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Hepatitis G Viremia as Predictor of Liver Disease Prognosis in High-Risk Populations and Chronic Liver Patients

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Abstract

Infection with the hepatitis G virus (HGV) is prevalent among high-risk groups such as hemophilia, thalassemia, healthcare workers, voluntary blood donors, and chronic liver patients worldwide. However, there is limited information regarding the clinical significance of HGV and its impact on liver diseases. To investigate the potential impact of HGV RNA on liver inflammation and damage by examining elevated liver function enzymes in high-risk groups and chronic liver patients. The study was conducted to assess HGV RNA and liver enzymes in individuals with chronic liver disease (previously infected with HBV and HCV), healthcare workers, blood donors, haemophilia and thalassemia patients. 211 individuals were found positive (RT-PCR). Liver function enzyme elevations were compared between high-risk groups and chronic liver patients with or without hepatitis viral RNA using RT-PCR. Another study tested 221 participants for HGV-RNA using RT-PCR. Of these, 97 (43.9%) were positive while 124 (56.1%) tested negative. Based on the analysis of liver enzymes, the study finding suggests the presence of HGV-RNA does not seem to have a significant impact on liver inflammation or damage. This implies that HGV may not pose a silent threat to liver health in high-risk populations and chronic liver patients. Thus, our research concludes a less meaningful association between hepatitis infection and liver function enzymes in these populations.

Keywords: Chronic liver disease; Hepatitis G viral infection; Liver function test; Liver inflammation and damage of Viremia.

Introduction

Hepatitis G virus (HGV/ GBV-C) was first discovered in 1995 and is a common virus found circulating in human populations worldwide(1). Its infection is usually asymptomatic; the prevalence rates of HGV-RNA in different groups include healthy blood donors, hemophilic/ thalassemic patients, and healthcare workers(2, 3).

Studies on Liver function enzyme (LFE) levels such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), serum albumin and total bilirubin in individuals testing positive for HGV-RNA. Some studies found no significant differences in mean LFE levels between HGV-RNA positive and negative individuals in healthy blood donors or high-risk groups such as hemophilics(4, 5). Yet other studies reported elevated AST and ALT levels associated with HGV RNA positivity, particularly in chronic liver disease patients(6).
Studies show that HGV can occur as either a single infection or coincident with other infections like HCV or HIV. The role of HGV in the progression or chronicity of hepatitis B or C infections remains unclear. HGV co-infection may contribute to worse outcomes in some chronic viral hepatitis cases, but more research is needed(3).

Previous studies have found HGV infection rate differences between geographic regions. HGV viremia, or the presence in the blood, was found to be higher in healthy blood donors in Africa (17.2%) compared to Asia (3.4%) and Europe (4.5%). This suggests that HGV exposure and transmission may be more common on the African continent than in other parts of the world like Asia and Europe(2).

A study conducted in Iraq found that 19 out of 90 chronic liver patients (21.11%) tested positive for HGV infection. This suggests that around one-fifth of chronic liver cases in Iraq may be exacerbated by HGV exposure(7).

A study found that 10.4% of thalassemia patients showed evidence of HGV viremia when tested using ELISA. However, when a more sensitive technique, RT-PCR was tested in the same patient group, a higher prevalence of 18.2% was found. In Iraq, 14.8% of thalassemia patients and 3.3% of healthcare workers were found to have HGV infection in Diyala Province(8, 9). 9.8% of HBV cases also have HGV infection(10). HGV RNA positive i.e., 5.0% of HBsAg-positive patients and 1.25% of Anti-HCV-positive patients(11), HGV seropositivity rate was 2.6% in thalassemic

patients, 0% in hemodialysis patients, 0.98% in normal population, and 1.6% in blood donors of Kirkuk city(12).

The frequency of HGV infection varies among different populations. In anaemic patients with multiple transfusions, the frequency is 18%. In haemophiliacs, the frequency is 26% in Spain and 12.5% in the USA. In North-eastern Thai blood donors co-infected with HBV, the frequency of HGV infection is 10%. In rural China, 12.9% of the population was found to have anti-HGV antibodies(9). In Khurassan, Iran, 5% of hemophiliacs were found to have HGV-RNA(5).

More data is needed to better understand if HGV acts as a silent infection or can contribute to ongoing liver damage over time in high-risk groups such as healthy blood donors, haemophiliac patients, thalassemia patients, health care workers, and chronic liver patients. HGV prevalence in healthy individuals and chronically infected hepatitis patients was not properly studied(1). Our study compares LFE levels in chronic liver patients, high-risk groups, and healthy populations based on their HGV RNA status. This may help clarify if the presence of HGV independently impacts liver health and function.

