

Prevention and Treatment Strategies of Viral Hepatitis

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/IST.2788-7030.2025.07.003

Abstract

Hepatitis is a viral infection defined by inflammation of the liver. It is a wide range of complex situations that affect and damage the liver through the short-term acute illness or a lifelong chronic infection. Viral hepatitis is the major cause of chronic liver disease that kills about 1.4 million people worldwide each year. If the disease is untreated may progress to liver cirrhosis, liver failure, and hepatocellular carcinoma (HCC). Poor sanitation and virus infected blood transfusion are the most common causes of viral hepatitis in low- and middle- income countries. Viral hepatitis is one of the emerging serious public health problems worldwide that needs special attention immediately. This study tries to discuss the aspects of viral hepatitis to reduce the global infection of this disease.

Keywords: viral hepatitis, epidemiology, vaccination, treatment

1. Introduction

Viral hepatitis is an inflammation of the liver that is caused by one of the five different types of infectious hepatitis viruses A, B, C, D, and E. Hepatotropic viruses are the etiological factor in most cases of acute hepatitis (Zeng et al., 2016). More than 325 million people worldwide have hepatitis B or C 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).

From 1990 to 2019, the incidence rates of hepatitis A virus (HAV), hepatitis C virus (HCV), and hepatitis E virus (HEV) infection have remained stable, but the incidence of hepatitis B virus (HBV) infection has declined due to increases in HBV vaccination rates. In 2020, HBV and HCV related disease led to 1.1 million deaths worldwide (Zeng et al., 2021).

Common symptoms of viral hepatitis are dark urine, pale or clay-colored stools, fatigue, usually low-grade fever, itching, jaundice, loss of appetite, nausea, vomiting, weight loss, abdominal pain, and breast development among males (Koff, 1998). At present there are effective and licensed vaccines to prevent the viruses HAV, HBV and HEV. Furthermore, HDV can be prevented as HBV immunization is protective against HDV (Health of Ministry, 2019).

2. Literature Review

In any research, the literature review is an elementary section where research works of previous researchers are introduced briefly to make familiar with the new researchers in the research area (Polit & Hungler, 2013). It helps the researchers to understand the subject, and it serves as an indicator of the subject that has been carried out before (Creswell, 2007). Raymond S. Koff has provided the conceptual framework of viral hepatitis by considering the epidemiology and virology of the disease (Koff, 1974). Haradhan Kumar Mohajan has realized that HBV infects liver and in advanced stage it may cause chronic hepatitis, cirrhosis and hepatocellular carcinoma (HCC). He has also discussed the HBV infection, its treatment, and prevention through the

vaccination (Mohajan, 2024g).

Ajeet S. Bhadoria and his coworkers have stated that viral hepatitis refers to a pathologic condition wherein an infection due to hepatitis viruses causes inflammation of the liver that contributes substantially to the global burden on healthcare (Bhadoria et al., 2022). Mirza Adil Beig and his coauthors have adopted various strategies for the elimination of viral hepatitis. They have also explained that to combat viral hepatitis it is necessary to enhance vaccination coverage and also it requires sustained collaborative efforts among all stakeholders, including governments, healthcare providers, and communities (Beig et al., 2023).

Sema Mandal and her coworkers have actively surveyed unexplained pediatric acute hepatitis in children younger than 16 years. The results can inform diagnostic testing recommendations, clinical management, and exploratory in vitro or clinical studies of pediatric acute hepatitis of unknown aetiology (Mandal et al., 2023). Roberta D'Ambrosio and Alessio Aghemo have studied antiviral treatment of chronic HCV for the persistent eradication of the virus through the sustained virological response (SVR) (D'Ambrosio & Aghemo, 2012).

3. Research Methodology of the Study

Research is a hard-working search, scholarly inquiry, and investigation that aim for the discovery of new facts and findings (Adams et al., 2007). Researchers often write a methodology section with details of the research analysis. It is considered as a way of explaining how a research work is carried out (Kothari, 2008). Methodology is a system of explicit rules and procedures in which research is based, and against which claims of knowledge are evaluated (Ojo, 2003). Research methodology provides the principles to the researchers for organizing, planning, designing and conducting good research (Legesse, 2014). It helps to identify research areas and projects within these areas (Blessing et al., 1998).

