Annals of Clinical Hepatology

0

Presentation of Hepatitis B and C Infections

Bogdan Ionescu*

University of Bucharest, Faculty of Biology, Microbiology and Immunology Department, Bucharest, Romania

Introduction

Liver afflictions are one of the most common health issues encountered in the word today. The most prevalent liver afflictions that affect people from all corners of the globe are hepatitis B and C. Approximately 370 million people are suffering from hepatitis B and 130 million are infected with hepatitis C. In the case of people suffering from hepatitis B around 15 million have also contracted hepatitis D. The clinical manifestations of hepatitis B and C virus infections can vary from the asymptomatic to acute [1].

Hepatitis B

In the case of infections with the hepatitis B virus the people infected can be carriers with normal liver histology or they can be suffering from chronic hepatitis which can be persistent or active chronic [2]. In the case of hepatitis C infections they can also manifest as chronic hepatitis with the persistent and active types but it can also develop to cirrhosis and carcinoma of the liver [3].

The HB virus has a spherical shape and is part of the Hepadnaviridae family. The surface of the virions presents three different proteins which form the surface antigen called HBsAg which is the one recognized by the immune system leading to the synthesis of antibodies. All the three proteins lHBs (large), mHBs (middle) and sHBs (small) contain a protein named sHBs which is a very important part of the antigen [4]. In the case of the hepatitis B virus the outer surface of the viral particle containing the nucleo capsid is formed out of the protein shell (HBs) named the Dane particle. The viral particle is a complete virion which consists of the core represented by the HBc antigen the endogenous antigen (HBe), DNA, the DNA polymerase as well as other proteins [1].

The HBe antigen can be found in the sera of patients under two different forms. The antigen can have a small spherical shape found only in the serum of carriers and also a long, filamentous form. In both cases the particles are incomplete presenting no genome. The particles do contain envelope proteins as well as lipids like: phospholipids, cholesterol and triglycerides. By comparison the spherical shaped HBe antigen contains only the middle and small proteins while the long molecules present all three types of proteins [4].

OPEN ACCESS

*Correspondence:

Bogdan Ionescu, University of Bucharest, Faculty of Biology, Microbiology and Immunology Department, Bucharest, Romania, E-mail: bogdanionescu87@yahoo.com Received Date: 14 May 2017 Accepted Date: 06 Nov 2017 Published Date: 16 Nov 2017

Citation:

Ionescu B. Presentation of Hepatitis B and C Infections. Ann Clin Hepatol. 2017; 1(1): 1004.

Copyright © 2017 Bogdan Ionescu. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. From the clinical pathology point of view of the hepatitis B infections the majority of young people and adults affected suffer from asymptomatic infections, very rarely followed by a development of acute hepatitis [5]. In the case of acute hepatitis the symptoms are not specific being represented by: vomiting, fatigue and apathy. The severity of the infection can be established by determining the titer of the virus, a higher titer being suggestive of a more severe affliction [6].

In the early stages of the infection the immune system is yet aware of the presence of the virus and the infection will progress from just a few cells to over 75% of the liver cell population. Following the early stages of the infection the immune system detects the viral DNA and the hepatitis B antigen. After 8 weeks from the onset of symptoms the titer of the antigen reaches the maximum level [5].

Liver necrosis is characterized by the presence of multifocal parenchymal cells and periportal inflammation. Necrosis affects cells at different intensities and dead liver cells are transported in the perisinusoidal space [7].

The acute liver infections develop in up to 6 months and are often associated with a high virus titer. A low percentage of acute infections lead to severe liver failure which will develop into hepatic coma [2]. Survivors fully recover and become immune to HBs and HBc. The severe cases are associated with the hepatitis D co-infection determining a rapid deposition of hepatitis B antigens in the kidneys, causing kidney failure. A low titer of anti-HBs antibodies will lead to the infection not being removed from the body. So in the case of infections that persist for more than 6 months they often evolve towards chronicity [8].

Hepatitis C

The hepatitis C virus is similar to other hepatitis viruses with a severe pathology and a variable incubation period between 2-26 weeks with an average of 7. The severity of hepatitis C infections is due to the persistence of the infection and the high likelihood of re infection [9].

In a quarter of cases the immune system is able to eliminate the virus from the body with leaving any long lasting effects but the organism is not protected from a new infection. In the rest of the cases the infections have a chronic evolution. The chronic evolution can lead to a persistent infection, to active chronic hepatitis, in the severe cases to cirrhosis and carcinoma [8]. In the case of the persistent hepatitis patients do not present clinical symptoms and are detected at while conducting usual blood tests. The persistent infection can become severe by association with alcoholism and other hepatitis infections [10]. The active hepatitis presents an increased viral titer. This type of hepatitis more often progresses to more severe forms. Chronic hepatitis is associated with chronic liver inflammation, necrosis, and cirrhosis. The damage to the liver is caused partially by the immune response [11].

The hepatitis C infection presents an indirect pathological process leading to the chronic stages. The persistent chronic infections are caused by the formation and deposition of immune complexes at the kidney level. The liver has features that induce tolerance to antigens [12]. The evolution to the persistent chronic stage is due to the immune response not being strong enough or the duration of it being too short. The persistent chronic type is the most common associated with the HCV infection [13-14].

Conclusions

Hepatitis B and C remain the most prevalent liver afflictions in the world today. The association of these infections with HIV has led to end-stage liver disease and cirrhosis becoming the leading cause of death in HIV positive patients. More effort needs to go into finding ways of reducing the number of people affected as well as developing better treatment methods.

References

- Harrison T, Geoffrey M, Arie J. Hepatitis viruses. In: Principles and practice of clinical virology. 5th edn. Chichester: John Wiley & Sons, Ltd; 2004.
- 2. Valsamakis A. Molecular testing in the diagnosis and management of chronic hepatitis B. Clin Microbiol Rev. 2007;20(3):426-39.
- Seeger C, William S. Hepatitis B virus biology. Microbiol Mol Biol Rev. 2000;61(4):51-68.
- Carter B, Venetia A. Virology: Principles and applications. Chichester: John Wiley & Sons; 2007.
- 5. Harrison T. Hepadnaviruses: General features. In: Desk Encyclopedia of Human and Medical Virology. 3rd edn; 2008.
- 6. Sploski R, Waren J. Interleukin 21. Basic biolgy and implications for cancer and autoimmunity. Ann Rev Immun. 2008;26:57-69.
- Brenchley J, Douek D. Microbial translocation across the GI tract. Ann Rev Immun. 2012;30:149-73.
- 8. Crispe I. The liver as a lymphoid organ. Ann Rev Immun. 2009;27:147-63.
- 9. Major E, Barbara R, Stephen M. Hepatitis C viruses. In: Fields virology; 2001.
- 10. Lindenbach D, Charles M. Flaviviridae: The viruses and their replication. In: Fields Virology; 2001.
- 11. Chambers T. Flaviviruses: General features. In: Desk Encyclopedia of Human Medical Virology. 3rd edn; 2008.
- 12. Batenschlager R, Buhler S. Hepatitis C virus. In: Desk Encyclopedia of Human Medical Virology. 3rd edn; 2008.
- Nisar ZN. Clinical significance of hepatitis C virus genotypes. Clin Microbiol Rev. 2000;13(2):223-35.
- Dustin B, Charles M. Flying under the radar: The immunobiology of hepatitis C. Annu Rev Immunol. 2007;25:71-99.