Materials and methods

Patients

An experimental research design was employed to evaluate HGV-RNA occurrence using RT-PCR and liver function enzyme levels in 211 participants. The participants included 44 (19.9%) chronic liver infected (confirmed previously infected with HBV and HCV), 22 (10.0%) blood donors, 45 (20.4%) healthcare workers, 44 (19.9%) hemophilia patients, 22 (10.0%) individuals who received blood, and 44 (19.9%) thalassemia patients. Volunteers were from various hospitals and blood centres, including Al-Karamah Teaching Hospital, Al-Ramadi Teaching Hospital, Al-Ramadi Teaching Hospital for Women and Children, Anbar -Blood Transfusion Centre, Gastroenterology and Haematology Teaching Hospital, and Paediatrics Teaching Hospital, during the period between September 22, 2022, and April 12, 2023. The participant's blood samples were used to determine the HGV RNA presence using a multiplex nested RT-PCR procedure. Liver function enzyme (LFE) tests, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, alkaline phosphatase (AP), and serum albumin, were also conducted. The levels of LFEs were compared between high-risk groups and chronic liver patients with or without GBV-

C/HGV RNA. Family consents were received from all participants, and the Approval Committee of Anbar Medical Colleges approved the study.

Statistical analysis

Studies were performed using SPSS version 26.0 (IBM, Armonk, NY) and Graph Pad Prism. The mean, SD, and P values of liver function enzymes in participants with or without GBV-C/HGV RNA to compare separate t-test. The liver function enzyme values in various age groups with or without HGV RNA were compared using one-way ANOVA.

Results

In the current study, out of the initial 307 participants, 86 individuals were excluded. The reasons for exclusion included 20 participants with malignant disease, 26 volunteers of age >73 years, 10 pregnant women, and 30 participants who did not take part. 221 participants fulfilled the enrolment criteria and participated. Among these volunteers were 44 chronic liver patients, 44 haemophilia, 44 thalassemia patients, 45 healthcare workers, 22 donors, and 22 blood receivers (**Figure 1**).

1. Means liver function enzymes in HGV PCR positive and negative participants

The RT-PCR approach revealed that 97 of the 221 participants tested positive for HGV RNA, whereas the remaining 124 tested negative.

1.1 Means of ALT, AST in HGV PCR positive and negative participants

The average AST level was 39.46 U/L with a standard deviation of 19.095 U/L in volunteers of HGV RNA-negative, while it was 42.48 U/L with a standard deviation of 22.365 U/L in HGV-positive participants. Similarly, the mean ALT was 25.23 U/L (SD = 16.322 U/L) in HGV RNA-negative individuals and 28.78 U/L (SD = 23.081 U/L) in HGV RNA-positive individuals.

