



Original Research Article

DISTRIBUTION OF HEPATITIS C GENOTYPES AND VIRAL LOAD PATTERN AMONG HEPATITIS C POSITIVE PATIENTS IN A TERTIARY CARE HOSPITAL IN ASSAM, INDIA

Mithu Medhi¹, Aparna Sonowal², Pranjal Sonowal³

¹Professor, Microbiology, Tinsukia Medical College, India.

²Associate Professor, Microbiology, Assam Medical College, India.

³Associate Professor, Community Medicine, Assam Medical College, India.

Received : 12/01/2026
Received in revised form : 06/03/2026
Accepted : 24/03/2026

Corresponding Author:

Dr. Pranjal Sonowal
Associate Professor, Community
Medicine, Assam Medical College,
India.
Email: drpranjals@gmail.com

DOI: 10.70034/ijmedph.2026.2.235

Source of Support: MRU of Assam
Medical College, Dibrugarh under the
Department of Health Research,
MoHFW, New Delhi

Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 1406-1410

ABSTRACT

Background: Hepatitis C virus (HCV) is a significant cause of chronic liver disease. Seven major HCV genotypes have been identified across the world. HCV genotyping reveals information regarding the viral genome's variability and therapeutic approaches. **Aim:** To determine the distribution pattern of HCV genotypes and their association with viral load among HCV infected patients in a tertiary care hospital in Assam.

Materials and Methods: 100 HCV-positive patients and not on antiviral therapy were enrolled at a Medical College in Assam. Blood samples were collected and demographic data were recorded. HCV RNA was isolated after plasma was separated. qPCR was performed to measure the viral load, and RT-PCR was performed to determine the genotype.

Results: Genotype 3 was the most prevalent (67%) HCV genotype, followed by genotype 1a (12%), genotype 6 (2%) and mix genotype (19%). Genotype 3 was found most commonly among 21-40 years age group. High viral load was observed amongst genotype 6 cases, followed by genotype 1a.

Keywords: Hepatitis C virus, HCV genotype, Viral load.

INTRODUCTION

Hepatitis C virus (HCV) is a significant cause of chronic liver disease belonging to the family of Flaviviridae in the genus Hepacivirus.^[1] The progression of Hepatitis C virus within the liver hepatocytes more often or not is quite protracted requiring timely diagnostic interventions like serology, biochemical tests and radiological examinations for proper detection.^[2] Seven major HCV genotypes have been identified across the world. Genotypes 1 and 4 are more resistant compared to genotypes 2 and 3 to therapy.^[3] Patient viral load also affects treatment duration and responses.^[4] Patients infected with genotype 1 are reported to have higher viral loads in comparison to those with genotypes 2 or 3.^[5]

Virus transmission occurs mainly between people who inject drugs; other mechanisms of HCV transmission include perinatal transmission, invasive medical and dental procedures, and rarely via sexual

intercourse. The HCV genome consists of a single strand of positive-sense ribonucleic acid (RNA) encoding a single polyprotein.^[6]

Genotype 1 is the most prevalent globally (46%) and predominates in Europe, North America, and Australia followed by genotype 3 (30%) primarily distributed in South Asia, particularly the Indian subcontinent. Genotypes 2, 4, and 6 are responsible for approximately 23% of cases, whereas genotype 5 and genotype 7 comprise less than 1%.^[7]

The estimated prevalence of HCV infection in India is approximately 0.5%–2.0%, with genotype 3 being most common.^[8] The State of Punjab in northwest India has among the highest rates of HCV infection estimated to be between 3.2% and 5.2%, predominantly genotype 3.^[9] In one epidemiological survey conducted in the Ludhiana, Moga, and Sangrur districts represented 30%, 18%, and 12% of the HCV cases, respectively.^[10]

HCV genotyping reveals information regarding the viral genome's variability, illness progression, and

therapeutic approaches.^[11] The viral load of the patient determines the duration and response to treatment.^[12] HCV treatment starts with the screening and management of alcohol use which will prevent the progression of the disease to cirrhosis.^[13] The disease progression and subsequent transmission could be prevented with early treatment. Since the early 1990s, the first pan-genotypic option has been the Interferon-alpha (IFN- α), with sustained virologic response (SVR) rates of 8%-21%.^[14]

Very few studies are there about the distribution of HCV genotype and viral load in Assam. A detailed study of genotype distribution in this region is needed towards more significant planning for HCV treatment. This study was conducted in this background to determine the distribution pattern of HCV genotypes in HCV positive individuals and their association with viral load.

