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Study on Genotypes of Hepatitis C Virus and Its Association with Demographic Profile and Viral Load in Tertiary Care Hospital

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ABSTRACT

Background: Hepatitis C virus (HCV) exhibits substantial genetic diversity, with genotype distribution varying across geographical regions. Characterizing circulating genotypes and their relationship with demographic characteristics and viral load may contribute to understanding regional HCV epidemiology and informing clinical management.
Objective: To determine the distribution of HCV genotypes and assess their associations with demographic characteristics, documented transmission risk factors, and quantitative HCV RNA levels among patients attending a tertiary care hospital in Kota, Rajasthan, India.
Methods: This laboratory-based cross-sectional study included 50 anti-HCV antibody-positive blood samples received at the Viral Research and Diagnostic Laboratory, Government Medical College and Associated Group of Hospitals, Kota, between January and May 2026. HCV RNA was quantified using a real-time polymerase chain reaction (PCR) assay, and genotyping was performed using a commercial real-time PCR-based genotyping kit. Demographic characteristics, documented risk factors, genotype distribution, and viral load measurements were analyzed using IBM SPSS Statistics version 26.0. Associations between sex and genotype were assessed using Fisher's exact test, with statistical significance set at $p < 0.05$.
Results: The study population comprised 42 males (84.0%) and 8 females (16.0%), with a mean age of 36.9 ± 14.1 years. Genotype 3 was predominant, accounting for 32 cases (64.0%), followed by genotype 1a in 13 (26.0%) and unclassified genotype 1 in 5 (10.0%). No statistically significant association was observed between sex and genotype distribution ($p = 0.635$). Transmission risk factors were undocumented in 35 patients (70.0%); intravenous drug use and previous blood transfusion were reported in 16.0% and 8.0%, respectively. Genotype 3 exhibited the highest mean HCV RNA level (7.71×10^4 IU/mL), compared with genotype 1a (1.57×10^4 IU/mL) and genotype 1 (2.08×10^4 IU/mL). These differences were descriptive, as no inferential comparison of viral loads was reported.
Conclusion: Genotype 3 was the predominant HCV genotype in this hospital-based cohort, with a marked male predominance and a high proportion of undocumented transmission risk factors. Larger studies are warranted to validate regional genotype patterns and clarify associations between genotype and viral load.

Keywords: Hepatitis C Virus (HCV); Genotype 3; Real-Time PCR; Quantitative Viral Load; Intravenous Drug Use (IDU); Micro-epidemiology; Kota; Direct-Acting Antivirals (DAAs)

INTRODUCTION

Hepatitis C virus (HCV) infection is a blood-borne viral disease caused by the Hepatitis C virus. It belongs to the *Flaviviridae* family and is associated with chronic liver disorders **[1]**. HCV represents a major public health challenge because the infection is frequently chronic and further complicated by a high mutation rate and the absence of an effective vaccine. Early stages of the infection are often missed because antibodies develop only after one and a half months post-infection, and serological tests for anti-HCV antibodies may return negative results during the initial window period before seroconversion **[2,3]**.

The HCV RNA genome is a single-stranded, positive-sense RNA of approximately 9,600 nucleotide bases. The HCV genome contains a single long open reading frame (3006–3037 codons) flanked by untranslated regions (UTRs) at the 5' and 3' ends **[4]**. HCV demonstrates high genetic heterogeneity; the International Committee on Taxonomy of Viruses (ICTV) has identified eight genotypes (numbered 1 through 8), encompassing around 90 subtypes and millions of quasispecies. The prevalence and distribution of HCV genotypes differ substantially between developed and developing nations **[5]**.

Globally, Genotype 1 is the most prevalent variant, accounting for nearly 49% of cases, followed by Genotype 3 (18%), Genotype 4 (17%), and Genotypes 5 and 6 (~5% each). Genotypes 4, 5, 6, and 7 are endemic to specific geographical regions, whereas the remaining types are distributed worldwide **[6]**. In India, Genotype 3 is the predominant circulating strain, followed by Genotype 1. Regional distribution in India suggests that Genotype 3 dominates in the eastern, western, and northern regions, whereas Genotype 1 predominated in Southern India **[1]**. Understanding the genotypic landscape in a specific region facilitates tailored treatment planning and offers valuable clinical insights into the risk of HCV-related liver disease

Objective:

The purpose of this study was to determine the genotypic distribution of HCV and evaluate its association with demographic profiles and viral load levels among patients attending the Outpatient Department (OPD) and inpatient wards at Govt. Medical College & Associated Group of Hospitals, Kota, Rajasthan.

