

Management of Acute and Chronic Hepatitis B and C Viral Infections

Haradhan Kumar Mohajan¹

¹ Associate Professor, Department of Mathematics, Premier University, Chittagong, Bangladesh

Correspondence: Haradhan Kumar Mohajan, Associate Professor, Department of Mathematics, Premier University, Chittagong, Bangladesh.

doi:10.63593/JIMR.2788-7022.2025.10.001

Abstract

Hepatitis is an inflammation of the liver that can be caused by over alcohol and toxin chemical consumption, some medications, and viruses, such as hepatitis B, C, D, and E. The hepatitis B virus (HBV) and hepatitis C virus (HCV) can develop acute and chronic viral infections that are major causes of liver cirrhosis, liver cancer, liver transplantation, and liver related death worldwide. Both of these two viruses can transmit through the vertical or horizontal route, such as blood or body fluids from an infected person, unprotected sex with an infected person, sharing needles with infected person, and from infected mother to baby during birth. Symptoms of both diseases are similar, and some common symptoms are jaundice, loss of appetite, fever, fatigue, dark urine, joint pain, abdominal pain, diarrhea, nausea, and vomiting, etc. The incubation period is 8-20 weeks for hepatitis B, and 2-6 weeks for hepatitis C. At present there are two vaccines of HBV, but no vaccine available for HCV.

Keywords: hepatitis B, hepatitis C, liver cirrhosis, hepatocellular carcinoma

1. Introduction

Hepatitis B is a viral infection that is caused by hepatitis B virus (HBV). It is a short-term acute illness or a lifelong chronic infection that may be a cause of life-threatening liver cirrhosis, liver failure, liver cancer, and hepatocellular carcinoma (Mohajan, 2024g). Hepatitis C is a blood-borne fatal disease of the liver that is caused by hepatitis C virus (HCV), which can be both an acute (short-term) illness (25-15%) and a chronic (long-term) infection (75-85%) that may gradually damage the liver (Mohajan, 2024h). The complications of HBV and HCV have been the seventh leading cause of death worldwide. Both of them are blood-borne viruses that cause liver inflammation and kill liver cells that result 1.45 million deaths worldwide each year, of which 47% are attributable to the HBV and 48% to the HCV (Sunbul, 2014).

The HBV is a deoxyribonucleic acid (DNA) virus with a nuclear capsule enveloped by an outer lipid layer containing hepatitis B surface antigen HBsAg that is reproduced in the cytoplasm of the hepatocyte and serves as an indicator of the carrier of the virus (Lau & Wright, 1993). On the other hand, the HCV is a single-stranded ribonucleic acid (RNA) Flavivirus encoding for a capsid protein, two envelope proteins, and some nonstructural proteins (Health of Ministry, 2019). The HBV is an entirely vaccine-preventable disease and there is no vaccine for HCV. The HBV is discovered in 1965 by American physician and geneticist Baruch Samuel Blumberg (1925-2011) (Blumberg, 2002). The HCV is discovered in 1989 by three scientists Harvey J. Alter, Michael Houghton, and Charles M. Rice as the major causative agent of “non-A, non-B hepatitis” (Choo et al., 1989).

More than 257 million individuals are chronically infected with HBV, and 71 million with HCV worldwide, and majority of them do not have access to life-saving medications. In the absence of vaccination most exposed neonates and young children will be infected, and will become lifelong carriers (Gow & Mutimer, 2001; Health of Ministry, 2019). Chronic HBV and HCV infections can lead to liver damage, the development of fibrous tissue in the liver (fibrosis), fat deposits in the liver (steatosis), liver scarring (cirrhosis), and liver cancer. In

severe cases, a person may require a liver transplant to avoid death (Stanaway et al., 2016).

2. Literature Review

Literature review is a secondary source and does not report a new or an original experimental work (Gibbs, 2008). It helps the novice researchers to understand the subject, and it serves as an indicator of the subject that has been carried out before (Creswell, 2007). Alessio Aghemo and his coauthors have observed that the chronic infection with the hepatitis B and C virus represents a major health problem worldwide. The infected people are at increased risk of developing cirrhosis, hepatocellular carcinoma (HCC), liver decompensation, and esophageal variceal bleeding. Ultimately some patients need liver transplantation or have to face liver-related miserable death (Aghemo et al., 2012).

