Short Communication

iMedPub Journals
http://www.imedpub.com

Journal of Clinical and Molecular Pathology

2021
Vol. 5 No. 2: 31

Immune System and Hepatitis B Virus

Cosmin Constantin Oprea*

Independent Research, Bucharest, Romania

Corresponding author:
Cosmin Constantin Oprea, Independent Research, Bucharest, Romania

cosminoprea1988@gmail.com

Citation: Oprea CC (2021) Immune System and Hepatitis B Virus. J Clin Mol Pathol Vol.5 No.2: 31.

Abstract

Purpose of Review: Hepatitis B virus infection and the immune response in babies and adults.

Findings: The presence of MDSC in new-borns makes Hepatitis B chronic over years with both antigens present in the blood, HBs and HBe. The immune system is not able to recognize the infection in the first years of life, which represents the stage of immunotolerance. The expansion of MDSC allows the immune system to maintain the infection with Hepatitis B, although the patient reaches adult age, because MDSC is responsible for the disfunction of the immune system and not the virus itself.

Summary: MDSC is responsible for chronic Hepatitis B infection. The expansion of MDSC is the format in which hepatitis B manage to escape the immune system's response.

MDSC cells have the ability to interact with these signals generated by common progenitor lymphoid cells and in this way the immune system cannot exercise its function.SSSS

Keywords: Hepatitis B; MDSC; Immune system; Antigen HBe

Received: April 16, 2021; **Accepted:** April 30, 2021; **Published:** May 09, 2021

Introduction

Hepatitis B is the most common serious liver infection in the world. It is caused by the hepatitis B virus that attacks and injures the liver.

Two billion people (or 1 in 3) have been infected and about 290 million people are living with a chronic hepatitis B infection.

Each year up to 1 million people die from hepatitis B despite the fact that it is preventable and treatable.

About the Hepatitis B Virus

The hepatitis B virus is a small DNA virus that belongs to the Hepadnaviridae family. Related viruses in this family are also found in woodchucks, ground squirrels, tree squirrels, Peking ducks, and herons.

Structure of the Hepatitis B Virus

The human hepatitis B virus belongs to the family of hepadnaviridae. The hepadnaviridae are subdivided into mammalian and avian hepadnaviruses. The mammalian hepadnaviruses include human hepatitis B virus (HBV), woodchuck hepatitis virus (WHV) and the ground squirrel hepatitis B virus (GSHV). The duck hepatitis B virus (DHBV) and the heron hepatitis B virus (HHBV) belong to the avian hepadnaviruses [1].

Life Cycle of the Hepatitis B Virus

HBV has a small (ca. 3.2 kbp), partially double-stranded (DS), relaxed circular (RC) DNA genome which is replicated via reverse transcription from an RNA intermediate, the pregenomic RNA (pgRNA) [2]. The RC DNA genome is packaged into an icosahedral capsid composed of the HBV core protein (HBc), which in turn is enclosed in an envelope layer composed of host cell derived lipid bilayer studded with three viral envelope or surface proteins [3].

Immune Response in Chronic Hepatitis B

A large number of clinical studies have shown that chronic HBV persistent infection causes the dysfunction of innate and adaptive immune response involving monocytes/macrophages, dendritic cells, natural killer (NK) cells, T cells.

Myeloid-derived suppressor cells (MDSCs) represent a heterogeneous population of immature myeloid cells with broadly distinct phenotypes that fail to terminally differentiate into granulocytes, macrophages, or DCs. They exhibit a remarkable capacity to inhibit immune responses mediated by T, B, and NK cells.

In addition, accumulated liver MDSCs due to HBV infection

suppress CD8+ T cell function and promote systemic CD8+ T cell exhaustion [4] characterized by high expression levels of inhibitory receptors such as CTLA-4, PD-1, and TIM-3 [5].

Conclusion

Unlike other viral infections such as HIV or Hepatitis C, in 95% of cases young people and adults that have acute Hepatitis B, the HBs Antigen is seroconverted.

In babies the situation is opposite, the earlier they got the infection, the greater the chances of chronicity. If a baby becomes infected through vertical transmission or in the first months of life, it will become chronic with the form of HBe positive antigen, respectively the form that is responsible for a high viral load and a high contagion, because we are talking about the inability of the immune system to present antigen and respond, practically an ideal environment for the development of Hepatitis B.

The fact that Hepatitis B manifests itself acutely (Ag HBe +) in the first period of life of new-borns, will induce tolerance towards both antigens in the blood, respectively HBs and HBe.

MDSC are rarely seen in healthy adults but are present in large numbers in new-borns. The MDSC frequency decreases rapidly in the first months of new-borns life.

If a child of 2 years or older becomes infected with Hepatitis B, the immune system seems to be able to eliminate the HBe antigen, by tolerating a single antigen, namely the HBs antigen. So, we are talking about a situation in which the immune system detects and eliminates the HBe antigen, the immune system recognizes the HBe antigen and can trigger specific T cells to seroconvert this antigen.

The above theory might explain the HBe antigen tolerance in new-borns.

The question now is whether we can recreate what happens in adults with acute Hepatitis B for persons with chronic Hepatitis B. With chronic Hepatitis persons, the HBs antigen is tolerated by the immune system because MDSC suppress CD8 cells. Inhibition of MDSC might be an option for exhausted T-Cells, because MDSC are responsible for maintaining tolerance by inhibiting receptors of T-Cells.

References

1. Locarnini S (2004) Molecular virology of hepatitis B virus. Semin Liver Dis 24:3–10.
2. Summers J, Mason WS (1982) Replication of the genome of a hepatitis B—like virus by reverse transcription of an RNA intermediate. Cell 29(2):403–15.
3. Hu J, Liu K (2017) Complete and Incomplete Hepatitis B Virus Particles: Formation, Function, and Application. Viruses 9(3).
4. Kong X, Sun R, Chen Y, Wei H, Tian Z, et al. (2014) γδT cells drive myeloid-derived suppressor cell-mediated CD8+ T cell exhaustion in hepatitis B virus-induced immunotolerance. J Immunol 193:1645–53.
5. Park JJ, Wong DK, Wahed AS, Lee WM, Feld JJ, et al. (2016) Hepatitis B virus--specific and global T-cell dysfunction in chronic hepatitis B. Gastroenterology 150: 684-95.