MATERIALS & METHODS

This laboratory-based cross-sectional study was conducted in the Viral Research & Diagnostic Laboratory (VRDL), Department of Microbiology, Govt. Medical College & Associated Group of Hospitals, Kota, from January 2026 to May 2026. The study was approved by the Institutional Ethics Committee of Govt. Medical College, Kota.

A total of 50 samples (Blood in EDTA vial) were included in this study which were received in VRDL Lab after tested positive for Hepatitis C virus antibody in attached group of hospitals, Govt. Medical College, Kota. After receiving, RNA was extracted from the sample by using TRUPCR Viral RNA extraction kit as per manufacturer's instructions. Extracted RNA was subjected first to TRUPCR HCV RNA viral load kit. TRUPCR HCV RNA Viral Load Kit is one step in vitro nucleic acid amplification assay for the quantitative detection of HCV viral RNA from HCV infected individual specimens using Real Time PCR System. TRUPCR HCV Viral Load Kit primers and probes target the highly conserved regions of the HCV genome.

After viral load testing, extracted sample was tested for HCV genotype by TRUPCR HCV Genotyping Kit. TRUPCR HCV Genotyping Kit is an in vitro Real-time PCR assay for the qualitative detection of Hepatitis C Viruses (HCV) and differentiation of genotypes 1, 1a, 2 (2a/2b),

3, 4, 5a and 6 RNA in human serum or plasma (EDTA) from HCV infected individual specimens.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics, version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized using means and standard deviations (SD), medians, and ranges, while categorical variables were presented as frequencies and percentages. The association between gender and HCV genotype was assessed using Pearson's chi-square test or Fisher's exact test as applicable. A two-sided *p*-value of less than 0.05 was considered statistically significant. Viral load measurements were summarized descriptively according to HCV genotype; no inferential statistical comparison of viral load between genotypes was reported.

RESULT

A total of 50 patients with laboratory-confirmed Hepatitis C Virus (HCV) infection were enrolled and evaluated. The study cohort demonstrated a distinctive male predominance, with 42 male patients (84.0%) and 8 female patients (16.0%), yielding a male-to-female ratio of 5.25:1. The overall mean age of the cohort was 36.9 ± 14.1 years, spanning a wide age distribution from 2 to 75 years. (Table 1)

Table-1 Baseline Demographics and Clinical Characteristics of the Study Cohort (N=50)

Parameter	Baseline Value (n=50)	Percentage/Range
Gender		
Male	42	84 %
Female	8	16 %
Age (Years)		
Mean ± SD	36.9 ± 14.1	-
Range	-	2 – 75

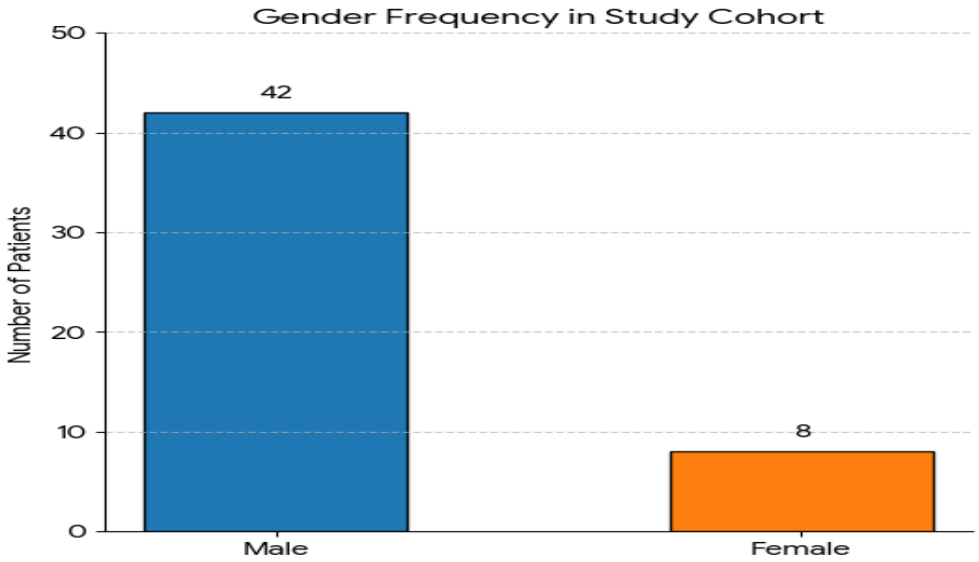

Figure 1: Gender frequency in the study.