Erzsébet Szabó and her coauthors have observed that the HBV and HCV are the cause of a wide spectrum of clinical manifestations, ranging from healthy carrier state to acute and chronic hepatitis, cirrhosis, and hepatocellular carcinoma (HCC). The HBV contains a DNA genome which replicates through an RNA intermediate and requires an active viral reverse transcriptase (RT) polymerase enzyme, while HCV is an RNA virus which has no RT activity and replicates on the cellular membrane by RNA replication (Szabó et al., 2004). Hammad Safdar Ali has perceived that HBV and HCV are responsible for considerable amount of liver disease globally, and both of the viruses have same mode of transmission, the co-infection of these viruses happens but is considered as uncommon (Ali, 2018).

John W. Ward and Alan R. Hinman have found that together HBV and HCV can cause 1.4 million deaths annually. To prevent more than 7 million deaths by 2030, the WHO set goals to eliminate HBV and HCV, defined as a 90% reduction in new infections and a 65% reduction in deaths, and prevent more than 7 million related deaths by 2030 through the elimination of these viruses (Ward & Hinman, 2019). Saleh Mohammed Abdullah has studied that the hepatitis is a serious health concern with a high rate of morbidity and mortality worldwide. They have found that the prevalence of HBV infection among Saudi subjects in Jazan was higher than the prevalence of HCV infection, and both HBV and HCV were higher in men than in women (Abdullah, 2018).

Malathi Sathiyasekaran and Ganesh Ramaswamy have observed that HBV and HCV in most children remain asymptomatic, but have the potential to progress to chronic hepatitis, cirrhosis, end-stage liver disease, and hepatocellular carcinoma. They have advised that prevention is better than cure, and every effort should be made to prevent these diseases as significant transmission occurs during the perinatal period (Sathiyasekaran & Ramaswamy, 2020). Douglas F. Johnson and his coauthors have found that international travelers are at risk of HBV and HCV infections. They have provided the modes of transmission, and the prevention of infection in travelers. The HBV vaccination is safe and efficacious with protective levels of antibodies achieved in greater than 90% of recipients (Johnson et al., 2013).

3. Research Methodology of the Study

Research is a hard-working search, scholarly inquiry, and investigation aimed at the discovery of new facts and findings (Adams et al., 2007). Methodology in any creative research is the organized and meaningful procedural works that follow scientific methods efficiently (Kothari, 2008). Research methodology shows the ways to the researchers for organizing, planning, designing and conducting good research (Legesse, 2014). In this paper, we have depended on the secondary data sources of the HBV and HCV infections (Mohajan, 2024a-f). We have taken help from the published journal articles, printed books of famous authors, conference papers, internet, websites, etc. (Mohajan, 2024i-n). Throughout the study we have tried to maintain the reliability and validity as far as possible (Mohajan, 2017, 2018, 2020).

4. Objective of the Study

Main objective of this article is to discuss the acute and chronic life-threatening HBV and HCV infections, such as liver cirrhosis, liver failure, liver cancer, and hepatocellular carcinoma (HCC). Both of the viruses are blood-borne. The HBV is a deoxyribonucleic acid (DNA) virus and the HCV is a single-stranded ribonucleic acid (RNA) virus. The HBV is an entirely vaccine-preventable disease and there is no vaccine for HCV (Mohajan, 2024g, h). Other minor objectives of the study are as follows:

- 1) to focus on HBV and HCV,
- 2) to highlight on their transmission, and
- 3) to show the treatment of both viruses.

5. Hepatitis B Virus (HBV)

It is estimated that one-third of the global population have been infected with HBV at some point in their lives and of these 257 million people are chronically infected, and one million people die annually from HBV

infection and its associated complications (WHO, 2017).

