

RESEARCH ARTICLE

A Comprehensive Study of Hepatitis B Infections in Bangladesh: Epidemiology, Risk factors and Clinical-Laboratory Correlations

Tanzila Akter¹, Md. Ramjan Ali², Md. Nurul Anwar Faruquee³, Md Samiul Bashir⁴, Shohanur Rahaman⁵, Md. Ashiqur Rahman⁶, Sadia Islam^{7*}

¹ *Tanzila Akter, Department of Virology, National Institute of Laboratory Medicine & Referral Center, Bangladesh, tanzilaishani@gmail.com*

² *Md. Ramjan Ali, Department of Laboratory Medicine, Dhaka medical College & Hospital, Bangladesh, ramjanalidmch1978@gmail.com*

³ *Md. Nurul Anwar Faruquee, Department of Laboratory Medicine, Dhaka medical College & Hospital, Bangladesh, sakib789736@gmail.com*

⁴ *Md Samiul Bashir, Department of Laboratory, Institute of Health Technology (IHT), Kurigram, Bangladesh, mtsamiulbashir@gmail.com*

⁵ *Shohanur Rahaman, Department of Microbiology, Popular Diagnostic Limited, Dhaka, Bangladesh, shohanbuhs@gmail.com*

⁶ *Md. Ashiqur Rahman, Department of Laboratory Medicine, Novus Clinical Research Services Limited (NCRSL), Bangladesh, ararashiqur@gmail.com*

⁷ *Sadia Islam, Department of Laboratory Medicine, Bangladesh Specialized Hospital, Bangladesh, sadia.buhs.6700@gmail.com*

* **Corresponding author:** Sadia Islam, sadia.buhs.6700@gmail.com

ABSTRACT

Background: Hepatitis B remains a major public health concern, with a significant burden in many countries, including Bangladesh. The disease can lead to chronic liver disease, cirrhosis, and hepatocellular carcinoma. Early detection through screening and understanding the clinical and laboratory profiles of infected individuals are crucial for timely intervention and reducing transmission. **Objective:** The objective of this study was to evaluate the clinical and laboratory profiles of patients with hepatitis B infection attending the outpatient departments of a tertiary care hospital in Bangladesh. **Methods:** This cross-sectional study was conducted at a tertiary care hospital in Bangladesh, including 90 hepatitis B patients diagnosed via serological markers. Demographic data, medical history (risk factors like drug use, sexual contacts, etc.), and clinical evaluations (liver function tests, serological markers) were recorded. Ultrasound imaging assessed liver abnormalities. Statistical analysis was performed with $p < 0.05$ considered significant. **Results:** Of the 90 patients, 55 (61.11%) were male and 35 (38.89%) were female. The mean age of male patients was 33.45 ± 9.28 years, and for female patients, it was 34.31 ± 13.14 years ($p = 0.5713$). The most common identified risk factor was mother-to-child transmission (MTC), observed in 16 (17.78%) patients. Other risk factors included chronic

ARTICLE INFO

Received: 8 October 2024 | Accepted: 6 November 2024 | Available online: 26 November 2024

CITATION

T. Akter, M. R. Ali, M. N. A. Faruquee, M. S. Bashir, S. Rahaman, M. A. Rahman, S. Islam. A Comprehensive Study of Hepatitis B Infections in Bangladesh: Epidemiology, Risk Factors and Clinical-Laboratory Correlations. *Viral Infections and Cancer Research*. 2024; 1(1): 8426. doi: 10.59429/vicr.v1i1.8426

COPYRIGHT

Copyright © 2024 by author(s). *Viral Infections and Cancer Research* is published by Arts and Science Press Pte. Ltd. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by/4.0/>), permitting distribution and reproduction in any medium, provided the original work is cited.

alcoholism (12.22%) and a history of surgeries (8.89%). A majority of patients (47.22%) were asymptomatic, with hepatitis B surface antigen (HBsAg) detected incidentally. HBsAg was positive in all patients, while hepatitis B e antigen (HBeAg) was present in 15 (16.67%) patients, indicating high infectivity. Anti-HBc and Anti-HBe were positive in 77 (85.56%) patients each. Hepatomegaly was found in 25 (27.78%) patients, and moderate fibrosis was detected in 3 (3.33%) patients on elastography. **Conclusion:** This study found that many hepatitis B patients were asymptomatic, with no identifiable risk factors. These findings highlight the need for widespread screening to detect asymptomatic cases and prevent transmission. Public education on risk factors and improved diagnostics are crucial for controlling hepatitis B in Bangladesh.

Keywords: Hepatitis B; Clinical presentation; Laboratory profile; Serology; Bangladesh

1. Introduction

Hepatitis B virus (HBV) infection remains a major global health challenge, contributing significantly to morbidity and mortality worldwide. Chronic hepatitis B affects approximately 350–400 million people globally, with regions such as South Asia, Sub-Saharan Africa, and Central Asia experiencing higher prevalence rates compared to other parts of the world. In Bangladesh, hepatitis B is a serious public health concern, with a significant proportion of the population affected. The prevalence of HBV in Bangladesh has been estimated at around 4–6%, but this varies across different regions and subpopulations, with higher rates observed in specific areas^[1].

