Original Research

SPECTRUM OF ACUTE HEPATITIS B- TEN YEARS EXPERIENCE AT TERTIARY CARE CENTRE OF NORTHEREN INDIA

Parveen Malhotra[1*], Rahul Siwach[2], Ankit Chahal[3], Avani Sharma[4], Abhishek Yadav[5], Pradeep Kumar[6], Chitrakshi Bhardwaj[7], Himanshu Shivanshu[8], Rajasvi Khurana[9]
[1*,2,3,4,5,6,7,8,9]Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India.
Corresponding Author: Parveen Malhotra
Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India.

ABSTRACT

Background: The spectrum of acute hepatitis B (HBV) infection, typically defined as the first six months post-exposure, ranges from asymptomatic infection and mild self-limiting illness to severe icteric hepatitis and rare fulminant liver failure. Most adults recover completely, though infants and children are more likely to develop chronic, lifelong infection. There is limited data regarding progression of acute hepatitis B virus (HBV) infection to chronic phase.

Aim: To determine the percentage of patients of acute hepatitis B who progressed to chronic hepatitis B stage.

Material and methods: It was a prospective study over a period of ten years in which total 1000 confirmed acute hepatitis B patients were enrolled but out of them complete data pertaining to 697 patients, (481 men and 216 women, 04–82 years old) was available. Hence, these patients were taken up in final analysis.

Results: Out of total of 697 acute hepatitis B patients, 658 patients resolved and became Hepatitis B surface antigen (HbsAg) and Hepatitis B virus DNA negative whereas 39 patients went into chronic phase. The progression into chronic phase was not dependent upon gender of patient, as results were comparable in both the groups. However, Serum Bilirubin, Serum amino transaminases, HbeAg and HBV DNA Quantitative levels were significantly lower in patients who progressed to chronic hepatitis stage.

Conclusions: The chronicity rate in overall pool of 697 patients was 5.59 % and on analysing for male and female group it was 5.61% and 5.55% respectively, meaning by almost comparable results in both the groups. Out of 27 males who went into chronic phase, 20 behave as inactive carriers and 7 required continued antiviral therapy. In 12 females who went into chronic phase, 10 behaved as inactive carriers and only two required antiviral therapy. The minimal representation from below 14 years of age group tell about the success and effectivity of hepatitis B vaccination (HBV) which is part of universal immunization in India for last 14 years. The majority of patients who advanced in chronic phase had mild hepatitis on comparison to patients who resolved, thus suggesting that patients with mild acute hepatitis B infection may have a higher risk of progressing to chronic infection.

Keywords: Hepatitis B, Acute Hepatitis B, Chronic Hepatitis B, Hbsag, HBV DNA Quantitative.

INTRODUCTION

Hepatitis B virus (HBV) is a DNA virus with 3200 base pairs and around 350–400 million people are chronically infected with HBV and more than 3 billion people have been exposed to HBV worldwide **[1,2]**. HBV can impact liver in many ways, ranging from acute or fulminant hepatitis to liver cirrhosis and hepatocellular carcinoma.

HBV infection is an important cause of liver cirrhosis and hepatocellular carcinoma and chronic carriers have an increased risk (15 to 40%) of developing cirrhosis, hepatic decompensation, and hepatocellular carcinoma, resulting in 1 million deaths each year [3, 4]. Acute hepatitis B is self-limited in most adult patients but 1-2% of patients progress to fulminant hepatic failure. The rate of progression from acute to chronic HBV infection is reported to be 90% in newborns and 5–10% in adults [5, 6]. HBV has 8 genotypes with a divergence of more than 8% of nucleotide sequences [7-9] and clinical presentation and routes of transmission vary between genotypes [10, 11]. The rate of chronicity of genotype A infections is reported to be higher than those of other genotypes [12-15]. It is important to understand the incidence, prevalence, and chronicity associated with HBV infection [16]. The present study was aimed to determine progression of acute hepatitis B infection into chronic phase.

www.ajmrhs.com
eISSN: 2583-7761

Date of Received: 06-04-2026
Date Acceptance: 13-04-2026
Date of Publication: 16-05-2026