5.1 HBV Genotypes

The hepatitis B virus (HBV) is an enveloped, circular, hepatotropic, small (3.2kb) and non-cytopathic partially double-stranded DNA virus, 40-42nm in diameter. It belongs to the Hepadnaviridae family (Huang et al., 2013). The virus has four open reading frames (ORFs) in which several genes overlap; as core, surface, X and polymerase. The viral genome is transferred into the nucleus, where a covalently closed circular form of DNA (cccDNA) is formed, which serves as a template for viral transcription (Szabó et al., 2004). The HBV virus is formed with an outer lipoprotein envelop that bears three related envelop glycoprotein (E-proteins) termed the surface antigens (Locarnini, 2004). The biomarkers related to HBV are hepatitis surface antigen (HBsAg), core antigen (HBcAg), envelope antigen (HBeAg), and the corresponding antibodies are anti-HBs, anti-HBc and anti-HBe (IgG, IgM) (Sathiyasekaran & Ramaswamy, 2020).

There are 10 genotypes (GTs) A to J of HBV globally that differ by 8-10% at the nucleotide level across the whole genome (Schaefer, 2005). More than 40 sub-GTs are identified, and there are no sub-GTs of E, G and H GTs. The GT-A has four sub-GTs: A1, A2, and A4, and quasisub-GT A3. It is highly prevalent in Africa, Northern Europe, India, and America. The GT-B has nine sub-GTs: B1, B2, B3, B4, B5, B6 B7, B8, B9, and quasisub-GT QS-B3. The GT-C has 16 sub-GTs: C1-C16. The GTs B and C are common in the Asia-Pacific region (Kramvis, 2014). The GT-D has nine sub-GTs: D1-D9. It is privilege in Iran, Russia, Europe, Alaska, Serbia, Somalia, India, Nigeria, and Indonesia. The GT-F has four sub-GTs: F1-F4. It is found in Central and South America, Alaska, and other parts of the world. The GT-I has two sub-GTs: I1 and I2. It is isolated in Vietnam and Laos (Shi et al., 2012).

5.2 Symptoms and Transmission of HBV

Most HBV infected persons show no symptoms. Some common symptoms of it are loss of appetite, tiredness, fever, headache, nausea and vomiting, abdominal pain, dark urine, diarrhea, clay-colored stool, and jaundice (Farooq et al., 2017). The HBV is highly contagious and relatively easy to transmit from one infected individual to another by exposure. It is transmitted through the perinatal, percutaneous, and sexual exposure of the HBV-infected person's body fluids, such as serum, saliva, semen, and vaginal fluids (Aghemo et al., 2012). Other possible routes of transmission are unprotected sexual contact, blood transfusion, reuse of contaminated needles and syringes and vertical transmission from mother to child during birth (Buddeberg et al., 2008). The HBV virus has an incubation period of 2-6 months and has human as the natural reservoir (Cheesbrough, 2006).

5.3 Vaccination and Treatment of Hepatitis B

There are two vaccines available for HBV immunization that utilizes recombinant DNA technology: Engerix-B and Recombivax. These two types of vaccines are equally effective and safe (WHO, 2017). The HBV antiviral medications are lamivudine, adefovir, tenofovir, telbivudine, interferon alpha-2a, and pegylated interferonalpha-2a. Among this lamivudine is a safe effective antiviral drug for treating chronic HBV infection and interferon alpha is the only drug licensed for the treatment of it (Kim et al., 2009).

6. Hepatitis C Virus (HCV)

Hepatitis C disease is potentially a life-threatening liver infection and a silent killer that has become a major public health challenge that affects 170 million people globally. About 71 million people are infected with HCV, and about 704,000 people die from HCV-related liver diseases (WHO, 2017).

6.1 HCV Genotypes

The HCV is enveloped in a lipid-glycoprotein bilayer 30-80nm single-stranded RNA virus, 9600 nucleotide length, that belongs to Hepacivirus genus in the Flaviviridae viral family, which is less infective but more sinister compared to HBV. The two biomarkers of HCV are anti-HCV antibody and HCV RNA (Sathiyasekaran & Ramaswamy, 2020). The core proteins and the envelope proteins, such as E1 & E2 form the structural proteins, the non-structural proteins, such as P7 viroporin, NS2, NS3, NS4A, NS4B, NS5A, and NS5B are encoded as in viral morphogenesis and assembly (Dufour, 2006).