The paper is prepared on the basis of secondary data sources of viral hepatitis. The essential and necessary data are collected from previous research articles of reputed journals, published books of world-famous authors, handbooks of renowned scholars, conference papers on recent important topics, websites, etc. (Mohajan, 2020, 2024a-e). In the study we have tried to maintain the reliability and validity throughout the research (Mohajan, 2017, 2018).

4. Objective of the Study

Main objective of this article is to discuss the aspects of viral hepatitis that leads to significant morbidity and mortality in patients with acute and chronic infections (Ahmed et al., 2022). Hepatitis viruses are either ribonucleic acid (RNA), such as hepatitis A, C, D, and E, or deoxyribonucleic acid (DNA), such as hepatitis B. An estimated 257 million individuals are chronically infected with HBV, and 71 million with HCV with 1.45 million deaths each year (Health of Ministry, 2019). Other minor objectives of the study are as follows:

- 1) to focus on chronic viral hepatitis,
- 2) to highlight on diagnosis and treatment of viral hepatitis, and
- 3) to discuss hepatitis viruses A, B, C, D, and E.

5. Hepatitis A Virus (HAV)

Hepatitis A virus (HAV) is a frequent type of viral hepatitis. It is a single-stranded RNA virus surrounded by a protein capsid that replicates in the liver, secreted in the bile, blood, and shed in stool. It is a common disease with serologic evidence of infection by an enterovirus of the Picornaviridae family that causes acute hepatitis (Heymann, 2008). It is typically a self-limiting disease and usually causes mild illness characterized by sudden onset of non-specific symptoms. In addition to the liver, HAV afflicts also other vital organs, such as heart, gastrointestinal tract, pancreas, and spleen (Koff, 1998).

The source of infection resides in contaminated food and the transmission takes place by the oral pathway. Transmission of HAV occurs almost exclusively through contact of an infected person, traveling to an endemic region, and ingestion of contaminated food and water (enterically). The incubation period of HAV is from 15-45 days (WHO, 2019).

Some symptoms of it are fatigue, itching, loss of appetite, low-grade fever, dark urine, nausea, vomiting, anorexia, malaise, diarrhea, headache, febrility, pale or clay-colored stools, and yellow skin (jaundice). During HAV infection the aspartate aminotransferase (AST) serum level increases and reaches its maximal values (Heymann, 2008). Urine contains serum bilirubin and urobilinogen, but does not increase significantly. The liver is moderately enlarged. The blood yields leukopenia with relative lymphocytosis. Necrosis may be of solitary, focal or diffuse characters (Murphy et al., 2013).

The VAH never progresses into chronicity. But it can cause debilitating symptoms and acute liver failure, which is associated with high mortality. A severe course of the disease with fulminant hepatic necrosis and hepatic coma leading to death is exceptional (0.1%) (Franco et al., 2012). There is no evidence of the disease transition

to chronic hepatitis and is rarely fatal, and within six months the infected patient is cured completely without causing any longstanding chronic hepatitis (Little et al., 2018). During the subsequent period, the jaundice disappears and in a majority of cases the disease retreats during 3–6 weeks, and the majority of patients become completely healthy with the physical and psychological activities return (Mohajan, 2024f). Sometimes the patient still suffers from weakness, increased tiredness, arthralgia or dyspeptic disturbances for several months after the recovery from the disease due to post hepatitis syndrome (Linder & Malani, 2017).

The VAH is spread world-wide and occurs in epidemics predominantly among children and young people who are often asymptomatic, but accurate figures are lacking (Mohajan, 2024l). It affects more often organized collectives, such as kindergartens, schools, police lines, military units, etc. (WHO, 2019). If an individual is infected with HAV s/he may not be infected further, since it causes lifetime immunity after first infection. Antibodies against the virus belong to the IgM class, in later period to that of IgG. At present there are two vaccines available that provide active immunization against HAV: Harvix and Vaqta (Ambrosch et al., 2004).