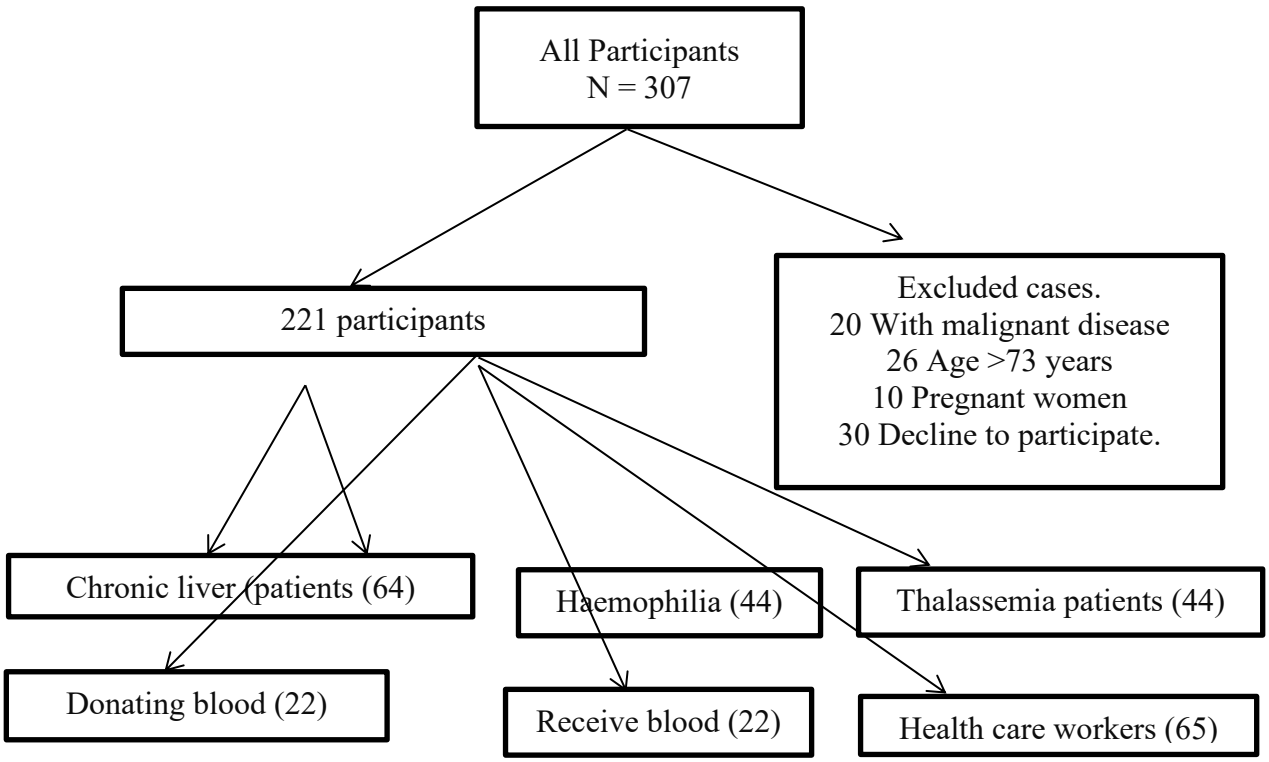

Figure 1. Flowchart of 307 participants to detect Hepatitis G virus.

However, these differences in mean AST and ALT levels were not statistically significant. In simpler terms, no significant difference in AST and ALT values between individuals who tested positive or negative for HGV RNA. Therefore, based on this data (**Table 1, 2**), HGV infection status did not have a major impact on liver enzyme levels, not significantly affected by HGV infection (**Figure 2).**

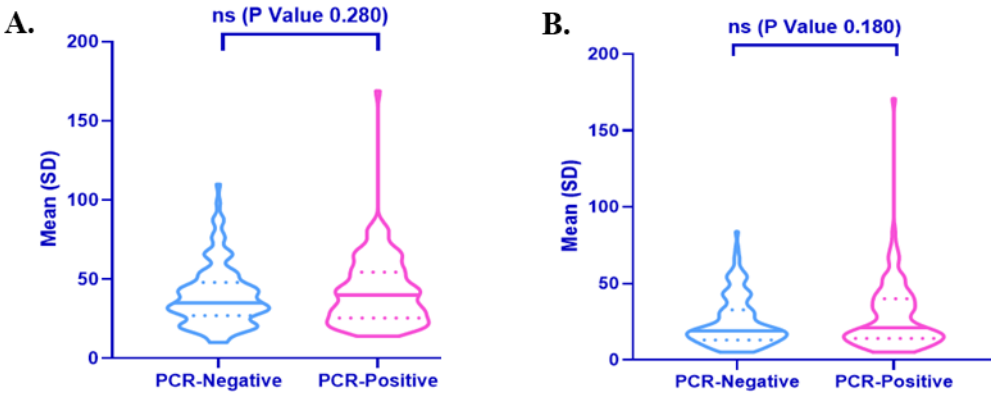

Figure 2. Means of AST (A) and ALT (B) in HGV PCR positive and negative participants.

Table 1: AST mean in PCR positive and negative cases:

	PCR-negative	**PCR-positive**
Number of values	124	97
Minimum	10	14
25%	27	25.50
Median	35	40
75% percentile	48	54.50
Maximum	110	169
Range	100	155
Mean	39.46	42.48
Std. deviation	19.10	22.36
Std. Error of mean	1.715	2.271
Coeff. Of variation	48.39%	52.64%
Sum	4893	4121

Table 2: ALT mean in PCR positive and negative cases:

	PCR-negative	**PCR-positive**
Number of values	124	97
Minimum	5	5
25%	13	14
Median	19	21
75% percentile	32.75	40
Maximum	84	171
Range	79	166
Mean	25.23	28.78
Std. deviation	16.32	23.08
Std. Error of mean	1.466	2.344
Coeff. Of variation	64.71%	80.19%
Sum	3128	2792