Genotypic stratification revealed that Genotype 3 was the most prevalent viral variant, identified in 32 patients (64.0%). This was followed by Genotype 1a in 13 patients (26.0%), and unclassified Genotype 1 in 5 patients (10.0%). Across all identified genotypes, male patients consistently comprised the vast majority of cases.

To investigate if viral genotype selection was influenced by patient gender, cross-tabulation and cross-cohort distribution testing were executed. Males comprised 81.3% (n=26/32) of the Genotype 3 group,

92.3% (n=12/13) of the Genotype 1a group, and 80.0% (n=4/5) of the unclassified Genotype 1 group. Because multiple clinical sub-strata contained low expected values (n<5), an exact inferential testing framework was required. Fisher's Exact Test confirmed no statistically significant association between biological sex and specific HCV genotype distribution (chi-square equivalent, p=0.635^{NS}) proving that male dominance remains homogeneous across all viral lineages. (Table-2)

Table-2 Cross-Tabulation and Inferential Statistics of HCV Genotypes by Gender

Circulating HCV Genotype	Combined Cohort Total (n=50, 100.0%)	Male Patient Subgroup (n=42, 84.0%)	Female Patient Subgroup (n =8, 16.0%)	Intragroup Genotype Male-to-Female Ratio	Exact Test Significance (p-value)
Genotype 3	32 (64 %)	26 (81.25%)	6 (18.75%)	4.33: 1	
Genotype 1a	13 (26%)	12 (92.31%)	1 (7.69%)	12.00: 1	p=0.635NS
Genotype 1 (Unclassified)	5 (10%)	4 (80.00%)	1 (20.00%)	4.00: 1	
Total Stratified Count	50 (100.0%)	42 (100.0%)	8 (100.0%)	5.25: 1	

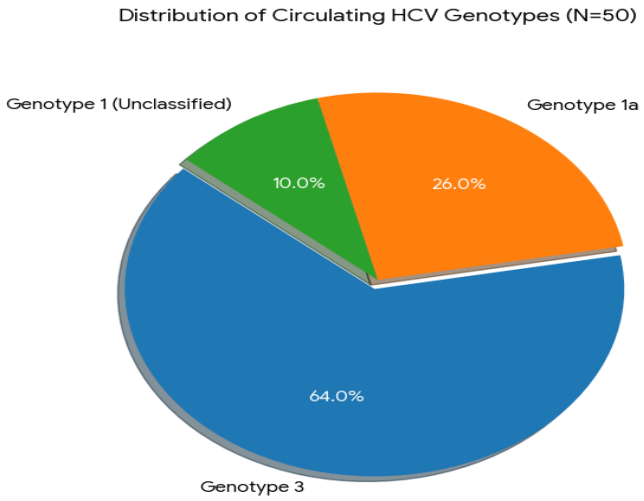

Figure 2: Distribution of circulating HCV genotype.

Identifiable high-risk epidemiological risk factors and medical comorbidities were documented in 15 patients (30.0%; 95% CI: 18.1%–45.4%), whereas the transmission vector remained idiopathic or undocumented in 35 patients (70.0%; 95% CI: 55.4%–82.1%).

Among those presenting with defined transmission vectors, a history of Intravenous Drug Use (IDU) was the leading exposure mode, reported exclusively by 8 patients (16.0%). Prior exposure to Blood Transfusions was identified as the secondary risk vector in 4 patients (8.0%). Specialized cohorts including High-Risk Sexual Behavior, Thalassemic / Hemophilic Variants, and Chronic Hemodialysis Dependence each accounted for 2.0% (n = 1) of total cases. (Table-3)

Table 3: Descriptive Epidemiology and Calculated 95% Confidence Intervals

Documented Epidemiological Vector	Patient Count (n = 50)	Cohort Percentage (%)	95% Confidence Interval (Lower – Upper CI)
Intravenous Drug User (IDU) History	8	16%	7.2% – 29.1%
Prior Blood Transfusion	4	8 %	2.2% – 19.2%
High-Risk Sexual	1	2%	0.1% – 10.5%

Behavior			
Thalassemic / Hemophilic Variant	1	2%	0.1% – 10.5%
Chronic Hemodialysis Dependent	1	2%	0.1% – 10.5%
Idiopathic / Not Documented	35	70 %	55.4% – 82.1%
Total	**50**	**100 %**	-