The HCV has 7 genotypes (GTs) and over 90 subtypes. The GTs 1, 2, and 3 are more common in the northern hemisphere (Smith et al., 2014). The GT1 is the most common in Northern and Western Europe, Asia, North and South America, and Australia. The GT2 is mostly present in West and Central Africa. The GT3 is the most common in South Asia. The GT4 is the most common in Egypt, GT5 is present only in South Africa, and GT6 is endemic in Hong Kong and Southern China, and GT7 infection has been reported that is isolated in Canada from a Central African immigrant (Messina et al., 2015).

6.2 Symptoms and Transmission of HCV

About 70-80% people have no symptoms when they are infected with hepatitis C. Some common symptoms of it

are jaundice, vague abdominal discomfort, loss of appetite, nausea and vomiting, weight loss, fatigue, abdominal pain, fever, itching, etc. (Purcell, 1997). The HCV is parenterally transmitted with blood and blood product exposure or injecting drug use (Mesquita et al., 1997). It can be transmitted with the contact of infected blood especially through the hemophiliacs, dialysis patients, and intravenous drug users (parenterally). Other modes of transmission are sexual, perinatal, idiopathic, tattooing, sharing of items, and pregnant mother to infant occurs in less than 10% of pregnancies (Tremolada et al., 1992).

6.3 Treatment of Hepatitis C

There is no vaccine for hepatitis C, but several vaccines are currently under development. Treatment of it is palliative and supportive (Strickland et al., 2008). The combination of PegIFN-alpha and ribavirin for 24 or 48 weeks is the standard of care for treatment of HCV infection. Recently, the combinations and newer treatments, such as polymerase inhibitors, protease inhibitors, and NS5A inhibitors are recommended for HCV (Berg et al., 2006).

7. Conclusions

From this study we have observed that both HBV and HCV damage liver, and in advanced stage these can develop chronic hepatitis, cirrhosis and hepatocellular carcinoma (HCC). It is matter of concern that hepatitis B and C are responsible for 96% of all hepatitis mortality worldwide. At present one-third of the global population are infected with HBV and more than 170 million people are infected with HCV worldwide. About 257 million people with HBV and 71 million people with HCV are chronically infected. From these infected patients about one million people die annually from HBV, and about 700,000 people die each year worldwide from chronic HCV infection. In this situation global health sector faces a high economic and health burdens due to various hepatic complications. Patients with HBV and HCV dual infection have more severe liver disease, and are at an increased risk for progression to hepatocellular carcinoma (HCC). Both of these diseases can be prevented or mitigated by early detection, treatment, and lifestyle changes.

References

- Abdullah, S. M., (2018). Prevalence of Hepatitis B and C Virus Infection and Their Co-relation with Hematological and Hepatic Parameters in Subjects Undergoing Premarital Screening in the Jazan Region, Kingdom of Saudi Arabia. *Pakistan Journal of Medical Sciences*, 34(2), 316-321.
- Adams, J., Khan, H. T. A., Raeside, R. and White, D., (2007). *Research Methods for Graduate Business and Social Science Students*. Sage Publications Ltd., London.
- Aghemo, A. et al., (2012). Assessing Long-Term Treatment Efficacy in Chronic Hepatitis B and C: Between Evidence and Common Sense. *Journal of Hepatology*, 57(6), 1326-1335.
- Ali, H. S., (2018). *Prevalence of Hepatitis B and Hepatitis C in Relation to Minor Risk Factors in Kahuta Region*. MS Thesis, Department of Biosciences, Faculty of Health and Life Sciences. Capital University of Science & Technology Islamabad.
- Berg, T. et al., (2006). Extended Treatment Duration for Hepatitis C Virus Type 1: Comparing 48 Versus 72 Weeks of Peginterferon-alfa-2a Plus Ribavirin. *Gastroenterology*, 130(4), 1086-1097.
- Blumberg, B. S., (2002). The Discovery of the Hepatitis B Virus and the Invention of the Vaccine: A Scientific Memoir. *Journal of Gastroenterology and Hepatology*, 17(Suppl), S502-S503.
- Budderberg, F. et al., (2008). Transfusion Transmissible Infections and Transfusion Related Immunomodulation. *Best practice and Research-Clinical Anesthesiology*, 22(3), 503-517.
- Cheesbrough, M., (2006). *District Laboratory Practice in Tropical Countries* (2nd Ed.). Cambridge University Press, New York, America.
- Choo, Q. L. et al., (1989). Isolation of a cDNA Clone Derived from a Blood-Borne Non-A, Non-B Viral Hepatitis Genome. *Science*, 244(4902), 359-362.
- Creswell, J. W., (2007). *Qualitative Inquiry and Research Design: Choosing Among Five Approaches*. Thousand Oaks, CA: Sage Publications.
- Dufour, D. L., (2006). Biocultural Approaches in Human Biology. *American Journal of Human Biology*, 18(1), 1-9.
- Farooq, U. et al., (2017). Detection of HBsAg Mutants in the Blood Donor Population of Pakistan. *PLoS One*, 12(11), e0188066.
- Gibbs, G., (2008). *Analysing Qualitative Data*. London: Sage Publications.
- Gow, P. J., Mutimer, D., (2001). Treatment of Chronic Hepatitis. *BMJ*, 323(7322), 1164-1167.