5.1 Hepatitis B Virus (HBV)

The HBV is a 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). Other antigenic determinants are deep antigen HBcAg that is reproduced in the nucleus, contains DNA; HBeAg that appears in the cytoplasm, reflects the replication activity of the virus (Lee, 1997). This virus has morphological and serological markers, and its particles are visible in an electron microscope in the nucleus and cytoplasm. It replicates in hepatocytes and to a lesser extent in stem cells in the pancreas, bone marrow and spleen. It is an irritation and swelling of the liver (Gerlich, 2013).

Early symptoms of it are loss of appetite, fatigue, low fever, muscle and joint aches, nausea and vomiting, jaundice, dark urine right upper quadrant pain, and hepatomegaly (Farooq et al., 2017; Mohajan, 2024k). The HBV is transmitted in human body through the bodily fluids (parenterally) that trigger an immune reaction through the sexual contact, intravenous drug use, transfusion of blood and blood products, and pregnant mother to infant (Aghemo et al., 2012).

The HBV can cause acute hepatitis as well as chronic hepatitis. Sometimes it may develop to more serious liver diseases, and ultimately may cause liver damage. The incubation period of HBV is from 40 to 180 days (Alotaibi, 2023). The disease is successfully treated with oral medications. About 90% of adult HBV patients have a full recovery, but 5-10% of the patients will develop chronic hepatitis with complications, such as cirrhosis and hepatocellular carcinoma (DePaola, 2003). Lamivudine is a safe effective antiviral drug for treating chronic HBV infection and Interferon Alfa is the only drug licensed for the treatment of it (Kim et al., 2009). The destruction of the hepatocyte and the elimination of the virus are carried out by the cells of the immune system. There are two vaccines available for HBV immunization that utilizes recombinant DNA technology: Engerix-B and Recombivax (WHO, 2017b).

African and the south-east Asian regions carry a high share of the global HBV burden, and the top three countries carrying the highest burden are China (74 million), India (17 million) and Nigeria (15 million) that make up 29%, 6.6% and 5.8% of the global burden of HBV, respectively (Rochwerg et al., 2019). Healthcare providers have three to five times the rate of HBV infection compared to the general population (Levy et al., 1998). The virus accounts for three-quarters of the 1.1 million annual deaths from complications of liver cirrhosis but the incidence of it is declining worldwide due to vaccination (Cooke et al., 2019). An estimated 257 million people worldwide have chronic hepatitis B and more than 686,000 people die every year due to complications of it, such as cirrhosis and liver cancer (WHO, 2021).

5.2 Hepatitis C Virus (HCV)

The HCV is a single-stranded RNA Flavivirus encoding for a capsid protein, two envelope proteins, and some nonstructural proteins (Mohajan, 2024h). There are no direct morphological markers of the virus. The virus replicates in the hepatocyte and in other cells, such as lymphocytes, and macrophages. It has a cytopathic effect and causes immune disorders (Waheed et al., 2009).

Most people (70-80%) have no symptoms when they are first infected with hepatitis C. Some symptoms of it are jaundice, loss of appetite, nausea, vomiting, fatigue, fever, itching, pain in the right upper abdomen, abdominal swelling due to fluid, clay-colored or pale stools, and dark urine (Purcell, 1997). Some nonspecific symptoms of HCV are fatigue, nausea, and/or abdominal pain. Chronic HCV infection is normally a slow progressive disease that may produce few or no symptoms for many years after infection (Franciscus, 2017).

The HCV can be spread in human body through the contact with infected blood especially through the hemophiliacs, dialysis patients, and intravenous drug users (parenterally). Other modes of transmission are sexual, perinatal, idiopathic, and pregnant mother to infant (Tremolada et al., 1992). The infection can be very serious. The incubation period of this virus is 15 to 150 days. This virus may stay in the liver for years and it is

not discovered until much damage is done in the liver (Liou et al., 1992).

The global incidence of HCV is 1.43-1.5 million individuals; the most affected regions are Central and East Asia and North Africa, and the top three countries with the highest disease burden are China (9.48 million), Pakistan (7.39 million) and India (6.13 million) (Dugan et al., 2021). About 170 million people are infected with HCV worldwide. About 71 million people chronically infected and about 700,000 people die each year worldwide from HCV (WHO, 2017a).