HBV transmission occurs through parenteral exposure to infected blood or body fluids, sexual contact, and vertical transmission from mother to child during childbirth. In Bangladesh, the widespread use of unsterilized needles, particularly in healthcare settings and for traditional practices such as tattooing, has been identified as a major risk factor for increased HBV transmission. Additionally, people with multiple sexual partners, those undergoing hemodialysis, and individuals with a history of intravenous drug use are at elevated risk of acquiring the infection. Despite the high prevalence of HBV in Bangladesh, a significant portion of affected individuals remain asymptomatic and unaware of their infection status, leading to delayed diagnosis and treatment^[2].

HBV infection can range from mild symptoms to severe liver disease, including chronic hepatitis, cirrhosis, and hepatocellular carcinoma (HCC). The progression depends on factors like age at infection, immune response, and coinfection with hepatitis C or HIV. Acute HBV may be asymptomatic or cause jaundice, fatigue, and abdominal pain, while chronic infection leads to long-term liver damage^[3]. Evaluation includes liver function tests (e.g., ALT, AST, bilirubin) to assess liver injury, and serological markers (e.g., HBsAg, HBeAg) to determine infection stage. Imaging techniques like ultrasound, elastography, CT, and MRI help assess liver structure, fibrosis, and complications like cirrhosis or liver cancer. Chronic HBV infection can lead to serious complications like cirrhosis and hepatocellular carcinoma (HCC)^[4].

The risk of these complications depends on factors such as viral load, infection duration, and comorbid conditions. Cirrhosis can result in liver failure, while HCC is a leading cause of cancer-related deaths, with HBV being a major risk factor. Vaccination is the most effective preventive measure, recommended for newborns and high-risk groups^[5]. For those with established infection, antiviral therapy (e.g., lamivudine, tenofovir, entecavir) and regular monitoring are key in managing liver disease and preventing progression to cirrhosis or HCC. Treatment decisions are based on HBV DNA levels, liver enzymes, and the presence of complications^[6,7].

This study aims to investigate the clinical and laboratory profiles of patients with hepatitis B infection attending the outpatient departments of a tertiary care hospital in Bangladesh. Understanding the clinical

presentation, laboratory markers, and associated risk factors of HBV in this population help improve early diagnosis, management, and prevention strategies, ultimately contributing to better health outcomes in patients with HBV infection in Bangladesh.

2. Methodology

This was a cross-sectional study conducted in the outpatient department of a tertiary care medical institute in Bangladesh. The study aimed to analyze the clinical and laboratory profiles of patients with hepatitis B infection. A total of 90 patients diagnosed with hepatitis B based on serological markers were included in the study, following predefined inclusion and exclusion criteria. The duration of the study was one year. Written informed consent was obtained from adult patients, and from parents or guardians for patients under the age of 18. Confidentiality of patient information was maintained throughout the study.

2.1. Sample size calculation

The sample size for this study was determined based on pilot studies conducted on the clinical profile of hepatitis B patients. Using a power (1-beta error) of 80% and a confidence interval (1-alpha error) of 95%, the minimum required sample size was calculated to be 70 patients. As a result, 90 patients were included in the study to ensure statistical robustness and account for any potential dropouts or incomplete data.

2.2. Demographic and clinical data collection

Demographic information such as age, gender, and socioeconomic status was collected for all patients. A detailed clinical history was recorded, focusing on potential risk factors for hepatitis B transmission, including intravenous drug use, history of tattooing, sexual contacts, blood transfusions, and family history of liver disease. Patients were also asked about any symptoms suggestive of liver involvement, including jaundice, fatigue, abdominal discomfort, and other signs indicative of liver disease. A thorough physical examination was performed on all patients, with particular attention to signs of chronic liver disease such as hepatomegaly, splenomegaly, spider angiomas, and ascites. Additionally, the clinical examination aimed to detect any other signs of hepatic dysfunction.

2.3. Laboratory and diagnostic investigations

All patients underwent serological tests, including hepatitis B surface antigen (HBsAg), hepatitis B e antigen (HBeAg), anti-HBc, and anti-HBs to determine the infection status and assess the immune response to HBV. Additionally, quantitative HBV DNA levels were measured using polymerase chain reaction (PCR) to assess viral replication and inform treatment decisions. Liver function was evaluated through serum levels of bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and albumin. Abdominal ultrasound was performed on all patients to assess liver structure, detect cirrhosis, hepatomegaly, splenomegaly, and identify any space-occupying lesions, such as hepatocellular carcinoma (HCC)^[8].

