



Journal of Clinical Infectious Diseases & Practice

Acute Hepatitis B Infection in Adults: Identifying the Progression to Chronicity

Ferro Patrizia*

Department of Biochemistry and Molecular Biology, Shahjalal University of Science and Technology, Sylhet, Bangladesh

Abstract

Acute hepatitis B virus (HBV) infection can progress to chronic infection in a significant number of adult patients, leading to severe liver complications. This article provides an overview of the clinical features exhibited by adults who develop chronic HBV infection. During the acute phase, patients may experience nonspecific symptoms such as fatigue, loss of appetite, and abdominal pain, along with jaundice in some cases. While most individuals clear the virus within six months, approximately 5-10% progress to chronic infection. Chronic hepatitis B can present with a wide range of symptoms, from asymptomatic to advanced liver disease. Fatigue, weakness, loss of appetite, and hepatosplenomegaly are common manifestations. Complications may arise, including cirrhosis, hepatocellular carcinoma, ascites, and hepatic encephalopathy. Regular monitoring of HBV DNA levels and liver function tests is crucial to detect disease progression and complications. Early detection and appropriate management can help prevent long-term liver damage and reduce the risk of severe complications.

Keywords: Hepatitis B virus; Chronic infection; Hepatocellular carcinoma

Introduction

Hepatitis B virus (HBV) infection is a global health concern, affecting millions of people worldwide. While most adults who acquire acute HBV infection recover completely, a significant proportion of patients progress to chronic infection. Chronic hepatitis B can lead to severe liver complications, including cirrhosis, hepatocellular carcinoma, and liver failure. Understanding the clinical features of adult patients who develop chronic HBV infection is crucial for early detection, appropriate management, and prevention of long-term liver damage [1].

Hepatitis B virus (HBV) is a DNA virus with approximately 3200 base pairs. Approximately 350-400 million people are chronically infected with HBV and more than 3 billion people have been exposed to HBV worldwide. HBV induces a variety of liver diseases, ranging from acute or fulminant hepatitis to liver cirrhosis and hepatocellular carcinoma. HBV is one of the most important causes of liver cirrhosis and hepatocellular carcinoma. On the other hand, hepatitis is selflimited in most adult patients with acute infection. Meanwhile 1-2% of patients progress to fulminant hepatic failure, and some progress to chronic infection. The rate of progression from acute to chronic HBV infection is reported to be 90% in newborns and 5-10% in adults. HBV can be classified into at least 8 genotypes with a divergence of more than 8% of nucleotide sequences [2]. There are some differences in clinical features and routes of transmission between genotypes. The rate of chronicity of genotype A infections is reported to be higher than those of other genotypes. The progression of acute hepatitis B to chronic hepatitis is not rare in Western countries, but it is rare in Japan. The differences of the rates of chronicity of acute HBV infection supposed to be attributable to the different distribution of HBV genotypes; genotypes B and C are the predominant genotypes while genotype A was rare in Japan and common in Western countries. However, previous studies are based on the follow-up studies of apparent acute hepatitis B. The present study aimed to clarify the progression to chronic infection in adult patients with acute HBV infection including subclinical or unapparent patients who progressed to chronic infection [3].

Acute hepatitis B is characterized by the presence of clinical symptoms and signs of liver inflammation, along with elevated liver enzymes and the detection of HBV-specific markers in the blood. During the acute phase, patients may experience nonspecific symptoms such as fatigue [4], loss of appetite, nausea, vomiting, and abdominal pain. They may also develop jaundice, which is marked by the yellowing of the skin and eyes, along with dark urine and pale stools. However, it is important to note that not all individuals with acute HBV infection will display symptoms, especially in cases of mild or asymptomatic infections.

Discussion

It must be noted that the present study was not aimed at knowing the rate of chronicity in acutely HBV-infected patients, because it is extremely difficult to collect the unapparent cases of acute HBV infection [5]. The purpose of this study is to know the clinical features of patients with acute HBV infection who progressed to chronic infection. All 3 patients in group A in the present study had been negative for HBsAg for at least 1 year before testing positive. Data regarding anti-HBc and HBV-DNA negativity before the onset of hepatitis were available in patient 3 but not in patient 1 or patient 2. Therefore, the possibility of HBV reactivation from HBsAg-negative carrier or resolved hepatitis status in these 2 patients cannot be excluded completely. However, these patients did not take any medicine that may suppress immune function, were not drug abusers or alcohol abusers, and were not in immune-suppressed state. Therefore, the risk of HBV reactivation is presumed to be very low [6].