About 85% HCV patients will develop chronic hepatitis that is the major cause of cirrhosis and hepatocellular carcinoma, and may be more progressive in men than women (Liang et al., 2000). Antiviral medicines, such as pegylated interferon and ribavirin can be used to limit liver damage. About 30% of the infected patients are cured within six months and about 70% can develop chronic infection (D'Ambrosio & Aghemo, 2012).

5.3 Hepatitis D Virus (HDV)

The HDV is a negative-strand incomplete RNA virus that requires HBsAg (HDV virion) for its viral envelope and transmission. It is the only member of the genus Deltavirus, is from the Deltaviridae family (Kiesslich et al., 2009). The HDV only occurs as a co-infection or super-infection with acute HBV (parenterally), which may then progress to severe fulminant infection and may cause acute hepatitis occurs immediately (Mohajan, 2024i). Super-infection is clinically manifested by exacerbation and rapid progression of the disease up to the development of liver cirrhosis (Caredda et al., 1985). The HDV may also be transmitted through sexual activity. It only occurs primarily in drug addicts and persons with hemophilia. The screening of the blood supply for HBV has altered the epidemiology of HDV (Liaw et al., 1990). Symptoms of it are abdominal pain, dark colored urine, fatigue, jaundice, nausea, vomiting, joint pain, and loss of appetite (Miao et al., 2020).

At present there is no vaccine available for HDV. No specific treatment is available for HDV infected people. Oral drugs effective against HBV are ineffective against HDV (Loureiro et al., 2021). The pegylated interferon alpha (Peg-IFN α) is the generally recommended treatment to suppress the HDV for some patients (De Ledingham et al., 2021). At present the worldwide prevalence of HDV is about to be 15 to 20 million that was about 48-70 million as previously. Most prevalence regions are the Mediterranean, Middle East, Pakistan, Central and Northern Asia, Japan, Taiwan, Greenland, parts of Africa, the Amazon Basin, and certain areas of the pacific (Niro et al., 2012).

Top three countries with HDV prevalence are Mongolia (36.9%), Guinea-Bissau (23.9%), and Gabon (22%). The highest prevalence is seen in individuals with intravenous drug user followed by commercial sex workers, men who have sex with men, hemodialysis recipients, HIV positive individuals, HCV-positive individuals, and patients with cirrhosis (Stockdale et al., 2020).

5.4 Hepatitis E Virus (HEV)

The HEV is a small, icosahedral, non-enveloped, single-stranded, positive-sense RNA virus with genome of 7.2kb and 27-34nm in diameter that is highly unstable due to the lack of a lipid membrane. It is highly unstable due to the lack of a lipid membrane (Mayr et al., 2018). The prevalence is highest in East and South Asia, and Bangladesh, India, China, Ethiopia, and Kenya carry the highest burdens of HEV infection (Zeng et al., 2021). Every year there are an estimated 20 million Hepatitis E infections, and 56,600 Hepatitis E-related deaths (WHO, 2020).

The transmission of HEV is similar to HAV, and is by the oral-fecal route (enterically), but it may develop into acute liver failure and is associated with higher mortality (Mayr et al., 2018). Other routes of transmission are consumption of contaminated food, such as raw or undercooked meat derived from infected animals and through transfusion of infected blood (Lewis et al., 2010). The symptoms of hepatitis E are jaundice, fever, tiredness, loss of appetite, malaise, anorexia, nausea, vomiting, abdominal pain, joint pain, hepatomegaly, pruritic, dark urine, pale stools, and arthralgia (Mirazo et al., 2014).

The HEV patient has a higher risk of fulminant hepatitis and may cause acute liver failure in a few days. There is no HEV vaccine available, and treatment is palliative (Wedemeyer et al., 2012). The HEV causes acute hepatitis that recovers completely without causing any longstanding chronic hepatitis. Acute hepatitis is marked by sudden and massive death of the hepatocytes over a short period of time. It creates a lifelong immunity following natural infection (Wu et al., 2020).