2.4. Inclusion and exclusion criteria

Patients diagnosed with hepatitis B infection based on serological tests, aged 12 years and older, and who provided written informed consent to participate in the study. Exclusion criteria included patients or guardians who refused consent, those younger than 12 years, individuals on medications known to cause hepatic injury (e.g., methotrexate, isoniazid, valproic acid), those with uncontrolled systemic conditions like diabetes, hypertension, or autoimmune diseases, and patients diagnosed with primary hepatic malignancies or metastatic liver cancer.

2.5. Statistical analysis

Data were analyzed using SPSS version 21.0. Descriptive statistics were used to summarize demographic and clinical characteristics, presenting qualitative data as frequencies and percentages. For quantitative variables, an unpaired t-test was used to compare continuous data between groups, while qualitative data were analyzed using the Chi-square test. A p-value of <0.05 was considered statistically significant.

3. Results

A total of 90 patients diagnosed with chronic hepatitis B infection were included in this study. Among them, 55 (61.11%) were male and 35 (38.89%) were female. The mean age of male patients was 33.45 ± 9.28 years, and for females, it was 34.31 ± 13.14 years. Statistical analysis showed no significant difference in mean age between genders ($p = 0.5713$). The majority of patients were in the age group of 20 to 40 years, with males predominantly in the 30-40 years age group (30.00%) and females most commonly in the 20-30 years age group (15.56%) (**Table 1**).

Table 1. Age distribution of study population.

Age Group	Males (n=55)	Females (n=35)	Total (n=90)
<20 years	2 (3.64%)	4 (11.43%)	6 (6.67%)
20–30 years	18 (32.73%)	8 (22.86%)	26 (28.89%)
31–40 years	22 (40.00%)	7 (20.00%)	29 (32.22%)
41–50 years	8 (14.55%)	6 (17.14%)	14 (15.56%)
51–60 years	3 (5.45%)	5 (14.29%)	8 (8.89%)
>60 years	2 (3.64%)	5 (14.29%)	7 (7.78%)
Mean Age	33.45 ± 9.28	34.31 ± 13.14	33.78 ± 10.94

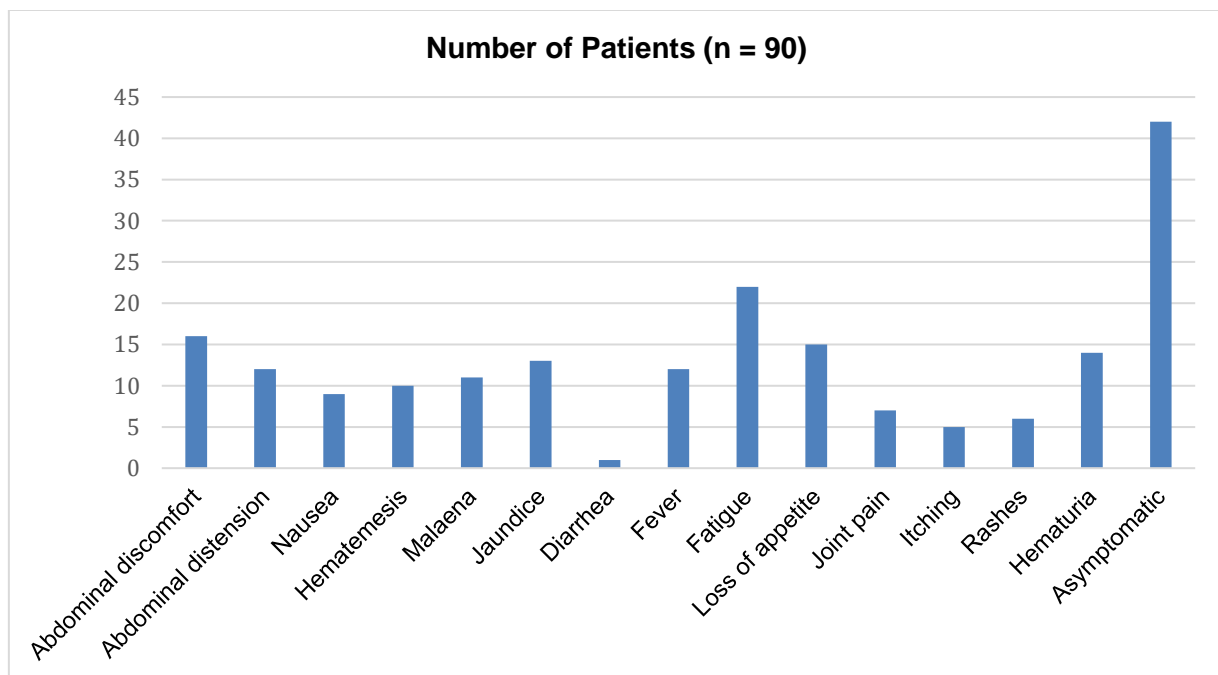


Figure 1. Clinical presentation of studied cases.