Baseline quantitative serum HCV RNA viral loads exhibited profound biological variation across the study sample, fluctuating from a minimum of 8.23 IU/mL to a peak of 5.90×10^5 IU/mL. When stratified by viral strain, patients infected with Genotype 3 manifested the highest replication kinetics, with a mean viral load of 7.71×10^4 IU/mL (median: 1.23×10^4 IU/mL; range: 8.23 to 5.90×10^5 IU/mL). In contrast, the Genotype 1a subgroup demonstrated lower baseline replication markers, presenting a mean viral load of 1.57×10^4 IU/mL (median: 1.52×10^3 IU/mL; range: 3.06×10^2 to 9.65×10^4 IU/mL). Patients with unspecified Genotype 1 infections presented intermediate replication profiles, with a mean viral load of 2.08×10^4 IU/mL (median: 3.14×10^3 IU/mL; range: 2.27×10^2 to 8.79×10^4 IU/mL). (Table-4)

Table- 4 Cross-Tabulation of HCV Genotypes and Quantitative Viral Loads

Genotype Profile	**Patient Count (n)**	**Percentage (%)**	**Mean Viral load (IU/ml)**	**Median Viral Load Viral load (IU/ml)**	**Range of Viral Load Concen. (IU/mL)**
Genotype 3	32	64 %	7.71 x 10^4 IU/ml	1.23 x 10^4 IU/ml	8.23 IU/ml to 5.90 x 10^5 IU/ml
Genotype 1a	13	26%	1.57x 10^4 IU/ml	1.52 x 10^3IU/ml	3.06 x 10^2 IU/ml to 9.65 x 10^4 IU/ml
Genotype 1	5	10%	2.08 x 10^4 IU/ml	3.14 x 10^3 IU/ml	2027 x 10^2 IU/ml to 8.79 x 10^4 IU/ml
Total Cohort	**50**	**100 %**	**5.55 x 10^4 IU/ml**	**1.00 x 10^4 IU/ml**	**8.23 IU/ml to 5.90 x 10^5 IU/ml**

DISCUSSION

The present study provides a critical cross-sectional analysis of the epidemiological profiles, genotypic stratification, and quantitative viral kinetics among 50 patients diagnosed with chronic Hepatitis C Virus (HCV) infection within an Indian tertiary care framework. Our baseline cohort demonstrated a striking male predominance (84.0%), a distinct overrepresentation of HCV Genotype 3 (64.0%), and a substantial portion of infections with undocumented or idiopathic transmission vectors (70.0%). Furthermore, our findings underscore a significant biological variance in baseline quantitative serum HCV RNA viral loads across different viral strains, offering essential parameters regarding the real-world performance and clinical landscape of regional HCV micro-epidemiology.

A defining feature of our study cohort is the profound male-to-female ratio of 5.25:1, with men constituting 84.0% of the evaluated patients. This distinct male skewness strongly matches contemporary baseline data reported by major epidemiological surveillance teams in India. A recent study by Mahesh Chandra et al. [7] showed a significantly higher prevalence of HCV in males (0.88%) as compared to females (0.29). This demographic pattern is primarily attributed to social mobility, broader occupational exposures, and a higher participation rate in screening behaviors compared to female cohorts rather than an underlying biological susceptibility.

Our inferential analysis using Fisher's Exact Test (p=0.635) demonstrated that this male dominance remains completely homogeneous across all identified viral lineages (Genotypes 3, 1a, and unclassified Genotype 1). This lack of statistical association implies that viral genotype selection operates independently of host biological sex within this Indian population subset. This observation is strongly corroborated by the work of Medhi et al. **[8]**, whose baseline evaluations of demographic distributions confirmed no distinct relationship between patient gender and underlying genotypic mutations ($p > 0.05$).

The overall mean age of our cohort was 36.9±14.1 years, capturing a predominantly young to middle-aged adult population at peak economic productivity. This age profile precisely mirrors national-level observations where active, screen-detected viral replication typically peaks between the third and fourth decades of life. However, our expansive age range spanning from 2 to 75 years highlights that active transmission and diagnostic presentation persist across the full lifecycle, demanding flexible, multi-generational diagnostic entry points.