Objectives of research

1. To determine the different genotypes of Hepatitis C virus among RT-PCR positive patients attending Assam Medical College and Hospital.
2. To determine the association of different genotype with viral load of the patients.

MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted in the Department of Microbiology and Multidisciplinary Research Unit (MRU), Assam Medical College, Dibrugarh, Assam, India. The study population included the Hepatitis C patients attending different outpatient and inpatient Departments of the institution. Study duration was one year from July 2023 to June 2024,

All consecutive samples confirmed as Hepatitis C positive by RT-PCR during the study period were included in the study. These confirmed positive samples were tested for determination of HCV genotype. The screening and confirmation for Hepatitis C was done under National Viral Hepatitis Control Programme (NVHCP). Genotype determination was done under Multidisciplinary Research Unit (MRU) of Assam Medical College, Dibrugarh under Department of Health Research, MoHFW, New Delhi.

Inclusion Criteria

- i) Newly diagnosed confirmed Hepatitis C Patients

Exclusion Criteria

- i) Patient whose viral load is below detection limit and
- ii) Who refused to give consent.
- iii) Undergoing treatment for HCV infection
- iv) Patient with mixed infection with both HBV and HIV

Ethical approval for the study will be obtained from Institutional Ethics Committee (Human).

Written and informed consent was obtained from the study subjects prior to enrolment. A predesigned proforma was used for collection of information from

each participant: demographics (age, sex, etc.), professional information (occupation, department etc.), clinical information, history of high risk behaviour, co-morbidities and history of close contact with Hepatitis C cases.

Sample Collection

For screening purpose, 2ml of venous blood samples was collected from the study subjects in a clot activator vial maintaining standard precautions. Serum was separated by centrifugation and samples were analyzed immediately. Again 5ml of venous blood was collected in EDTA vial from screening positive subjects, for performing RT-PCR for confirmation and viral load detection as well as genotype analysis. Plasma was separated by centrifugation and samples were processed immediately. The remaining plasma samples were aliquoted and stored at -80°C freezer for further study.

Screening and confirmation of HCV

Screening for HCV was done by 3rd generation ELISA method using Erba Lisa HCV ELISA kit (Transasia). All reactive samples were confirmed and viral load was determined by using quantitative RT-PCR. For extraction of viral RNA, QIAGEN VIRAL RNA extraction MINI KIT was used and the procedure was carried out according to the manufacturer's protocol. Hepatitis C virus RNA detection and quantification (viral load) was done by using GENO-SENS HCV REAL TIME PCR RG KIT with the Taqman principle according to the manufacturer's protocol. The HCV RNA load in plasma was expressed as log₁₀ International Units per milliliter (log₁₀ IU/ml).

HCV Genotype detection

All samples showing detectable amount of viral load are processed further for genotype analysis. Hepatitis C virus Genotyping was determined by using PathoDetect HCV genotyping RealTime PCR kit (Mylab Discovery solutions). Catalog no-PHCVGC100 (100x). This kit can detect HCV genotypes 1a, 1b, 2, 3, 4, 5 and 6 based on Taqman probes. The test is based on real time one step PCR technology. It includes reverse transcriptase (RT) reaction to convert RNA into cDNA followed by PCR for amplification of specific genotype sequences i.e. 5'UTR region (Genotype 2, 3, 4, 5 and 6) and NS5b region (Genotype 1a and 1b) using target specific probes.^[15] Here, we defined RNA copies of < 800,000 IU/mL as low viral load, while \geq 800,000 IU/mL as high viral load.^[16] Results were statistically evaluated in SPSS25 (trial version) and showed in percentages.

RESULTS

A total number of 100 HCV infected patients were recruited from different outpatient and inpatient Departments of Assam Medical College. The patients included were 85% males and 15% females, and the mean age was 25.85 \pm 8.759 years. As shown in Figure 1, genotype 3 was most prevalent (67%). In 19%

cases infection with mix genotype of 1a, 1b, 3,4 was found (19%). Genotype 1a accounted for 12% cases and followed by genotype 6 (2%).

In the present study, high viral load (100%) was observed amongst HCV genotype 6 patients followed by HCV genotype 1a (58.33%), mixed genotype 52.63% and HCV 3 (38.81%). Genotype 3 was more commonly associated with low viral load. However, there was no statistical significance ($p>0.05$) amongst genotype and viral load (Table:1).