5.5 Hepatitis F Virus (HFV)

Hepatitis F is a hypothetical virus linked to viral hepatitis. An enteric agent responsible for sporadic non-A, non-E hepatitis is tentatively called hepatitis F virus (HFV) or hepatitis French virus (HFV) and has been described by two groups (Fagan et al., 1992). In 1987, the disease was transmitted to cynomolgus macaques and tamarins, and 27-34nm viral-like particles that consist of double-stranded DNA of approximately 20kb were observed in stool samples by electron microscopy. The virology, epidemiology, hepatotoxicity, and clinical

importance of HFV are quite uncertain, and are not determined yet (Bradley et al., 1987; Mohajan, 2024l).

5.6 Hepatitis G Virus (HGV)

The hepatitis G virus (HGV) is also known as GB virus C (GBV-C) that is a newly described human virus of member of the Flaviviridae family and is similar genome organization as hepatitis C virus (HCV), and may be a cause of chronic liver disease (Mohajan, 2024m). It is a single-stranded, spherical enveloped, positive-sense RNA virus of the Flaviviridae family and a member of the genus Pegivirus and about 50nm in diameter (Alter, 1996). Blood-borne, and sexual and vertical transmissions of GBV-C have been identified. Children remain infected and asymptomatic for long periods (Brechot et al., 1998). Some studies suggested that GBV-C is a major cause of life-threatening liver diseases, and also some studies suggested that it does not cause chronic liver disease in human (Linnen et al., 1996).

6. Chronic Viral Hepatitis

This may be caused by all hepatitis viruses, except for HAV in which chronic inflammation predominates. The liver becomes large, and becomes red in color. The incubation period is from 2 to 26 weeks (Mohajan, 2024m). It develops necrosis of hepatocytes, hydropic and balloon dystrophy of hepatocytes, and the Kaunsilmen's bodies. In the portal tracts and in the lobular stroma there is abundant infiltration, represented mainly by lymphocytes and macrophages (Brundage & Fitzpatrick, 2006). During pre-jaundice period nonspecific symptoms are seen and during jaundice period clinical manifestations, such as cyclic icteric are visible (Mohajan, 2024j). To confirm the damage, a morphological examination of the liver biopsy is necessary (Jackson et al., 2018).

7. Diagnosis and Treatment

Diagnosis of HAV is the combination with serologic tests for IgM anti-HAV and IgG anti-HAV. The main diagnostic test for HCV is the enzyme-linked immunosorbent assay (ELISA) for anti-HCV and RT-PCR (Larson & Carithers, 2001). Serologic testing for HDV and anti-HDV is used to detect infection. Treatment of the viral hepatitis is palliative and supportive. Prevention is the most effective way against the disease. Bed rest and fluids may be prescribed especially during the acute phase (Lavanchy, 2004). No special drug considerations are required for a patient who has completely recovered from viral hepatitis. If a patient has chronic active hepatitis or is a carrier of the viruses and has impaired liver function, then dose modification is necessary (Capolunghi et al., 2013). HCV can be treated through the combination therapy with interferon and ribavirin. There is currently no treatment for HDV infection. A liver transplant is considered for patients with end-stage liver disease (D'Ambrosio & Aghemo, 2012).

8. Conclusions

Five main viral hepatitis are A, B, C, D, and E that are caused by a viral infection affecting the liver and its cells. Viral hepatitis is a preventable disease that becomes a global public health threat and an increased risk for liver disease, cancer, and premature death. It is matter of concern that hepatitis B and C are responsible for 96% of all hepatitis mortality worldwide. It is a significant cause of acute and chronic viral hepatitis worldwide. It is recognized as a severe global health problem in both developing and developed countries for the morbidity and mortality. Therefore, the burden of viral hepatitis is increasing globally. At present there are many options for treatment and vaccination of the viral hepatitis. The advent of new therapies and vaccines, the possibility of controlling of the disease is satisfactory. Recently, various strategies are adopted for the elimination of viral hepatitis. For this we require sustained collaborative efforts of the society, such as governments, healthcare providers, and communities.