MATERIAL AND METHODS

It was an epidemiology based, prospective study conducted at Medical Gastroenterology Department, PGIMS, Rohtak over a period of ten years in which

total 1000 confirmed acute hepatitis B patients were enrolled but out of them complete data pertaining to 697 patients, (481 men and 216 women, 04–82 years old) was available. Hence, these patients were taken up in final analysis. All patients who were found to be having features of acute hepatitis and confirmed to be positive for HbsAg on Enzyme linked Immunoassay test (ELISA) and HBV DNA on PCR testing were enrolled in the study. The HbsAg, HbeAg, anti-HBe, IgM Anti-Hbc was assayed by chemiluminescence immunoassay and HBV-DNA level was assayed using polymerase chain reaction. The criteria of acute HBV infection were positive for IgM anti-Hbc and HbsAg in a previously HbsAg-negative patient. The criterion of progression to chronic infection was persistence of HbsAg from the onset of the disease for more than 6 months. The percentage and clinical features of subjects who progressed and did not progress to chronic infection were determined and analysed.

OBSERVATIONS

Among six hundred and ninety- seven (697 patients), only thirty- nine (5.55%) progressed to chronic infection while the remaining 658 (94.45%) did not. Out of these 697 patients, 221(72.69%) were males and 83 (27.30%) were females. On analysis of 221 males, 201 (90.95%) resolved and 20 (9.04%) developed chronic hepatitis B whereas out of 83 females, 78 (93.97%) resolved and 5 (6.02%) patients progressed to chronic hepatitis B stage. The maximum number of patients were seen in younger age group of 20-50 yrs i.e. 215 patients (70.72%). The tendency of going in chronic phase decreased as age increased i.e. 66.66 % in 0-10 yrs of age group to 0% in above 70 yrs of age. On evaluation of risk factors, previous history of surgery, dental procedures and smoking was seen more in group of 25 patients who developed chronic hepatitis B whereas history of alcohol intake was more commonly seen in group of 279 patients who resolved. The history of tattooing and HCV co-infection was comparable in both the groups. All 304 subjects were HIV negative. On analysis of aspartate and alanine aminotransferase level, it was seen that levels of both of them were higher in resolved group of 658 patients in comparison to 39 patients who went into chronic phase of hepatitis B infection. In resolved group, AST and ALT level ranged from 120-4400 I.U. (mean of 1038 I.U.) and 208-4950 I.U. (mean of 1378 I.U.) respectively whereas in chronic phase group, AST and ALT level ranged from 102-2120 I.U. (mean of 703 I.U.) and 102-2040 I.U. (mean of 953 I.U.) respectively. In resolved group, serum bilirubin level ranged from 2.8- 36.5 mg % (mean of 9.5 mg %) whereas in chronic phase group, serum bilirubin level ranged from 1.6- 19.1 mg % (mean of 5.4 mg %). In resolved group, HBV DNA Quantitative level ranged from 10^3- 10^{10} I.U. (mean of 10^6 I.U.) whereas in chronic phase group, HBV DNA Quantitative level ranged from 10^2- 10^6 I.U. (mean of 10^4 I.U.). In resolved group of 658 patients, Hepatitis B antigen (HbeAg) was positive in 630 patients (95.74 %) whereas in chronic phase group of 39 patients, Hepatitis B antigen (HbeAg) was positive only in 15 patients (38.46 %).

Table 1- Showing Percentage of Acute Hepatitis B Patients who progressed to Chronic Stage

Total Acute Hepatitis B Patients	Patients with HBV Resolution	Patients who Progressed to Chronic Hep B Stage
697	658 (5.55%)	39 (94.45%)

Table 2- Showing Gender Distribution in Acute Hepatitis B Patients

Total Acute Hepatitis B Patients	Males	Females
697	481 (69.01%)	216 (30.99%)

Table 3- Showing Gender Distribution in Acute Hepatitis B Patients who resolved

Total Acute Hepatitis B Who Resolved	Males	Females
658	454 (68.99%)	204 (31.01%)

Table 4- Showing Gender Distribution in Acute HBV Patients who progressed to Chronic Stage

Total Acute Hepatitis B Patients who Progressed to Chronic Hepatitis B S	Males	Females
39	27 (69.23%)	12 (30.76%)

Table 5- Showing Age Distribution Both Genders in Acute HBV Patients

Total Patients (697) Males (471) Females (216)	Males who Resolved (454)	Males who Progressed to Chronic Hep B (27)	Females who Resolved (204)	Females who Progressed to Chronic Hep B (12)
0-10 yrs age	1	1	1	0
11-20 yrs age	53	9	31	1