Regarding clinical presentation, 47.22% of patients were asymptomatic and diagnosed incidentally. Among symptomatic patients, fatigue was the most common complaint (28.89%), followed by abdominal discomfort (20.00%) and loss of appetite (18.89%). Other symptoms included jaundice (17.77%), abdominal distension (16.67%), and hematuria (15.56%) (**Figure 1**).

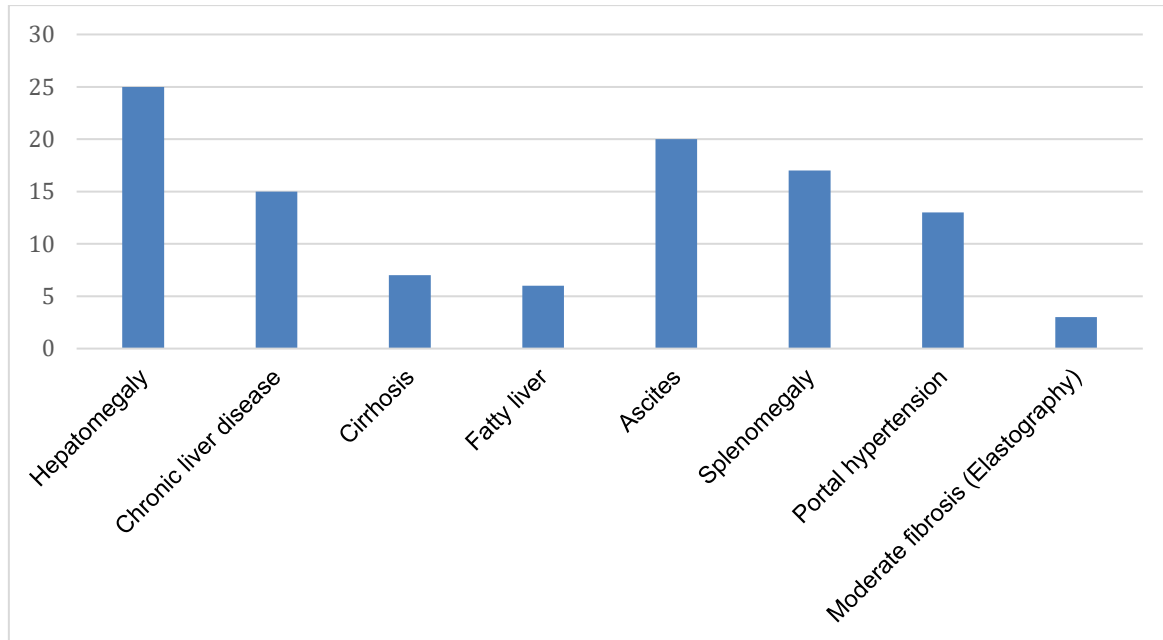


Figure 2. Ultrasonography of the cases with hepatitis B.

Abdominal ultrasonography was performed on all patients, revealing several notable findings. Hepatomegaly was observed in 27.78% of patients, while chronic liver disease was identified in 16.67%, including cirrhosis in 7.78% and fatty liver in 6.67%. Additional findings included ascites in 22.22%, splenomegaly in 18.89%, and portal hypertension in 14.44%. In selected cases, elastography was performed, revealing moderate fibrosis in 3.33% of patients (**Figure 2**).

The hematological and biochemical profiles of the patients revealed several important findings. Hemoglobin levels were above 12 g/dL in 77.78% of patients, indicating normal red blood cell production. Platelet counts exceeded 1.5 lakhs per cubic millimeter in 73.33% of patients. Serum total bilirubin was below 2 mg/dL in 88.89% of patients. Elevated SGOT and SGPT levels were observed in 70.00% and 67.78% of patients, respectively, suggesting liver stress. Serum albumin was above 3.5 g/dL in 83.33% of patients, indicating adequate hepatic synthetic function. Prothrombin time was within normal limits (<18 seconds) in 81.11% of patients. However, serum creatinine levels were elevated above 1 mg/dL in 44.44% of patients, pointing to renal involvement. Elevated alkaline phosphatase levels were noted in 16.67% of patients (**Table 2**).

Table 2. Hematological and biochemical profile of patients.

Parameter	Normal Range	Number of Cases (%)
Hemoglobin (g/dL)	>12	70 (77.78%)
Platelet count (×10 ⁴ /cmm)	>1.5 lakhs	54 (73.33%)
SGOT (IU/L)	<40	63 (70.00%)
SGPT (IU/L)	<40	61 (67.78%)
Total Bilirubin (mg/dL)	<2	80 (88.89%)

Parameter	Normal Range	Number of Cases (%)
Serum Albumin (g/dL)	>3.5	75 (83.33%)
Prothrombin time (s)	<18	73 (81.11%)
Serum Creatinine (mg/dL)	<1	50 (55.56%)
Alkaline Phosphatase (U/L)	<180	75 (83.33%)

Table 2. (Continued)

The serological profile of the patients revealed the following key findings: HBsAg was positive in 100% of patients, confirming chronic hepatitis B infection. HBeAg was detected in 16.67% of patients, suggesting high infectivity. Additionally, both Anti-HBc and Anti-HBe were positive in 85.56% of patients, indicating chronic infection with ongoing viral replication (**Table 3**).