References

- Adams, P. C. et al., (2018). Therapeutic Recommendations in HFE Hemochromatosis for p.Cys282Tyr (C282Y/C282Y). Homozygous Genotype. *Hepatology International*, 12(2), 83-86.
- Aghemo, A., Lampertico, P. and Colombo, M., (2012). Assessing Long-Term Treatment Efficacy in Chronic Hepatitis B and C: between Evidence and Common Sense. *Journal of Hepatology*, 57(6), 1326-1335.
- Ahmed, Z. et al., (2022). Viral Hepatitis: A Narrative Review of Hepatitis A-E. *World Journal of Meta-Analysis*, 10(3), 99-121.
- Alotaibi, B. S., (2023). Hepatitis B Virus Infection, Structure, Genotypes, and Epidemiology: A Review. *Pharmacy Practice*, 21(3), 2856.
- Alter, H. J., (1996). The Cloning and Clinical Implications of HGV and HGBV-C. *New England Journal of Medicine*, 334(23), 1536-1537.
- Ambrosch, F. et al., (2004). Rapid Antibody Response after Vaccination with a Virosomal Hepatitis A Vaccine. *Infection*, 32(3), 149-152.

- Beig, M. A. et al., (2023). Tackling the Burden of Viral Hepatitis in India: A Call for Collaborative Efforts. *Journal of Hepatitis Research*, 8(1), 1049.
- Bhadoria, A. S. et al., (2022). Viral Hepatitis as a Public Health Concern: A Narrative Review about the Current Scenario and the Way Forward. *Cureus*, 14(4), e21907.
- Blessing, L. T. M. et al., (1998). An Overview of Descriptive Studies in Relation to a General Design Research Methodology. In E. Frankenberger, P. Badke-Schaub & H. Birkhofer (Eds.), *Designers: The Key to Successful Product Development*. Berlin: Springer Verlag.
- Bradley, D. W. L. et al., (1987). Enterically Transmitted Non-A, Non-B Hepatitis: Serial Passage of Disease in Cynomolgus Macaques and Tamarins and Recovery of Disease Associated 27-34 nm Virus Like Particles. *Proceedings of the National Academy of Sciences*, 84(17), 6277-6281.
- Brechot, C. et al., (1998). Impact of HBV, HCV and GBV-c/HGV on Hepatocellular Carcinomas in Europe. Results of a European Concerted Action. *Journal of Hepatology*, 29(2), 173-183.
- Brundage, S. C., Fitzpatrick, E. N., (2006). Hepatitis A. *American Family Physician*, 73(12), 2162-2168.
- Capolunghi, F. et al., (2013). Why Do We Need IgM Memory B Cells? *Immunology Letters*, 152(2), 114-120.
- Careda, F. et al., (1985). Hepatitis B Virus-Associated Coinfection and Superinfection with Delta Agent: Indistinguishable Disease with Different Outcome. *Journal of the Infectious Diseases*, 151(5), 925-928.
- Cooke, G. S. et al., (2019). Accelerating the Elimination of Viral Hepatitis: A Lancet Gastroenterology & Hepatology Commission. *Lancet Gastroenterology & Hepatology*, 4(2), 135-184.
- Creswell, J. W., (2007). *Qualitative Inquiry and Research Design: Choosing Among Five Approaches*. Thousand Oaks, CA: Sage Publications.
- D'Ambrosio, R., Aghemo, A., (2012). Treatment of Patients with HCV Related Cirrhosis: Many Rewards with Very Few Risks. *Hepatitis Monthly*, 12(6), 361-368.
- De Ledinghen, V. et al., (2021). Safety and Efficacy of 2mg Bulevirtide in Patients with Chronic HBV/HDV Coinfection. *Hepatology*, 74(Suppl.1), 16A-17A.
- DePaola, L. G., (2003). Managing the Care of Patients Infected with Bloodborne Diseases. *Journal of the American Dental Association*, 134(3), 350-358.
- Dugan, E. et al., (2021). Global Prevalence of Hepatitis C Virus in Women of Childbearing Age in 2019: A Modelling Study. *Lancet Gastroenterology Hepatology*, 6(3), 169-184.
- Fagan, E. A. et al., (1992). Toga Virus-Like Particles in Fulminant Sporadic Non-A, Non-B Hepatitis and after Transplantation. *Journal of Medical Virology*, 38(1), 71-77.
- Farooq, U. et al., (2017). Detection of HBsAg Mutants in the Blood Donor Population of Pakistan. *PLoS One*, 12(11), e0188066.
- Franciscus, A., (2017). *A Guide to Understanding Hepatitis C*. Hepatitis C Support Project, HCV Advocate.
- Franco, E. et al., (2012). Hepatitis A: Epidemiology and Prevention in Developing Countries. *World Journal of Hepatology*, 4(3), 68-73.
- Gerlich, W. H., (2013). Medical Virology of Hepatitis B: How It Began and Where We Are Now. *Virology Journal*, 10(1), 239.
- 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.
- Heymann, D. L., (2008). Viral Hepatitis A. In: *Control of Communicable Diseases Manual*, 19th Ed., pp. 278-284. American Public Health Association, Washington.
- Jackson, K. et al., (2018). Diagnostics of Hepatitis B Virus: Standard of Care and Investigational. *Clinical Liver Disease*, 12(1), 5-11.
- Kiesslich, D. et al., (2009). Influence of Hepatitis B Virus (HBV) Genotype on the Clinical Course of Disease in Patients Coinfected with HBV and Hepatitis Delta Virus. *Journal of the Infectious Diseases*, 199(11), 1608-1611.
- 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.
- Koff, R. S., (1974). Current Concepts of Viral Hepatitis and a Peek into the Future. *The Yale Journal of Biology and Medicine*, 47(4), 252-259.