1.2 Means of serum albumin, ALP in HGV PCR positive and negative participants

Serum albumin and alkaline phosphatase (ALP) levels were compared for volunteers who tested negative for HGV RNA and those who tested positive. The serum albumin (average) was 4.006 g/dL (with a standard deviation of 0.7035) in the HGV RNA negative group and 3.851 g/dL (with a standard deviation of 0.7915) in the HGV RNA positive group. Similarly, the average ALP level was 284.17 U/L (with a standard deviation of 146.096) among HGV RNA-negative participants, and 279.18 U/L (with a standard deviation of 155.886) among HGV RNA-positive participants. However, The differences were not found to be statistically significant in serum albumin or ALP levels in HGV RNA-positive or negative participants (**Figure 3, Table 3, 4).**

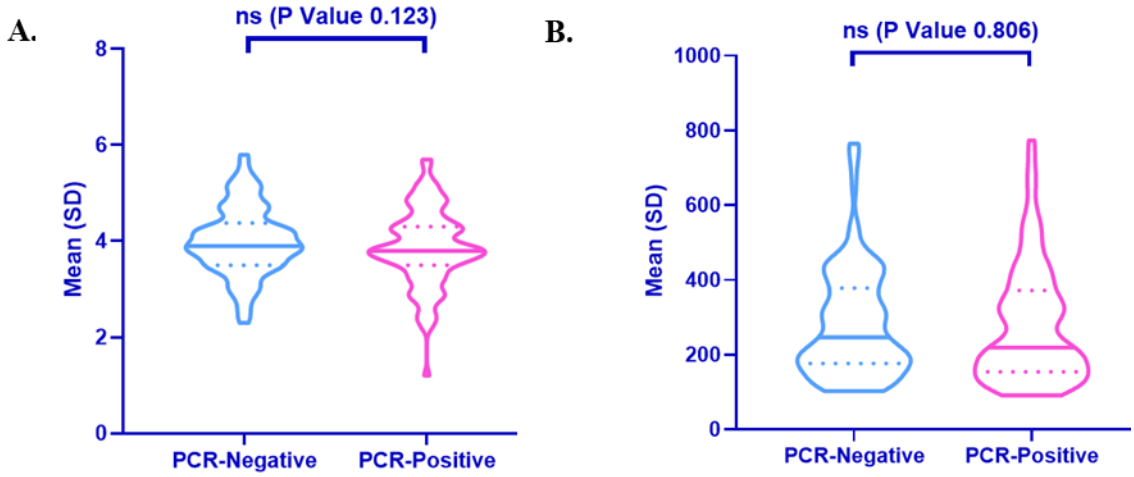

Figure 3. Mean serum albumin, and ALP in HGV PCR positive and negative participants.

Table 3: Mean serum albumin in PCR positive and negative cases:

	PCR-negative	**PCR-positive**
Number of values	124	97
Minimum	2.3	1.2
25%	3.5	3.5
Median	3.9	3.8
75% percentile	4.375	4.3
Maximum	5.8	5.7
Range	3.5	4.5
Mean	4.006	3.85
Std. deviation	0.704	0.79
Std. Error of mean	0.063	0.08
Coeff. Of variation	17.56%	20.56%

Sum	496.8	373.5

Table 4: ALP mean in PCR positive and negative cases:

	PCR-negative	**PCR-positive**
Number of values	124	97
Minimum	102	91
25%	176	154
Median	246.5	219
75% percentile	378	372
Maximum	765	774
Range	663	683
Mean	284.2	279.2
Std. deviation	146.1	155.9
Std. Error of mean	13.12	15.83
Coeff. Of variation	51.41%	55.84%
Sum	35237	27080

1.3 Means of serum albumin, ALP in HGV PCR positive and negative participants

The study compared the average total bilirubin levels between HGV-RNA-negative and positive participants. In the group who were HGV negative, the bilirubin (average) was 1.878 mg/dL with a standard deviation of 1.0619. On the other hand, in the HGV-positive group, was 1.958 mg/dL with a standard deviation of 1.1054. However, the difference in mean total bilirubin levels between HGV-positive and HGV-negative participants is statistically non-significant (**Figure 4, Table 5).**

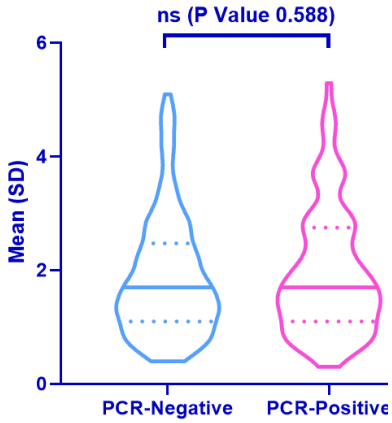

Figure 4. Mean of Total bilirubin in HGV participants (PCR positive vs negative).