- Health of Ministry, (2019). *National Guidelines for the Management of Viral Hepatitis*. Department of Health, Republic of South Africa.
- Huang, C. C. et al., (2013). One Single Nucleotide Difference Alters the Differential Expression of Spliced RNAs between HBV Genotypes A and D. *Virus Research*, 174(1-2), 18-26.
- Johnson, D. F. et al., (2013). Hepatitis B and C Infection in International Travelers. *Journal of Travel Medicine*, 20(3), 194-202.
- Kim, J. H. et al., (2009). Efficacy of Lamivudine on Hepatitis B Viral Status and Liver Function in Patients with Hepatitis B Virus-Related Hepatocellular Carcinoma. *Liver International*, 29(2), 203-207.
- Kothari, C. R., (2008). *Research Methodology: Methods and Techniques* (2nd Ed.). New Delhi: New Age International (P) Ltd.
- Kramvis, A., (2014). Genotypes and Genetic Variability of Hepatitis B Virus. *Intervirology*, 57(3-4), 141-150.
- Lau, J. Y., Wright, T. L., (1993). Molecular Virology and Pathogenesis of Hepatitis B. *Lancet*, 342(8883), 1335-1340.
- Legesse, B., (2014). *Research Methods in Agribusiness and Value Chains*. School of Agricultural Economics and Agribusiness, Haramaya University.
- Locarnini, S., (2004). Molecular Virology of Hepatitis Bvirus. *Seminar in Liver Disease*, 24(1) 3-10.
- Mesquita, P., Granato, C. and Castelo, A., (1997). Risk Factors Associated with Hepatitis C Virus (HCV) Infection among Prostitutes and Their Clients in the City of Santos, Sao Paulo State, Brazil. *Journal of Medical Virology*, 51(4), 338-343.
- Messina, J. P. et al., (2015). Global Distribution and Prevalence of Hepatitis C Virus Genotypes. *Hepatology*, 61(1), 77-87.
- Mohajan, H. K., (2017). Two Criteria for Good Measurements in Research: Validity and Reliability. *Annals of Spiru Haret University Economic Series*, 17(3), 58-82.
- Mohajan, H. K., (2018). *Aspects of Mathematical Economics, Social Choice and Game Theory*. PhD Dissertation, Jamal Nazrul Islam Research Centre for Mathematical and Physical Sciences (JNIRCMPS), University of Chittagong, Chittagong, Bangladesh.
- Mohajan, H. K., (2020). Quantitative Research: A Successful Investigation in Natural and Social Sciences. *Journal of Economic Development, Environment and People*, 9(4), 50-79.
- Mohajan, H. K., (2024a). Alcoholic Liver Disease: Diagnosis and Treatment Strategies. Unpublished Manuscript.
- Mohajan, H. K., (2024b). Alcoholic Hepatitis: Diagnosis and Management Procedures. Unpublished Manuscript.
- Mohajan, H. K., (2024c). Anatomy of Human Liver: A Theoretical Study. Unpublished Manuscript.
- Mohajan, H. K., (2024d). Liver Diseases: Epidemiology, Prevention, and Management Strategy. Unpublished Manuscript.
- Mohajan, H. K., (2024e). A Study on Functions of Liver to Sustain a Healthy Liver. Unpublished Manuscript.
- Mohajan, H. K., (2024f). Hepatitis A Virus (HAV) Infection: A Prevention Strategy through Hygienic Maintenance and Vaccination. Unpublished Manuscript.
- Mohajan, H. K., (2024g). Prevention of Hepatitis B Virus (HBV) is Essential to Avoid Chronic Liver Disease. Unpublished Manuscript.
- Mohajan, H. K., (2024h). Management Strategies of Fatal Liver Infection Due to Hepatitis C Virus (HCV). Unpublished Manuscript.
- Mohajan, H. K., (2024i). Clinical Practice, and Diagnosis and Treatment Strategies of Chronic Hepatitis D Virus (HDV). Unpublished Manuscript.
- Mohajan, H. K., (2024j). Transmission, Diagnosis, and Treatment of Acute and Chronic Hepatitis E. Unpublished Manuscript.
- Mohajan, H. K., (2024k). Hepatitis G Viruses (HGV): A Study on Prevalence, Transmission, and Co-Infection. Unpublished Manuscript.
- Mohajan, H. K., (2024l). Epidemiological Investigation of Hepatitis F Viruses (HFV). Unpublished Manuscript.
- Mohajan, H. K., (2024m). Alcoholic Liver Cirrhosis: A Chronic Liver Failure Due to Alcohol Abuse. Unpublished Manuscript.