21-30 yrs age	150	7	53	7
31-40 yrs age	108	3	46	0
41-50 yrs age	71	2	36	4
51-60 yrs age	34	2	23	0
61-70 yrs age	30	2	12	0
71-80 yrs age	5	1	2	0
81-90 yrs age	2	0	0	0

Table 6- Showing need of antiviral treatment in patients progressing to chronic stage

Total Acute HBV Patients who progressed to Chronic Hep B Stage	Males who progressed to Inactive Carrier Stage	Males who progressed to active stage requiring antiviral treatment	Females who progressed to Inactive Carrier Stage	Females who progressed to active stage requiring antiviral treatment
39 Males-27 Females-12	20	7	10	2

DISCUSSION

The present study was aimed at knowing the rate of chronicity in acutely HBV-infected patients and patients who progressed into chronic phase was analysed. All enrolled patients had no past record of HbsAg positivity but as acute hepatitis B can be asymptomatic in many cases, hence, the possibility of HBV reactivation from HbsAg-negative carrier or resolved hepatitis status in every patient cannot be ruled out. Many studies which have highlighted factors pertaining to the severity of acute hepatitis B infection and its clinical outcomes have been reported **[17-19]**. Both host and viral factors are suspected to affect the progression to chronic infection in acute hepatitis B infected patients **[20-22]** and rate of chronicity in immunocompromised conditions like HIV coinfection is high **[23-24]**. In the present study, 39 of 697 patients with acute HBV infection progressed to chronic infection. The serum Bilirubin, ALT, AST levels, HbeAg positivity and HBV DNA Quantitative levels were significantly more in resolved group than those in developed chronic hepatitis B infection and differences were statistically significant i.e. p value <0.05. It is hypothesized that the progression from acute to chronic infection appears to represent a failure of immune clearance of virus-infected cells. The accompanying acute hepatitis is typically mild and subclinical with only modest serum ALT elevations and no jaundice in patients with acute hepatitis B who progressed to chronic infection **[25]**. Many published studies have shown chronicity of acute hepatitis B in patients up to 12% **[26]** but in our study, it was less i.e. 5.55%. Several factors are associated with the increased risk of chronic HBV infection, including male gender, various causes of immune deficiency, genome variations, and genetic, hormonal and nutritional factors **[27]**. Regarding gender, male patients were more likely to become chronic carriers, although this difference was not statistically significant. This result was in alignment with other studies, which have reported a higher evolution to chronicity in male individuals **[28-30]**. Only two patients in total pool of 697 acute hepatitis B were having HCV co-infection, one each in resolved and chronic phase group. The co-infection is due to common routes of transmission of these viruses i.e. sexual, parenteral and vertical **[31]** and is associated with worse outcomes **[32]**.

CONCLUSION

More researches dealing in depth analysis of factors leading to chronicity of acute hepatitis B infection is need of hour. The adult patients, especially males with mild or subclinical acute hepatitis B infection as evidenced by low serum transaminases and bilirubin levels may have a higher possibility of progressing to chronic infection whereas those with high HBV DNA levels and Hepatitis B antigen positivity have high chances of resolution of disease. This highlights the importance of control measures, epidemiological assessments, appropriate follow-up of patients and their contacts for decreasing morbidity and mortality associated with this deadly disease.

Conflict of Interest: The authors declare that there is no conflict of interests regarding the publication of this paper.

REFERENCES

1. Lavanchy D. "Hepatitis B virus epidemiology, disease burden, treatment, arid current and emerging prevention and control measures," Journal of Viral Hepatitis, 2004; 11(2): 97- 107. https://doi.org/10.1046/j.1365-2893.2003.00487.
2. Yokosuka O, Kurosaki M, Imazeki F et al., "Management of hepatitis B: consensus of the Japan Society of Hepatology 2009," Hepatology Research, 2011; 41(1): 1-21.