Table 3. Serological profile of hepatitis b infected patients.

Serological Marker	Number of Cases (%)
HBsAg	90 (100.00%)
HBeAg	15 (16.67%)
Anti-HBc	77 (85.56%)
Anti-HBe	77 (85.56%)

The statistical analysis revealed significant correlations between clinical features, serological markers, and biochemical parameters. Key findings included a strong positive correlation between HBeAg positivity and elevated SGOT and SGPT levels ($p < 0.001$), suggesting active viral replication and liver inflammation. Hepatomegaly was positively correlated with elevated SGOT levels ($p = 0.004$), indicating that liver enlargement is associated with liver injury. Chronic alcohol use was significantly linked to elevated SGPT levels ($p = 0.02$), pointing to alcohol-induced liver damage. Additionally, platelet count showed a negative correlation with serum creatinine levels ($p = 0.03$), suggesting that renal dysfunction may impact hematopoiesis in these patients (**Table 4**).

Table 4. Correlation between serological markers and liver enzymes (SGOT, SGPT).

Parameter	SGOT (IU/L)	SGPT (IU/L)
HBeAg	0.612 ($p < 0.001$)	0.523 ($p < 0.001$)
Anti-HBc	0.310 ($p = 0.005$)	0.265 ($p = 0.015$)
Anti-HBe	0.275 ($p = 0.022$)	0.217 ($p = 0.05$)
HBsAg	0.490 ($p < 0.001$)	0.432 ($p < 0.001$)

This table shows the relationships between clinical features and biochemical parameters. For fatigue, positive correlations with SGOT, SGPT, and total bilirubin suggest that fatigue is linked to liver dysfunction, while a negative correlation with platelet count may reflect impaired hematopoiesis. Jaundice has strong positive correlations with liver enzymes and bilirubin, indicating it is directly related to liver impairment. A negative correlation with platelet count and serum albumin suggests worsened liver function. Abdominal discomfort also correlates with elevated liver enzymes and bilirubin, and negative correlations with platelet count and albumin reflect liver injury. Ascites is strongly correlated with higher liver enzymes and bilirubin, while being negatively correlated with platelet count and albumin, which points to liver decompensation (**Table 5**).

Table 5. Correlation between clinical features and biochemical parameters.

Clinical Feature	SGOT (IU/L)	SGPT (IU/L)	Total Bilirubin (mg/dL)	Platelet Count ($\times 10^4/\text{cmm}$)	Serum Albumin (g/dL)
Fatigue	0.317 (p=0.004)	0.287 (p=0.011)	0.234 (p=0.034)	-0.211 (p=0.045)	-0.192 (p=0.063)
Jaundice	0.479 (p<0.001)	0.467 (p<0.001)	0.372 (p<0.001)	-0.382 (p<0.001)	-0.308 (p=0.003)
Abdominal Discomfort	0.310 (p=0.005)	0.289 (p=0.009)	0.246 (p=0.027)	-0.241 (p=0.031)	-0.208 (p=0.050)
Ascites	0.509 (p<0.001)	0.493 (p<0.001)	0.432 (p<0.001)	-0.436 (p<0.001)	-0.373 (p<0.001)

This table highlights the correlations between ultrasonographic findings and biochemical parameters. Hepatomegaly is positively correlated with elevated SGOT, SGPT, and bilirubin, suggesting that liver enlargement is linked to liver injury. A negative correlation with serum albumin further supports liver dysfunction. Chronic liver disease shows strong positive correlations with liver enzymes and bilirubin, and negative correlations with albumin, indicating reduced liver function. Cirrhosis demonstrates the strongest correlations with elevated liver enzymes, bilirubin, and decreased albumin, highlighting severe liver damage. Finally, ascites is correlated with elevated liver enzymes and bilirubin and lower albumin levels, indicating liver decompensation and portal hypertension (**Table 6**).

Table 6. Correlation between hepatic abnormalities (ultrasonography findings) and biochemical parameters.