Table 5: Bilirubin in HGV PCR positive and negative participants:

	PCR-negative	**PCR-positive**
Number of values	124	97
Minimum	0.4	0.3
25%	1.1	1.1
Median	1.7	1.7
75% percentile	2.475	2.750
Maximum	5.1	5.3
Range	4.7	5
Mean	1.878	1.958
Std. deviation	1.062	1.105
Std. Error of mean	0.095	0.112
Coeff. Of variation	56.54%	56.46%
Sum	232.9	189.9

2. Means liver function enzymes in HGV PCR positive and negative participants

221 individuals underwent RT-PCR testing to detect HGV RNA. Out of the 221 participants tested, 97 of them (43.9%) tested positive for HGV RNA, while the remaining 124 participants (56.1%) tested negative. The volunteers were segregated into different age groups: 47 individuals (21.3% of the total) were aged 17 years or younger, 86 individuals (38.9% of the total) were between 18 and 36 years old, 77 individuals (34.8% of the total) were between 37 and 54 years old, and 11 individuals (5.0% of the total) were 55 years old or older.

2.1 Means of ALT and AST in HGV positive and negative cases according to age groups

The average ALT levels in individuals aged 17 or below who tested negative for HGV RNA were 24.69 U/L, with a standard deviation of 19.100 U/L. However, HGV-RNA positive had a higher average ALT level of 38.00 U/L, with a standard deviation of 39.370 U/L. For individuals between the ages of 18 and 36, the mean ALT levels were 24.22 U/L for HGV RNA negatives, with a standard deviation of 15.608 U/L, compared to 27.35 U/L for HGV RNA positives, with a standard deviation of 19.285 U/L.

In the age group of 37-54 years, the ALT between HGV RNA negative participants (26.80 U/L, standard deviation of 15.389 U/L) and HGV RNA positive participants (27.58 U/L, standard

deviation of 15.311 U/L), almost same levels were detected. Lastly, for adults aged 55 years and above, the ALT was lower in HGV RNA-positive participants (17.17 U/L, standard deviation of 12.123 U/L) compared to negative participants (25.20 U/L, standard deviation of 17.541 U/L). To summarize, there was a significantly higher average ALT level in young HGV-positive participants (below 18 years old) compared to HGV RNA-negatives. However, in other age groups, the mean ALT was almost similar between HGV negative and positive participants.

Overall, the average AST levels (aspartate aminotransferase) showed minor differences volunteers who tested negative and positive for HGV (hepatitis G virus) across different age groups. In the age group of < 17 years, the mean AST was slightly higher in HGV-positive participants compared to HGV-negative participants, but the difference was small. Similarly, the mean AST level showed the same pattern in the age group of 18-36 years. However, in the age group of 37-54 years, the difference in the mean AST levels between HGV negative and positive participants was not statistically significant **(Figure 5).**

