST:aspartate aminotransferase, INR: international normalized ratio

5. Discussion

The importance of HBsAg levels is due to the consensus that HBsAglevelsis related to the covalently closed circular (ccc) DNA inside the hepatocytes [3].

Quantification of HBsAg can reflect the natural history of HBV. High HBsAg and HBV DNA serum levels are characteristics of immune tolerance stage. The in which HBsAg and HBV DNA serum levels are lower than those in HBeAg-postive phases, including two phases: HBeAg-negative CHB and inactive carriers phases. HBsAg serum levels are significantly lower in Inactive Carriers (IC) than CHB patients [4].

This patient has been in the period of HBeAg-negative phase. Spontaneous HBV DNA clearance appeared in the first attack. It can be said that he entered the period of inactive carrier for about 3 years. In the fourth year, he entered the period of HBeAg-negative CHB without obvious inducement accompanied by the rebound of HBsAg and HBV DNA, and obvious liver function damage. The second spontaneous elimination of epistaxis occurred only 2 months later. A large meta-analysis showed that the SLS was very low, only 1.17% per year [5].

We report a case of E antigen negative of HBV with specific SLS. The mechanism is unknown.

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