Ultrasonography Finding	SGOT (IU/L)	SGPT (IU/L)	Total Bilirubin (mg/dL)	Serum Albumin (g/dL)
Hepatomegaly	0.398 (p<0.001)	0.370 (p<0.001)	0.310 (p=0.003)	-0.268 (p=0.014)
Chronic Liver Disease	0.507 (p<0.001)	0.493 (p<0.001)	0.432 (p<0.001)	-0.389 (p<0.001)
Cirrhosis	0.567 (p<0.001)	0.523 (p<0.001)	0.502 (p<0.001)	-0.474 (p<0.001)
Ascites	0.521 (p<0.001)	0.487 (p<0.001)	0.469 (p<0.001)	-0.398 (p<0.001)

The analysis of risk factors revealed that 43.33% (n = 39) of the patients did not report any identifiable risk factors. Among those with known risk factors, mother-to-child transmission (MTC) was found in 17.78% of patients (n = 16). Chronic alcoholism was observed exclusively in male patients, accounting for 12.22% (n = 11) of the male cohort. Additionally, intravenous drug use (IVDU) was reported in 3 male patients (3.33%). Other risk factors included dental implants (5.56%, n = 5) and surgeries (8.89%, n = 8). Abortion as a risk factor was recorded in 2 females (2.22%) (**Table 7**).

Table 7. Distribution of risk factors for hepatitis b infection among male and female patients.

Risk Factor	Male (n = 49)	Female (n = 41)	Total (n = 90)	Percentage (%)
No Risk Factor	28	11	39	43.33%
Mother-to-Child Transmission (MTC)	7 (8.75%)	9 (11.25%)	16	17.78%
Chronic Alcoholism	11 (12.22%)	0	11	12.22%
Intravenous Drug Use (IVDU)	3 (3.33%)	0	3	3.33%
Dental Implants	2 (2.5%)	3 (3.75%)	5	5.56%
Surgeries	5 (6.25%)	3 (3.75%)	8	8.89%
Abortion	0	2 (2.22%)	2	2.22%

4. Discussion

The clinical and laboratory profiles of Chronic Hepatitis B (CHB) patients provide valuable insights into the epidemiology, disease progression, and management of the infection, which is particularly relevant in countries like Bangladesh where hepatitis B is a significant public health concern. Understanding these profiles helps physicians identify at-risk populations, determine common modes of transmission, and tailor appropriate diagnostic and therapeutic strategies. This is essential for the development of effective prevention programs and improving clinical outcomes for patients with hepatitis B infection^[9].

In our study, we observed a notable gender disparity with a higher incidence of hepatitis B in males (61.11%, $n = 55$) compared to females (38.89%, $n = 35$), consistent with previous studies from other regions, including Bangladesh^[10,11]. This male preponderance may be influenced by various factors, including higher rates of risk behavior such as intravenous drug use (IVDU) and chronic alcohol consumption, which are more common in males. Our findings also revealed that males were predominantly in the 30–40-year age group (30.00%), while females were more commonly in the 20–30-year age group (15.56%). The mean age of male patients was slightly younger (33.45 ± 9.28 years) compared to female patients (34.31 ± 13.14 years), although this difference was not statistically significant. This suggests that chronic hepatitis B predominantly affects young adults, which is a critical time for intervention and health education.

Similar studies have reported a wide age distribution, though some studies, like that of Osasona et al., reported a slightly higher prevalence in females, with a mean age of 35.7 ± 11.2 years^[12]. This disparity could be attributed to regional variations in risk factors, healthcare access, and preventive measures. However, the age distribution observed in our study aligns with findings from other studies in Bangladesh, where the most common age group for chronic hepatitis B infection is between 20 and 40 years^[13].

The identification of risk factors is crucial for understanding the epidemiology of hepatitis B and for planning preventive interventions. In our study, 43.33% of patients did not report any identifiable risk factors, which may reflect underreporting, lack of awareness, or unrecognized transmission routes. This is consistent with findings from other studies conducted in Bangladesh, where a significant proportion of chronic hepatitis B cases were linked to mother-to-child transmission (MTC), as observed in 17.78% of our cohort. This suggests that vertical transmission remains an important route of infection, particularly in countries with low vaccination coverage and high rates of perinatal transmission^[14,15]. Other known risk factors, including dental implants and surgeries, were less common in our cohort, with only 5.56% of patients reporting dental procedures and 8.89% having a history of surgery.

Alcohol use was a notable risk factor in our study, with 12.22% of male patients reporting chronic alcohol consumption. This risk factor is particularly relevant in the context of liver disease progression, as chronic alcohol use can accelerate liver damage in hepatitis B patients. The exclusive presence of alcohol-related liver disease (ALD) in male patients, along with intravenous drug use (IVDU) in 3.33% of males, further supports the notion that certain risk behaviors are more prevalent in males and contribute to the higher disease burden observed in this group. These findings are consistent with studies from other regions, which identified medical^[16] and lifestyle-related factors as important contributors to hepatitis B transmission^[17].