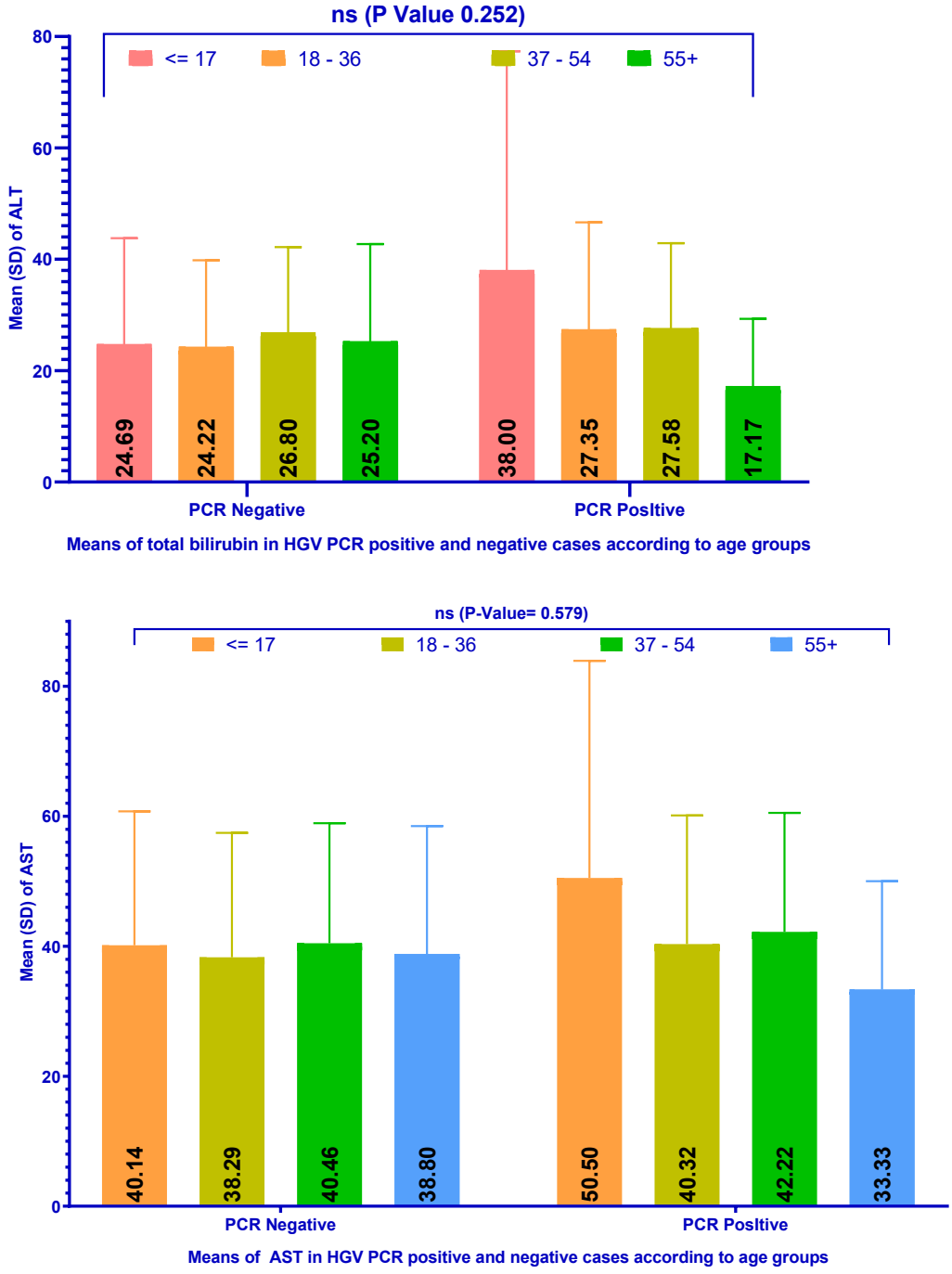

Figure 5. Means ALT and AST in HGV PCR positive and negative participants according to age groups.

2.2 Means of Total bilirubin and Serum Albumin in HGV positive and negative cases according to age groups

Among participants aged 17 or younger, the mean (SD) total bilirubin levels were 1.600 (1.1103) mg/dL in HGV RNA-negative participants and 2.078 (1.4575) mg/dL in HGV RNA-positive participants. For participants aged 18-36 years, the mean (SD) total bilirubin levels were 1.886

(1.0484) mg/dL in HGV RNA-negative individuals and 1.697 (0.8943) mg/dL in HGV RNA-positive individuals.

Among individuals aged 37-54 years, the mean (SD) total bilirubin levels were 2.044 (1.0696) mg/dL in HGV RNA-negative participants and 2.097 (1.0485) mg/dL in HGV RNA-positive participants. For individuals aged 55 or older, the mean (SD) total bilirubin levels were 2.060 (0.7266) mg/dL in HGV RNA-negative participants and 2.367 (1.4278) mg/dL in HGV RNA-positive participants.

The findings suggest that HGV infection may be associated with higher total bilirubin levels in individuals aged 55 or older, but not in younger age groups. More research is required to investigate this association in larger studies.

Total bilirubin and serum albumin's statistically non-significant differences between HGV RNA-negative and HGV RNA-positive participants, regardless of their age. Specifically, among participants aged ≤ 17 years, the mean (SD) serum albumin levels were 3.966 (0.6466) g/dL for HGV RNA-negative individuals and 3.683 (0.7950) g/dL for HGV RNA-positive individuals. For participants aged 18–36 years, the mean (SD) serum albumin levels were 3.941 (0.7866) g/dL for HGV RNA-negative individuals and 3.792 (0.6829) g/dL for HGV RNA-positive individuals.

Similarly, among participants aged 37–54 years, the mean (SD) serum albumin levels were 4.105 (0.6607) g/dL for HGV RNA-negative individuals and 4.158 (0.7105) g/dL for HGV RNA-positive individuals. Lastly, among participants aged 55+ years, the mean (SD) serum albumin levels were 4.080 (0.5762) g/dL for HGV RNA-negative individuals and 2.867 (1.0093) g/dL for HGV RNA-positive individuals.