https://doi.org/10.1111/j.1872-034X.2010.00739.x
3. Michitaka K, Nishiguchi S, Aoyagi Y, Hiasa Y, Tokumoto Y and Onji M. "Etiology of liver cirrhosis in Japan: a nationwide survey," Journal of Gastroenterology, 2010; 45(1): 86-94. https://doi.org/10.1007/s00535-009-0128-5
4. McMahon BJ. Natural history of chronic hepatitis B – clinical implications. Medscape J Med. 2008; 10(4): 91. PMID: 18504503; PMCID: PMC2390714.
5. Yim HJ and Lok AS. "Natural history of chronic hepatitis B virus infection: what we knew in 1981 and what we know in 2005," Hepatology, 2006; 43(S1): S173-S181. https:// doi.org/ 10.1002/hep.20956
6. Hoofnagle JH, Doo E, Liang TJ, Fleischer R and Lok ASF. "Management of hepatitis B: summary of a clinical research workshop," Hepatology, 2007; 45(4): 1056-1075. https://doi.org/10.1002/hep.21627
7. Okamoto H, Tsuda F, Sakugawa H et al., "Typing hepatitis B virus by homology in nucleotide sequence: comparison of surface antigen subtypes," Journal of General Virology, 1988; 69(10): 2575-2583. https://doi.org/10.1099/0022-1317-69-10-2575
8. Kato H, Gish RG, Bzowej N et al., "Eight genotypes (A-H) of hepatitis B virus infecting patients from San Francisco and their demographic, clinical, and virological characteristics," Journal of Medical Virology, 2004; 73(4): 516-521. https: //doi.org /10.1002 /jmv. 20120
9. Kurbanov F, Tanaka Y and Mizokami M. "Geographical and genetic diversity of the human hepatitis B virus," Hepatology Research, 2010; 40(1): 14-30. https://doi.org/10.1111/j.1872-034X.2009.00601.
10. Chu CJ, Keeffe EB, Han SH et al., "Hepatitis B virus genotypes in the United States: results of a nationwide study," Gastroenterology, 2003; 25(2): 444-451. https://doi.org/10.1016/S0016-5085(03)00895-3
11. Lin CL and Kao JH. "The clinical implications of hepatitis B virus genotype: recent advances," Journal of Gastroenterology and Hepatology, 2011; 26(1): 123-130. https://doi.org/10.1111/j.1440-1746.2010.06541.x
12. Yano K, Tamada Y, Yatsuhashi H et al., "Dynamic epidemiology of acute viral hepatitis in Japan," Intervirology, 2010; 53(1): 70-75. https://doi.org/10.1159/000252788
13. Ozasa A, Tanaka Y, Orito E et al., "Influence of genotypes and precore mutations on fulminant or chronic outcome of acute hepatitis B virus infection," Hepatology, 2006; 44(2): 326-334. https://doi.org/10.1002/hep.21249
14. Yotsuyanagi H, Okuse C, Yasuda K. et al., "Distinct geographic distributions of hepatitis B virus genotypes in patients with acute infection in Japan," Journal of Medical Virology, 2005; 77(1): 39-46. https://doi.org/10.1002/jmv.20411
15. Suzuki Y, Kobayashi M, Ikeda K et al., "Persistence of acute infection with hepatitis B virus genotype A and treatment in Japan," Journal of Medical Virology, 2005; 76(1): 33-39. https://doi.org/10.1002/jmv.20320
16. Hyams KC. Risks of chronicity following acute hepatitis B virus infection: a review. Clin Infect Dis. 1995; 20(4): 992-100. https://doi.org/10.1093/clinids/20.4.992
17. Chen Y, Michitaka K, Matsubara H, Yamamoto K, Horiike N and Onji M. "Complete genome sequence of hepatitis B virus (HBV) from a patient with fulminant hepatitis without precure and core promoter mutations: comparison with HBV from a patient with acute hepatitis infected from the same infectious source," Journal of Hepatology, 2003; 38(1): 84-90. https://doi.org/10.1016/S0168-8278(02)00325-2
18. Yamamoto K, Michitaka K, Miyata T et al., "An adult patient with hepatitis B who progressed to chronic infection," Kanzo, 2002; 43: 406-410. https:// doi. org/ 10.2957/ kanzo.43.406
19. Omata M, Ehata T, Yokosuka O, Hosoda K and Ohto M. "Mutations in the precore region of hepatitis B virus DNA in patients with fulminant and severe hepatitis," The New England Journal of Medicine, 1991; 324(24): 1699-1704. https:// doi.org/ 10.1056/ NEJM 1991 0613 3242404
20. Sato S, Suzuki K, Akahane Y et al., "Hepatitis B virus strains with mutations in the core promoter in patients with fulminant hepatitis," Annals of Internal Medicine, 1995; 122(4): 241-248.https://doi.org/10.7326/0003-4819-122-4-199502150-00001
21. Michitaka K, Horiike N, Chen Y et al., "Infectious source factors affecting the severity of sexually transmitted acute hepatitis due to hepatitis B virus genotype C," Intervirology, 2005; 48(2-3): 112-119. https://doi.org/10.1159/000081737
22. Chook JB, Ngeow YF, Khang TF, Ng KP, Tiang YP and Mohamed R. "Comparative analysis of viral genomes from acute and