- Koff, R. S., (1998). Hepatitis A. *Lancet*, 351(9116), 1643-1649.
- Kothari, C. R., (2008). *Research Methodology: Methods and Techniques* (2nd Ed.). New Delhi: New Age International (P) Ltd.
- Larson, A. M., Carithers, R. L., (2001). Hepatitis C in Clinical Practice. *Journal of Internal Medicine*, 249(2), 111-1120.
- Lau, J. Y., Wright, T. L., (1993). Molecular Virology and Pathogenesis of Hepatitis B. *Lancet*, 342(8883), 1335-1340.
- Lavanchy, D., (2004). Hepatitis B Virus Epidemiology, Disease Burden, Treatment, and Current and Emerging Prevention and Control Measures. *Journal of Viral Hepatitis*, 11(2), 97-107.
- Lee, W. M., (1997). Hepatitis B Virus Infection. *New England Journal of Medicine*, 337(24), 1733-1745.
- Legesse, B., (2014). *Research Methods in Agribusiness and Value Chains*. School of Agricultural Economics and Agribusiness, Haramaya University.
- Levy, M. J., Herrera, J. L. and DiPalma, J. A., (1998). Immune Globulin and Vaccine Therapy to Prevent Hepatitis an Infection. *American Journal of Medicine*, 105(5), 416-423.
- Lewis, H. C. et al., (2010). Transmission Routes and Risk Factors for Autochthonous Hepatitis E Virus Infection in Europe: A Systematic Review. *Epidemiology and Infection*, 138(2), 145-166.
- Liang, T. J. et al., (2000). Pathogenesis, Natural History, Treatment, and Prevention of Hepatitis C. *Annals of Internal Medicine*, 132(4), 296-305.
- Liaw, Y. F. et al., (1990). Heterosexual Transmission of Hepatitis Delta Virus in the General Population of an Area Endemic for Hepatitis B Virus Infection: A Prospective Study. *Journal of the Infectious Diseases*, 162(5), 1170-1172.
- Linder, K. A., Malani, P. N., (2017). Hepatitis A. *JAMA*, 318(23), 2393.
- Linnen, J. et al., (1996). Molecular Cloning and Disease Association of Hepatitis G Virus. A Transfusion-Transmissible Agent. *Science*, 271(5248), 505-508.
- Liou, T.-C. et al., (1992). Detection of HCV RNA in Saliva, Urine, Seminal Fluid and Ascites. *Journal of Medical Virology*, 37(3), 197-202.
- Little, J. W. et al., (2018). *Dental Management of the Medically Compromised Patient* (9th Ed.) St. Louis: Missouri.
- Loureiro, D. et al., (2021). New Therapies for Hepatitis Delta Virus Infection. *Liver International*, 41(Suppl.1), 30-37.
- Mandal, S. et al., (2023). Paediatric Acute Hepatitis of Unknown Aetiology: A National Investigation and Adenoviraemia Case-Control Study in the UK. *Lancet Child & Adolescent Health*, 7(11), 786-796.
- Mayr, U. et al., (2018). Impact of Large Volume Paracentesis on Respiratory Parameters Including Transpulmonary Pressure and on Transpulmonary Thermodilution Derived Hemodynamics: A Prospective Study. *PLoS One*, 13(3), e0193654.
- Miao, Z. et al., (2020). Estimating the Global Prevalence, Disease Progression, and Clinical Outcome of Hepatitis Delta Virus Infection. *Journal of the Infectious Diseases*, 221(10), 1677-1687.