In the present study, as shown in Table: 2, 75% of HCV 1a genotype was found among 21-40 years age group patients. Similarly, amongst 21-40 years age group genotype 3, genotype 6 and mixed genotype (1a, 1b, 3, and 4) was found to be 82.1%, 100% and 94.7% respectively. No statistical significance ($p>0.05$) was found amongst different age group and HCV genotype. Similarly, though all genotypes were more prevalent amongst male patient, no statistical significance was found amongst sex of the patients and HCV genotypes ($p>0.05$).

In the present study, (Table: 3) maximum patients with genotype HCV3 (49.5%), HCV 1a (50%), HCV 6 (50%) and mixed genotype (1a, 1b, 3 and 4) (36.8%) were IV drug users followed by HIV co infection, but there was no statistical significance ($p>0.05$) between any specific risk factors with a particular genotype.

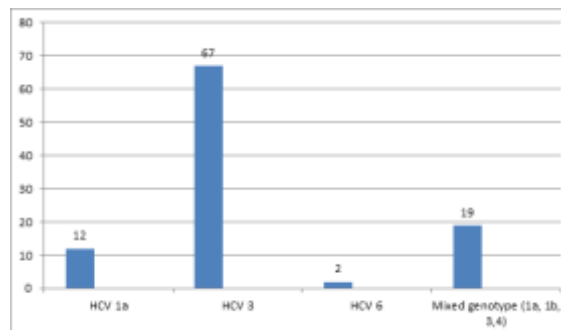


Figure 1: Distribution of HCV genotype

Table 1: Distribution of HCV Genotypes with Viral Load

Genotype	Low/Moderate Viral Load (<8,00,000 IU/ml) n (%)	High Viral Load (>8,00,000 IU/ml) n (%)	p-value
HCV 1a	5 (41.67%)	7 (58.33%)	0.188
HCV 3	41 (61.19%)	26 (38.81%)	
HCV 6	0	2 (100%)	
Mixed genotype (1a, 1b, 3, 4)	9 (47.37%)	10 (52.63%)	
Total	55	45	

Table 2: Distribution of HCV genotype according to age and sex

Age group	HCV 1a (n=12)	p-value	HCV 3 (n=67)	p-value	HCV 6 (n=2)	p-value	Mixed genotype (1a, 1b, 3, 4) (n=19)	p-value
0-20 years	2 (16.7%)	0.613	9 (13.4%)	0.819	0 (0%)	0.943	1 (5.3%)	0.542
21-40 years	9 (75%)		55 (82.1%)		2 (100%)		18 (94.7%)	
41- 60 years	1 (8.3%)		2 (3%)		0 (0%)		0 (0%)	
>60 years	0 (0%)		1 (1.5%)		0 (0%)		0 (0%)	
Sex								
Male	10 (83.3%)	1.000	57 (85.1%)	1.000	1 (50%)	0.279	17 (89.5%)	0.729
Female	2 (16.7%)		10 (14.9%)		1 (50%)		2 (10.5%)	

Table 3: Distribution of HCV Genotypes with high risk behavior

Risk Behavior/ Factors	HCV 3 (n=67)	p-value	HCV 1a (n=12)	p-value	HCV 6 (n=2)	p-value	HCV mixed genotype (1a, 1b, 3, 4) (n=19)	p-value
IV Drug User	33 (49.3%)	>0.05	6 (50%)	>0.05	1 (50%)	>0.05	7 (36.8%)	>0.05
Multiple Sexual Partner	3 (4.5%)		0		0		1 (5.3%)	
Sharing Personal Materials	7 (10.4%)		0		0		3 (15.8%)	
STD History	17 (25.4%)		0		0		3 (15.8%)	
Co infection with HIV	20 (29.9%)		2 (16.7%)		0		5 (26.3%)	
Hospital Admission (Last 2 yrs)	13 (19.4%)		0		0		2 (10.5%)	
Invasive procedure done in last 2 years	8 (11.9%)		0		0		2 (10.5%)	

DISCUSSION

A total of 100 HCV infected patients were recruited from different outpatient and inpatient Departments of Assam Medical College. The patients were mostly male (85%), and the mean age was 25.85 ± 8.759 years. In the present study there was no statistical significance was found amongst sex of the patients and HCV genotypes ($p > 0.05$). Riaz S et. al,^[11] in their study observed significant gender differences ($p < 0.005$) in prevalence of all genotypes among the patients except 2a ($p = 0.257$), with male to female ratios ranging from 1.72 for genotype 3b to 5.48 for genotype 1a. In the present study there was no statistical significance was found amongst sex of the patients and HCV viral load ($p > 0.05$). However in a study by Ali IM et al,^[17] observed high viral load among male patients.