Bilirubin and serum albumin between participants who were negative for HGV RNA and those who were positive, regardless of their age showed insignificant differences. Overall, no substantial variation in serum albumin in different age groups for both HGV RNA-negative and HGV RNA-positive individuals. The mean serum albumin ranges 3.683 to 4.158 g/dL for HGV RNA-positive individuals and from 3.792 to 4.105 g/dL for HGV RNA-negative individuals across different age groups. The highest difference was observed in the oldest age group (55+ years) for HGV RNA-positive individuals, with a mean serum albumin level of 2.867 g/dL **(Figure 6)**.

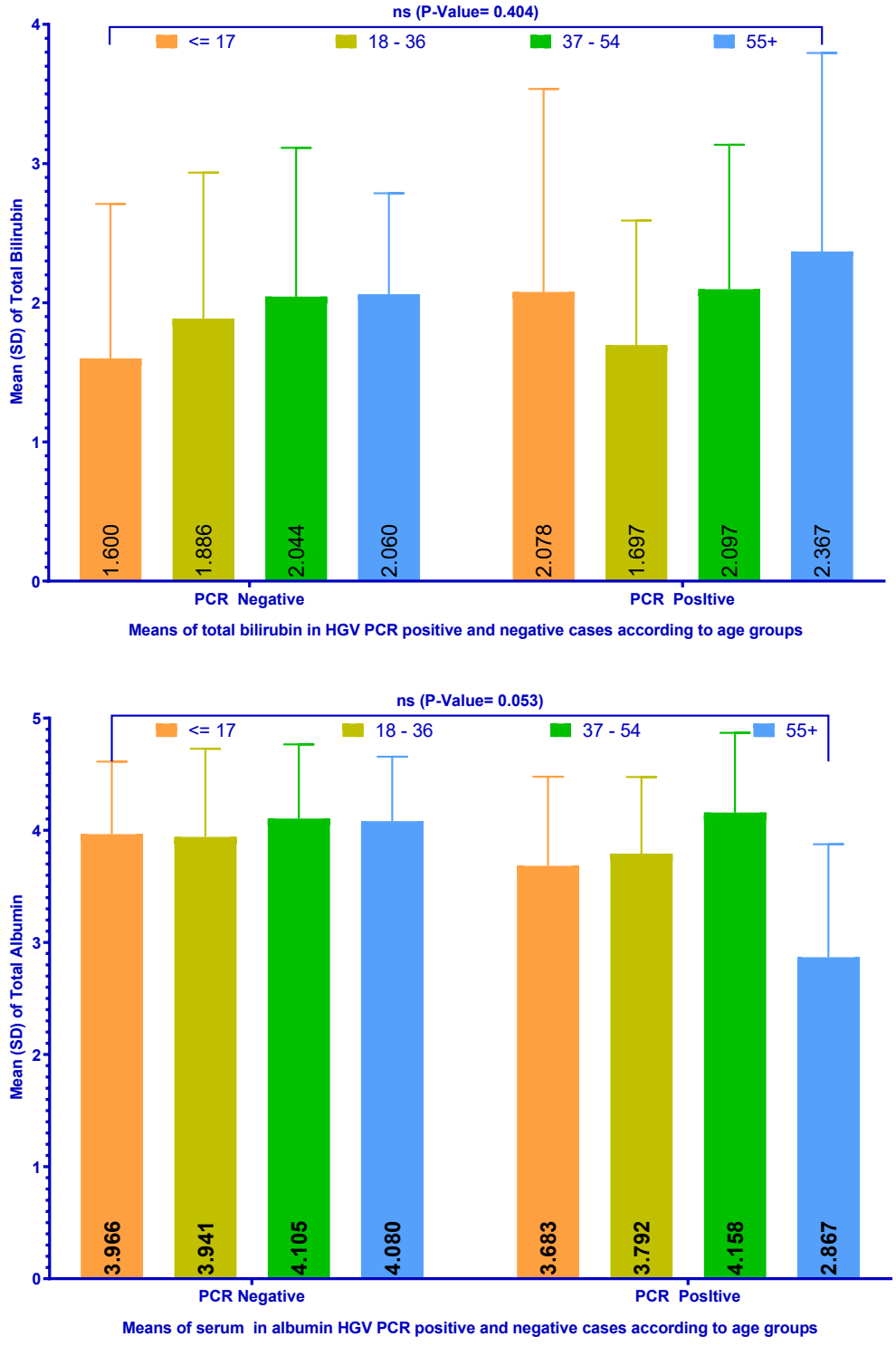

Figure 6. Means Total bilirubin and Serum Albumin in HGV PCR positive and negative participants according to age groups.