chronic hepatitis B reveals novel variants associated with a lower rate of chronicity," Journal of Medical Virology, 2013; 85(3): 419424. https:// doi.org/ 10.1002/jmv.23500

23. Yanagimoto S, Yotsuyanagi H, Kikuchi Y et al., "Chronic hepatitis B in patients coinfected with human immunodeficiency virus in Japan: a retrospective multicentre analysis," Journal of Infection and Chemotherapy, 2012; 18(6): 883-890. https://doi.org/10.1007/s10156-012-0433-4
24. Pol S. "Management of HBV in immunocompromised patients," Liver International, 2013; 33(supplement 1): 182-187. https://doi.org/10.1111/liv.12055
25. Mayerat C, Mantegani A and Frei PC. "Does hepatitis B virus (HBV) genotype influence the clinical outcome of HBV infection?" Journal of Viral Hepatitis, 1999; 6(4): 299-304. https://doi.org/10.1046/j.1365-2893.1999.00174.
26. Barker LF, Murray R. Acquisition of hepatitis-associated antigen. Clinical features in young adults. JAMA. 1971; 216(12): 1970-6. https:// doi.org/ 10.1001/ jama. 1971. 03180380028006
27. Hyams KC. Risks of chronicity following acute hepatitis B virus infection: a review. Clin Infect Dis. 1995; 20(4): 992- 1000. https://doi.org/10.1093/clinids/20.4.992
28. Kim WR, Benson JT, Therneau TM, Torgerson HA, Yawn BP, Melton LJ. Changing epidemiology of hepatitis B in a U.S. community. Hepatology. 2004; 39(3): 811-6. https://doi.org/10.1002/hep.20098
29. Meffre C, Le Strat Y, Delarocque-Astagneau E, Dubois F, Antona D, Lemasson JM, et al. Prevalence of hepatitis B and hepatitis C virus infections in France in 2004: social factors are important predictors after adjusting for known risk factors. J Med Virol. 2010; 82(4): 546-55. https://doi.org/10.1002/jmv.21734
30. Taylor BC, Yuan JM, Shamliyan TA, Shaukat A, Kane RL, Wilt TJ. Clinical outcomes in adults with chronic hepatitis B in association with patient and viral characteristics: a systematic review of evidence. Hepatology. 2009; 49(5 Suppl): S85-95. https://doi.org/ 10.1002/hep.22929
31. Souza MG, Passos ADC, Machado AA, Figueiredo JFC, Esmeraldino LE. Co-infecção HIV e vírus da hepatite B: prevalência e fatores de risco. Rev Soc Bras Med Trop. 2004; 37(5): 391-5. https://doi.org/10.1590/S0037-86822004000500004
32. Sherman M. Strategies for managing coinfection with hepatitis B virus and HIV. Cleve Clin J Med. 2009; 76(Suppl 3): S30-3. https://doi.org/10.3949/ccjm.76.s3.07 DOI: http://dx.doi.org/10.12974/2312-5411.2020.07.4

How to cite this article: Parveen Malhotra, Rahul Siwach, Ankit Chahal, Avani Sharma, Abhishek Yadav, Pradeep Kumar, Chitrakshi Bhardwaj, Himanshu, Shivanshu, Rajasvi Khurana, SPECTRUM OF ACUTE HEPATITIS B- TEN YEARS EXPERIENCE AT TERTIARY CARE CENTRE OF NORTHEREN INDIA, Asian J. Med. Res. Health Sci., 2026; 4 (2):269-273.
Source of Support: Nil, Conflicts of Interest: None declared.