In the present study, genotype 3 was found to be most prevalent (67%) genotype. In 19% cases infection with mix genotype of 1a, 1b, 3, 4 was found. Genotype 1a accounted for 12% cases and followed by genotype 6 (2%). Riaz S et. al,^[11] in their study observed genotype 3a as most prevalent among the patients, followed by 3b, 1a, 1b and 2a (detected in 51.5, 22.7, 9.2, 3.2 and 0.48% of the samples, respectively). Similar study by Bhattacharjee D et. al^[18] at a Tertiary Care Hospital in Kolkata, India observed Genotype 1a in 18 (27%) patients. Genotype 3 was observed in remaining 48 (73%) patients. Of these, 44 showed infection with subtype 3a (67%) while 4 had subtype 3b (6%).

In the present study, high viral load (100%) was observed amongst HCV genotype 6 patients followed by HCV genotype 1a (58.33%), mixed genotype (52.63%) and HCV 3 (38.81%). However, there was no statistical significance ($p > 0.05$) amongst genotype and viral load. Whereas Ali IM et. al,^[17] observed genotypes 4 and then genotype 1 having high viral load and HCV patients infected with genotype 2 and 3 were significantly higher viral load than average viral load among the patients.

In the present study, it was observed that 75% of HCV 1a genotype was found among 21-40 years age group patients. Similarly, amongst 21-40 years age group genotype 3, genotype 6 and mixed genotype (1a, 1b, 3, and 4) was found to be 82.1%, 100% and 94.7% respectively. No statistical significance ($p > 0.05$) was found amongst different age group and HCV genotype. Similarly, though all genotypes were more prevalent amongst male patient, no statistical significance ($p > 0.05$) was observed. In a study by Kaur D et. al,^[19] in Kolar region, Karnataka, India, observed that majority of HCV patients 25.9% was in age of 22-41 years. In the age group 22-41 years among male patients 45% were genotype 3, followed by genotype 2 (25%), genotype 4 (20%), genotype 1(5%) and mixed genotype 1 (5%). No genotype 1 and mix type-infected female patients were identified. Similar study by Pandey G et. al,^[16] observed that out of 100 clinically significant HCV

positive patients, 67 belongs to the age group of 21-40, 27 belongs to the age group of 41-60, 4 belongs to the age group of >60. Genotype 1a, 1b and 3 were more common in patients of the age groups 21 to 40 years.

In the present study, maximum patients with genotype HCV3 (49.5%), HCV 1a (50%), HCV 6 (50%) and mixed genotype (1a, 1b, 3 and 4) (36.8%) were IV drug users, but there was no statistical significance ($p > 0.05$). However, in a study by Chakravarti A,^[20] et. al observed that the mode of HCV transmission was after undergoing surgery in 26.76% patients, blood transfusion in 21.12% patients, i.v. drug abuse in 4.23% cases, dental procedure in 14.08% patients, tattooing in 8.45%, dialysis in 2.82% and unknown or not reported in 22.53% cases.

CONCLUSION

The present study highlighted genotype 3 as the most predominant genotype in this geographical region followed by genotype 1a. However, no significant association was observed between HCV genotypes and viral load in the present study. The limitation of the study includes only the cases in outpatient and inpatient departments in a tertiary care hospital whereas the chronic hepatitis in the community may show different results. These findings might help to individualize antiviral therapy, reduce side effects of antiviral therapy, economic burden and promote optimum response rates.

Acknowledgement

The authors would like to acknowledge the Multidisciplinary Research Unit (MRU) of Assam Medical College, Dibrugarh under Department of Health Research, MoHFW, New Delhi for providing a platform for conducting our research.