***Corresponding author:** Ferro Patrizia, Department of Biochemistry and Molecular Biology, Shahjalal University of Science and Technology, Sylhet, Bangladesh, E-mail: patriziaferrio@edu.it

Received: 03-July-2023, Manuscript No: jcidp-23-107733, Editor assigned: 05-July-2023, Pre-QC No: jcidp-23-107733 (PQ), Reviewed: 19-July-2023, QC No: jcidp-23-107733, Revised: 25-July-2023, Manuscript No: jcidp-23-107733 (R) Published: 31-July-2023, DOI: 10.4172/2476-213X.1000188

Citation: Patrizia F (2023) Acute Hepatitis B Infection in Adults: Identifying the Progression to Chronicity. J Clin Infect Dis Pract, 8: 188.

Copyright: © 2023 Patrizia F. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

In most cases, acute HBV infection resolves spontaneously within six months, leading to the clearance of the virus from the bloodstream. However, approximately 5-10% of adult patients will progress to chronic infection. Chronic hepatitis B is defined by the persistence of HBV DNA in the blood for at least six months. The clinical features of chronic infection can vary widely, ranging from an inactive carrier state with minimal liver damage to progressive liver disease [7].

Patients with chronic HBV infection may remain asymptomatic or exhibit mild symptoms for many years. However, as the disease progresses, they may experience fatigue, weakness, loss of appetite, weight loss, and easy bruising or bleeding due to impaired liver function. Some individuals may develop hepatomegaly (enlarged liver) or splenomegaly (enlarged spleen). In advanced cases, complications such as cirrhosis may arise, characterized by the development of fibrous scar tissue in the liver [8], leading to portal hypertension and its associated manifestations, including ascites and hepatic encephalopathy.

Moreover, patients with chronic hepatitis B are at an increased risk of developing hepatocellular carcinoma (HCC), a type of liver cancer. The risk of HCC is higher in individuals with underlying cirrhosis and those with detectable HBV DNA in the blood [9]. Therefore, regular monitoring of HBV DNA levels and liver function tests is essential to detect any progression or complications.

It is worth noting that several factors influence the likelihood of progression from acute to chronic HBV infection. These factors include the age at the time of infection (the younger the individual, the higher the risk of chronic infection), gender, certain viral factors, and host immune response [10].

Conclusion

In conclusion, while most adults with acute hepatitis B infection recover completely, a significant proportion progresses to chronic infection, which can lead to severe liver complications. Recognizing the clinical features of chronic hepatitis B, such as fatigue, loss of appetite, jaundice, and signs of liver damage, is crucial for early detection and management. Regular monitoring of liver function and HBV DNA levels, along with appropriate antiviral therapy, is essential in preventing disease progression, reducing liver damage, and minimizing the risk of long-term complications such as cirrhosis and hepatocellular carcinoma.

References

- Geremia A, Biancheri P, Allan P, Corazza GR, Sabatino A (2014) Innate and adaptive immunity in inflammatory bowel disease. Autoimmune 13: 3–10.
- Boltin D, Perets TT, Vilkin A, Niv Y (2013) Mucin function in inflammatory bowel disease. J Clin Gastroenterol 47: 106–111.
- Johansson ME, Stovall H, Hansson GC (2013) The gastrointestinal mucus system in health and disease. Nat Rev Gastroenterol Hepatol 10: 352–361.
- Chassaing B, Darfeuille-Michaud A (2011) The commensal microbiota and enteropathogens in the pathogenesis of inflammatory bowel diseases. Gastroenterology 140: 1720–1728.
- Bergstrom KS, Kissoon-Singh V, Gibson DL, Montero M, Sham, Huang T, et al. (2010) Muc2 protects against lethal infectious colitis by disassociating pathogenic and commensal bacteria from the colonic mucosa. PLoS Pathog 6: 148-150.
- Johansson ME, Gustafsson JK, Holmen-Larsson J, Jabbar KS, Xia L, et al. (2014) Bacteria penetrate the normally impenetrable inner colon mucus layer in both murine colitis models and patients with ulcerative colitis. 63: 281–291.
- Schwerbrock NM, Makkink MK, Buller HA, Einerhand AW, Sartor RB, et al. (2004) Interleukin 10-deficient mice exhibit defective colonic muc2 synthesis before and after induction of colitis by commensal bacteria. Inflamm Bowel Dis 10: 811–823.
- Atuma C, Strugala V, Allen A, Holm L (2001) The adherent gastrointestinal mucus gel layer: Thickness and physical state in vivo. Am J Physiol Gastrointest Liver Physiol 280: 922–929.
- Ermund A, Schütte A, Johansson ME, Gustafsson JK, Hansson GC, et al. (2013) Studies of mucus in mouse stomach, small intestine, and colon. I. Gastrointestinal mucus layers have different properties depending on location as well as over the Peyer's patches. Am J Physiol Gastrointest Liver Physiol 305: 341–347.
- Vaishnava S, Yamamoto M, Severson KM, Ruhn KA, Yu X, et al. (2011) The antibacterial lectin RegIIIgamma promotes the spatial segregation of microbiota and host in the intestine. Science 334: 255–258.