A significant proportion of patients in our study (47.22%) were asymptomatic and diagnosed incidentally, highlighting the silent nature of chronic hepatitis B infection. This is a critical finding, as it suggests that many individuals with hepatitis B may unknowingly transmit the virus, posing a challenge for controlling its spread. This silent presentation of hepatitis B is also commonly observed in other studies from Bangladesh, where a large number of individuals remain asymptomatic until significant liver damage

occurs^[18,19]. Among symptomatic patients, fatigue (28.89%) was the most common complaint, followed by abdominal discomfort (20.00%) and loss of appetite (18.89%). These nonspecific symptoms are frequently encountered in patients with liver disease and may not always be recognized as signs of hepatitis B infection. Jaundice was present in 17.77% of patients, which suggests that liver dysfunction had progressed in some individuals, but overall, the majority of patients were diagnosed before reaching advanced stages of liver disease.

Ultrasonography revealed hepatomegaly in 27.78% of patients, with signs of chronic liver disease, including cirrhosis and fatty liver, in 16.67% of patients. These findings are consistent with other studies, such as that of Kumar et al., who observed that a significant proportion of hepatitis B patients had hepatomegaly and other signs of liver damage, including ascites and portal hypertension^[20]. Abdominal ultrasound is an important tool for assessing liver involvement in hepatitis B and is useful for detecting complications such as cirrhosis and portal hypertension.

In terms of laboratory findings, the majority of patients in our study had normal hemoglobin levels (>12 g/dL), platelet counts (>1.5 lakh per cubic millimeter), and total bilirubin levels (<2 mg/dL), suggesting that most patients did not present with severe anemia or jaundice. However, liver enzymes (SGOT and SGPT) were elevated in 70.00% and 67.78% of patients, respectively, indicating liver injury. These elevated enzyme levels are consistent with the results reported by Kumar who found that liver enzymes were significantly higher in symptomatic patients with hepatitis B^[21]. The serological profile of our cohort revealed that all patients were HBsAg positive, confirming chronic hepatitis B infection^[22]. Anti-HBc and anti-HBe markers were positive in 85.56% of patients, suggesting ongoing viral replication and an increased risk of liver damage. The presence of HBeAg in 16.67% of patients indicates high infectivity, which underscores the need for close monitoring and antiviral therapy for these individuals to prevent transmission and progression to advanced liver disease.

Our statistical analysis revealed significant correlations between clinical features, serological markers, and biochemical parameters. Notably, HBeAg positivity showed a strong positive correlation with elevated SGOT and SGPT levels ($p < 0.001$), suggesting that active viral replication is associated with liver inflammation. Additionally, hepatomegaly was positively correlated with elevated SGOT levels ($p = 0.004$), indicating that liver enlargement is a sign of liver injury in hepatitis B patients. The negative correlation between platelet count and serum creatinine levels ($p = 0.03$) suggests that renal dysfunction may contribute to hematopoiesis disruption in chronic hepatitis B patients, a finding consistent with reports of hepatorenal syndrome in cirrhotic patients.

5. Conclusion

In conclusion, this study provides valuable insights into the clinical, biochemical, and serological profiles of patients with chronic hepatitis B in Bangladesh. The findings underscore the importance of early detection, continuous monitoring, and targeted treatment to prevent disease progression and improve patient outcomes. The high prevalence of asymptomatic cases, coupled with the significant proportion of patients diagnosed incidentally, highlights the need for widespread screening and public health initiatives to address the silent spread of hepatitis B. Moreover, the male preponderance, along with lifestyle-related risk factors such as alcohol use and intravenous drug use, suggests that targeted prevention strategies for these high-risk groups are essential. The study also emphasizes the role of serological markers and imaging techniques in the assessment of disease severity and progression, which are crucial for informing treatment decisions and reducing the burden of hepatitis B in Bangladesh.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgment

No

References

1. Al-Busafi SA, Alwassief A. Global Perspectives on the Hepatitis B Vaccination: Challenges, Achievements, and the Road to Elimination by 2030. *Vaccines*. 2024 Mar 9;12(3):288.
2. Uz-Zaman MH, Rahman A, Yasmin M. Epidemiology of hepatitis B virus infection in Bangladesh: prevalence among general population, risk groups and genotype distribution. *Genes*. 2018 Nov 8;9(11):541.
3. Pelizzaro F. HIF-1 α and VEGF as prognostic biomarkers in hepatocellular carcinoma patients treated with transarterial chemoembolization. *CIRCULATING BIOMARKERS AND CLINICAL FACTORS ASSOCIATED WITH PROGNOSIS IN PATIENTS WITH HEPATOCELLULAR CARCINOMA.*:143.
4. Osasona OG, Oguntoye OO, Arowosaye AO, Abdulkareem LO, Adewumi MO, Happi C, Folarin O. Patterns of hepatitis b virus immune escape and pol/rt mutations across clinical cohorts of patients with genotypes a, e and occult hepatitis b infection in Nigeria: A multi-centre study. *Virulence*. 2023 Dec 31;14(1):2218076.
5. Lin CL, Kao JH. Development of hepatocellular carcinoma in treated and untreated patients with chronic hepatitis B virus infection. *Clinical and Molecular Hepatology*. 2023 Jul;29(3):605.
6. Bisht D, Deo N, Sharma D, Gautam S, Patil SA. Biochemical Aspects of Leprosy. *IAL Textbook of Leprosy*. 2023 Jan 25:185.
7. Patel NH. Investigating the Unique Immunological and Virological Characteristics of Hepatitis B in Special Populations.
8. Chanda M, Biswas T, Roy MN, Sampa SR, Saha P, Sharna RJ, Islam S, Mahbub A, Rahman MA. Association of Liver Enzymes and Lipid Profile in Adults at Tertiary Level Hospital in Bangladesh. *J Natl Inst Lab Med Ref Bangladesh*. 2021 Jun;1(1):17-24.
9. Gebrelibanos MT, Karri KC, Abraha TG, Gebreyesus H, Kidane HH, Shfare MT. Prevalence of hepatitis B virus surface antigen, associated risk factors, and liver enzyme abnormalities among individuals with diabetes in Aksum town public hospitals, Tigray, northern Ethiopia. *The Pan African Medical Journal*. 2024 Sep 4;49:6.
10. Lindor KD, Bowlus CL, Boyer J, Levy C, Mayo M. Primary biliary cholangitis: 2018 practice guidance from the American Association for the Study of Liver Diseases. *Hepatology*. 2019 Jan;69(1):394-419.
11. Lindor KD, Bowlus CL, Boyer J, Levy C, Mayo M. Primary biliary cholangitis: 2018 practice guidance from the American Association for the Study of Liver Diseases. *Hepatology*. 2019 Jan;69(1):394-419.
12. Kao JH, Chen PJ, Chen DS. Recent advances in the research of hepatitis B virus-related hepatocellular carcinoma: epidemiologic and molecular biological aspects. *Advances in cancer research*. 2010 Jan 1;108:21-72.
13. Zhang L, Li MH, Cao WH, Qi TL, Lu Y, Wu SL, Hao HX, Shen G, Liu RY, Hu LP, Chang M. Negative correlation of serum hepatitis B surface antigen and hepatitis B e antigen levels with the severity of liver inflammation in treatment-naïve patients with chronic hepatitis B virus infection. *Chinese Medical Journal*. 2017 Nov 20;130(22):2697-702.
14. Islam MN, Rahaman MS, Islam S, Sakib KR, Ferdous K, Emran MR, Rahman MA. Serological profile and interpretation of different hepatitis B virus marker. *World Journal of Biology Pharmacy and Health Sciences*. 2023;14(3):213-8.
15. Mahulette SJ, Putri AF, Anfa AA, Yano Y, Setiawan J, Putri WA. Functional 3D Structure Analysis of Quasispecies Variants of Hepatitis B Virus Surface and Core Protein in Advanced Liver Disease and Chronic HBV Infection Patients in Indonesia: In Silico. *Jurnal Biota*. 2024 May 31.
16. Krajden M, McNabb G, Petric M. The laboratory diagnosis of hepatitis B virus. *The Canadian journal of infectious diseases & medical microbiology*. 2005 Mar;16(2):65.
17. Sausen DG, Shechter O, Bietsch W, Shi Z, Miller SM, Gallo ES, Dahari H, Borenstein R. Hepatitis B and Hepatitis D Viruses: A Comprehensive update with an immunological focus. *International Journal of Molecular Sciences*. 2022 Dec 15;23(24):15973.
18. Yuen MF, Chen DS, Dusheiko GM, Janssen HL, Lau DT, Locarnini SA, Peters MG, Lai CL. Hepatitis B virus infection. *Nature reviews Disease primers*. 2018 Jun 7;4(1):1-20.
19. Srinivasa Babu A, Wells ML, Teytelboym OM, Mackey JE, Miller FH, Yeh BM, Ehman RL, Venkatesh SK. Elastography in chronic liver disease: modalities, techniques, limitations, and future directions. *Radiographics*. 2016 Nov;36(7):1987-2006.

20. Wait S, Kell E, Hamid S, Muljono DH, Sollano J, Mohamed R, Shah S, Abbas Z, Johnston J, Tanwantee T, Wallace J. Hepatitis B and hepatitis C in southeast and southern Asia: challenges for governments. *The Lancet Gastroenterology & Hepatology*. 2016 Nov 1;1(3):248-55.
21. Rahaman S, Islam S, Sakib KR, Ferdous K, Islam MN, Hossen MA, Rahman MA. Comparison of ELISA & ICT Methods Determining Hepatitis B Surface in Suspected Patient Attending at Bangladesh Institute of Health Science (BIHS) General Hospital, Dhaka. *American Journal of Medical Science and Innovation*. 2023 Jul 18;2(2):31-5.
22. Banik S, Datta A, Ghosh A, Ghosh KY, Debi H. The prevalence of hepatitis B virus infection in Bangladesh: A systematic review and meta-analysis. *Epidemiology & Infection*. 2022 Jan;150:e47.