Funding

This research project was funded as Intramural Project by the Multidisciplinary Research Unit (MRU) of Assam Medical College, Dibrugarh under the Department of Health Research, MoHFW, New Delhi (Grant No: 2023/AMC/MRU/24, dated 16/03/2023)

Conflict of Interest: Nil

REFERENCES

1. Seeff LB, Hollinger FB, Alter HJ, Wright EC, Cain CM, Buskell ZJ, et al. Long-term mortality and morbidity of transfusion associated non-A, non-B, and type C hepatitis: A National Heart, Lung, and Blood Institute collaborative study. *Hepatology*. 2001; 33: 455–63.
2. Lauer GM, Walker BD. Hepatitis C virus infection. *N Engl J Med*. 2001; 345 (1): 41–52.
3. Bukh J, Purcell RH, Miller RH. Importance of primer selection for the detection of hepatitis C virus RNA with the polymerase chain reaction assay. *Proc Natl Acad Sci*. 1992; 89 (1):187–91.
4. Jimenez MR, Urbie SF, Guillen LP, Garza CL, Hernandez CG. Distribution of HCV genotypes and HCV RNA viral load in different geographical regions of Mexico. *Annals Hepatology*. 2010; 9: 33–39.

5. (EASL) EAfSotL EASL Clinical Practice Guidelines: management of hepatitis C virus infection. *J Hepatol.* 2011; 55: 245–64.
6. Soriano V, Mocroft A, Rockstroh J, Ledergerber B, Knysz B, et al. Spontaneous viral clearance, viral load, and genotype distribution of hepatitis C virus (HCV) in HIV-infected patients with anti-HCV antibodies in Europe. *J Infect Dis.* 2008; 198: 1337–44.
7. Panigrahi AK, Panda SK, Dixit RK, et al. Magnitude of hepatitis C virus infection in India: prevalence in healthy blood donors, acute and chronic liver diseases. *J Med Virol* 1997; 51: 167–74.
8. Das BR, Kundu B, Khandapkar R, Sahni S. Geographical distribution of hepatitis C virus genotypes in India. *Indian J Pathol Microbiol* 2002; 45:323–8. 17. Dhiman RK. Future of therapy for Hepatitis C in India: a matter of accessibility and affordability? *J Clin Exp Hepatol* 2014; 4, 85–86. 18.
9. Dhiman RK, Satsangi S, Grover GS, Puri P. Tackling the hepatitis C disease burden in Punjab, India. *J Clin Exp Hepatol* 2016; 6:224–32. 21.
10. Singh P, Kaur R, Kaur A. Frequency distribution of hepatitis C virus in different geographical regions of Punjab: retrospective study from a tertiary care centre in North India. *J Nat Sci Biol Med* 2014; 5: 56–8. 22.
11. Riaz S, Bashir MF, haider S, Rahid N. Association of genotypes with viral load and biochemical markers in HCV-infected Sindh patients. *Brazilian Journal of Microbiology* 47 (2016): 980–986.
12. European Association for the Study of The Liver, *Journal of Hepatology* 2011 55:245
13. Martin NK, Hickman M, Hutchinson SJ, et al. Combination Interventions to Prevent HCV Transmission Among People Who Inject Drugs: Modeling the Impact of Antiviral Treatment, Needle and Syringe Programs, and Opiate Substitution Therapy. *Clin Infect Dis* 2013; 57(Suppl 2):S39–45.
14. Shivkumar S, Peeling R, Jafari Y, Joseph L, Pai NP. Accuracy of rapid and point-of-care screening tests for hepatitis C: a systematic review and meta-analysis. *Ann Intern Med.* 2012 Oct 16; 157 (8): 558-66.
15. Mylab Discovery solutions PathoDetectTMHCV Genotyping PCR kit insert.
16. Pandey G, Gyawali M, Dhungana NS, Paudel R, Dahal S (2022) Distribution of Hepatitis C Genotypes and Viral Load Pattern Among Hepatitis C Positive Patients of Different Age-Group in Nepal. *J Hepatol Gastroint Dis.* 8:212.
17. Ali IM, Amirhalingam R. Distribution pattern of HCV genotypes and its significance with viral load. *Int J Med Res Health Sci.* 2013;2(3):569-576.
18. Bhattacharjee D, Mukherjee K, Chakraborti G, Ghosh R, Mandal N, Bose M. Correlation Study Between HCV Genotypes Distribution Pattern and Viral Load in a Tertiary Care Hospital in Kolkata, India. *Journal of Clinical and Diagnostic Research.* 2015 May, Vol-9(5): DC15-DC17.
19. Kaur D, Prabhakar K, Das S. Distribution of HCV genotypes and HCV RNA viral load in hepatitis infected patients of Kolar region, Karnataka, India. *Bioinformation.* 2022; 18(4): 387-391.
20. Chakravarti A, Dogra G, Verma V, Srivastava AP. Distribution pattern of HCV genotypes & its association with viral load. *Indian J Med Res* 133, March 2011, pp 326-331.