Genotypic stratification revealed that Genotype 3 was the most prevalent variant, identified in 64.0% of the study sample, followed by Genotype 1a (26.0%) and unclassified Genotype 1 (10.0%). This finding mirrors the molecular epidemiology of HCV across the Indian subcontinent. An open-label prospective study from J.N. Medical College, Aligarh, Uttar Pradesh by Muhammed Shahanas S et al. **[9]** reported a highly similar Genotype 3 dominance of 66.0% with Genotype 1a serving as the secondary variant (24%). This confirms that Genotype 3 remains firmly hyper-endemic across both northern and eastern administrative corridors of India.

From a clinical standpoint, the dominance of Genotype 3 is notable. Indian isolates of Genotype 3 are frequently linked to faster progression of hepatic steatosis, accelerated liver fibrosis, and an elevated lifetime relative risk of developing hepatocellular carcinoma (HCC) compared to Genotype 1. Even with pangenotypic Direct-Acting Antiviral (DAA) therapies like Sofosbuvir/Velpatasvir widely available under Indian national public health initiatives, Genotype 3 remains a primary cause of late-stage treatment failures and post-SVR cirrhosis management. Conversely, mapping the Genotype 1a and unclassified Genotype 1 subgroups (accounting for 36.0% collectively in our cohort) remains critical. This is because non-pangenotypic drug regimes can be severely compromised by the baseline resistance-associated substitutions (RASs) historically observed in Genotype 1 strains in urban environments.

Our epidemiological assessment revealed a highly concerning trend: the transmission vector remained entirely idiopathic or undocumented in the vast majority of cases (70.0%; 95% CI: 55.4%–82.1%). This high percentage of unknown exposure highlights a significant gap in patient recall, social stigma surrounding risk-factor disclosure, or the silent propagation of HCV via unrecognized community routes. This observation is highly consistent with studies by Kamran B Lankarani et al. **[10]** and Javed Akhtar et al. **[11]**. In developing healthcare environments, where idiopathic transmission is frequently tied to minor medical or cosmetic interventions, such as unscreened dental procedures, informal therapeutic injections, ritual scarification, or commercial barbering using unsterilized equipment.

Among the 30.0% of patients with identifiable risk profiles, a history of Intravenous Drug Use (IDU) was the leading exposure mode, reported exclusively by 16.0% of the total cohort. This finding is identical to global patterns established by the World Health Organization (WHO) **[12]** and Nelson et al. **[13]** which identify injection drug use as the primary engine driving new HCV infections worldwide. This emphasizes the critical need for harm-reduction programs, localized

needle-exchange initiatives, and targeted addiction counselling.

Prior blood transfusions represented the secondary identified vector (8.0%), illustrating that while modern centralized blood banking protocols have drastically reduced transfusion-transmitted infections, residual risks from older transfusions or unregulated centers persist, mirroring observations made by Saurabh Chhotalal Gamit et al. **[14]**

A compelling biological feature observed in this study is the profound variation in baseline quantitative serum HCV RNA viral loads, fluctuating from a minimal baseline of 8.23 IU/mL to a peak replication ceiling of 5.90x 10^5 IU/mL. When parsed by specific viral strains, Genotype 3 demonstrated substantially higher replication kinetics, yielding a mean viral load of $7.71x10^4$ IU/mL and a median of $1.23x10^4$ IU/mL. In contrast, Genotype 1a exhibited a markedly lower baseline mean replication profile (1.57 x 10^4 IU/mL).

These differences indicate that Genotype 3 variants within this population may possess superior in-vivo replicative fitness or a greater capacity to evade early host innate immune pressures. This specific observation of higher baseline viral loads in Genotype 3 relative to Genotype 1 variants runs parallel to the virological evaluations published by Devinder Kaur et al. **[15]** who noted that regional Genotype 3 isolates often exhibit aggressive replication baselines.

CONCLUSION

This study successfully mapped the epidemiological blueprint of Hepatitis C Virus infections at a tertiary care center in Kota, Rajasthan. Genotype 3 stands out as the heavily dominant circulating strain, impacting young adult males at a disproportionate rate. The distinct biological variations noticed in baseline viral loads across different genotypes - coupled with a prominent risk factor signature in intravenous drug use—emphasizes the urgent need for universal, mandatory molecular genotyping. Implementing routine viral load quantifications will vastly optimize direct-acting antiviral (DAA) therapeutic protocols, reduce long-term chronic liver sequelae, and significantly advance localized viral eradication strategies in the Hadoti region.

Declaration by Authors

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Conflict of Interest: The authors declare no conflict of interest.

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