- Mirazo, S. et al., (2014). Transmission, Diagnosis, and Management of Hepatitis E: An Update. *Hepatic Medicine*, 6(4), 45-59.
- 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.
- Murphy, T. V. et al., (2013). Hepatitis A Vaccines. In: Plotkin, S. A., Orenstein, W. A., Offit, P. A., (Eds.), *Vaccines*, 6th Ed., pp. 183-204, Philadelphia: Elsevier.
- Niro, G. A. et al., (2012). Epidemiology and Diagnosis of Hepatitis D Virus. *Future Virology*, 7(7), 709-717.
- Ojo, S. O., (2003). Productivity and Technical Efficiency of Poultry Egg Production in Nigeria. *International Journal of Poultry Science*, 2(6), 459-464.
- Polit, D. F., Hungler, B. P., (2013). *Essentials of Nursing Research: Methods, Appraisal, and Utilization* (8th Ed.). Philadelphia: Wolters Kluwer/Lippincott Williams and Wilkins.
- Purcell, R., (1997). The Hepatitis C Virus: Overview. *Hepatology*, 26(S3), 11S-14S.
- Rochweg, B. et al., (2019). High Flow Nasal Cannula Compared with Conventional Oxygen Therapy for Acute Hypoxemic Respiratory Failure: A Systematic Review and Meta-Analysis. *Intensive Care Medicine*, 45(5), 563-572.
- Stockdale, A. J. et al., (2020). The Global Prevalence of Hepatitis D Virus Infection: Systematic Review and Meta-Analysis. *Journal of Hepatology*, 73(3), 523-532.
- 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.
- Waheed, Y. et al., (2009). Hepatitis C Virus in Pakistan: A Systematic Review of Prevalence, Genotypes and Risk Factors. *World Journal of Gastroenterology*, 15(45), 5647-5653.
- Wedemeyer, H. et al., (2012). Pathogenesis and Treatment of Hepatitis E Virus Infection. *Gastroenterology*, 142(6), 1388-1397.
- WHO, (2017a). World Health Organization. Hepatitis C. <http://www.who.int/mediacentre/factsheets/fs164/en/>
- WHO, (2017b). Hepatitis B Vaccines: WHO Position Paper–July 2017. *Weekly Epidemiological Record*, 92(27), 369-392.
- WHO, (2019). *WHO Immunological Basis for Immunization Series: Module 18: Hepatitis A*. Geneva: World Health Organization.
- WHO, (2020). World Health Organization. Hepatitis E. <https://www.who.int/news-room/factsheets/detail/hepatitis-e>.
- WHO, (2021). World Health Organization. Hepatitis B Fact Sheet N°204. <https://www.who.int/news-room/factsheets/detail/hepatitis-b>.
- Wu, C. et al., (2020). Hepatitis E Virus Infection during Pregnancy. *Virology Journal*, 17(1), 73.
- Zeng, D. Y. et al., (2021). Global Burden of Acute Viral Hepatitis and Its Association with Socioeconomic Development Status, 1990-2019. *Journal of Hepatology*, 75(3), 547-556.
- Zeng, F. et al., (2016). Epidemiology of Hepatitis B Virus Infection: Results from a Community Based Study of

0.15 Million Residents in South China. *Scientific Reports*, 6(1), 36186.

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/>).