- Mohajan, H. K., (2024n). Prevention and Treatment Strategies of Viral Hepatitis. Unpublished Manuscript.
- Purcell, R., (1997). The Hepatitis C Virus: Overview. *Hepatology*, 26(S3), 11S-14S.
- Sathiyasekaran, M., Ramaswamy, G., (2020). Diagnosis and Management of Hepatitis B and Hepatitis C Infections in Children. *Pediatric Infectious Disease*, 2(1), 12-18.
- Schaefer, S., (2005). Hepatitis B Virus: Significance of Genotypes. *Journal of Viral Hepatitis*, 12(2), 111-124.
- Shi, W. et al., (2012). Subgenotype Reclassification of Genotype B Hepatitis B Virus. *BMC Gastroenterology*, 12(1), 116.
- Smith, D. B. et al., (2014). Expanded Classification of Hepatitis C Virus into 7 Genotypes and 67 Subtypes: Updated Criteria and Genotype Assignment Web Resource. *Hepatology*, 59(1), 318-327.
- Stanaway, J. D. et al., (2016). The Global Burden of Viral Hepatitis from 1990 to 2013: Findings from the Global Burden of Disease Study 2013. *Lancet*, 388(10049), 1081-1088.
- Strickland, G. T. et al., (2008). Hepatitis C Vaccine: Supply and Demand. *The Lancet Infectious Diseases*, 8(6), 379-386.
- Sunbul, M., (2014). Hepatitis B Virus Genotypes: Global Distribution and Clinical Importance. *World Journal of Gastroenterology*, 20(18), 5427-5434.
- Szabó, E. et al., (2004). Similarities and Differences in Hepatitis B and C Virus Induced Hepatocarcinogenesis. *Pathology Oncology Research*, 10(1), 5-11.
- Tremolada, F. et al., (1992). Long-term Follow-up of Non-A, Non-B (Type C) Post-Transfusion Hepatitis. *Journal of Hepatology*, 16(3), 273-281.
- Ward, J. W., Hinman, A. R., (2019). What is Needed to Eliminate Hepatitis B Virus and Hepatitis C Virus as Global Health Threats. *Gastroenterology*, 156(2), 297-310.
- WHO, (2017). Global Hepatitis Report, 2017. Geneva: World Health Organization. www.who.int/hepatitis/publications/globalhepatitisreport2017/en/.

Copyrights

Copyright for this article is retained by the author(s), with first publication rights granted to the journal.

This is an open-access article distributed under the terms and conditions of the Creative Commons Attribution license (<http://creativecommons.org/licenses/by/4.0/>).