2.2 Means of ALP in HGV positive and negative cases according to age groups

In participants aged 17 years and below, HGV RNA-positive had significantly higher mean ALP levels compared to negative. However, in individuals aged 18-36 years, mean ALP levels between

HGV RNA positive and negative participants were non-significant. Additionally, ALP levels between HGV RNA positive and negative participants in the age groups of 37-54 years and 55+ years showed non-significant differences. Overall, the standard deviation and range of ALP levels were higher in HGV RNA-positive individuals compared to HGV RNA-negative individuals in all ages.

The ALP levels between HGV RNA negative and positive participants within the age groups of 37-54 years ($p = 0.23$) and 55+ years ($p = 0.41$). In the 37-54 age group, the mean (SD) ALP level was 305.51 ± 168.282 IU/L in HGV RNA negative participants and 273.22 ± 162.263 IU/L in HGV RNA positive participants. In the 55+ age group, the mean (SD) ALP level was 213.60 ± 77.632 IU/L in HGV RNA negative participants and 231.33 ± 163.362 IU/L in HGV RNA positive participants **(Figure 7).**

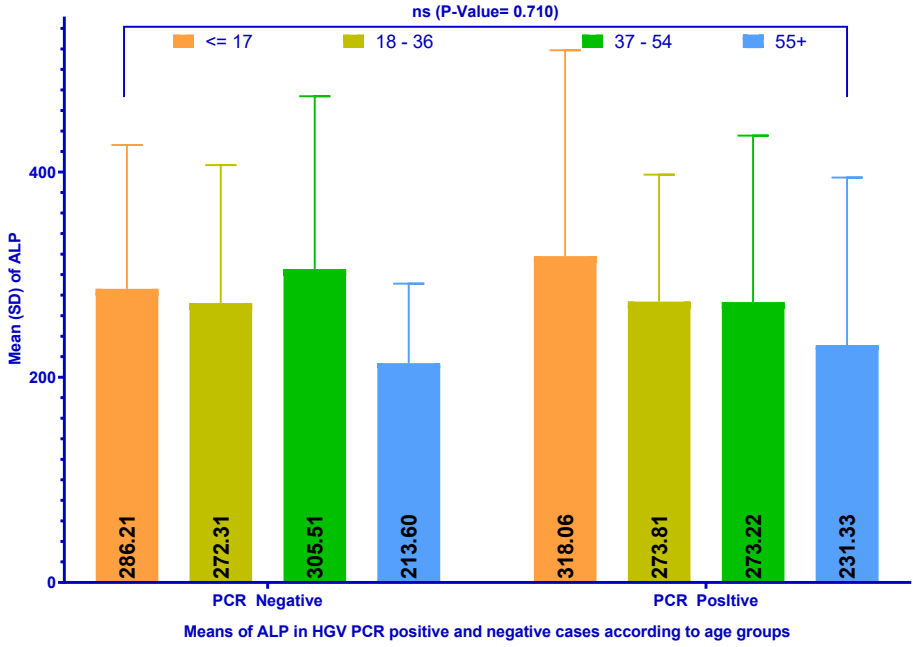

Figure 7. Means Total ALP in HGV PCR positive and negative participants according to age groups.

Discussion

HGV RNA may not be a significant contributor to liver inflammation and damage, as shown by our current study, non-significant differences in liver function enzyme levels (ALT, AST, serum albumin, ALP, and total bilirubin) between high-risk groups and chronic liver patients with or without HGV RNA. Additionally, LFEs were observed in various age groups of participants with and without HGV RNA and were shown to have no significant differences. Our Findings are

similar to previously reported studies which suggested that HGV RNA is not a major contributor to liver inflammation and damage(13).

A study published in the Journal of European journal of gastroenterology & hepatology in 2019 reported Liver enzymes or liver inflammation not correlated with HGV-RNA in patients with chronic liver disease(14). Another study published in 2014 showed HGV-RNA was not a predictor of hepatic disease progression in HBV infection(15).

The current study's findings are also supported by the fact that non-significant differences in LFE in the age groups with or without HGV RNA. This further suggests that HGV RNA may not have an impact on liver function across different age groups. Overall, these results and previous studies showed HGV RNA may not pose a silent threat to liver health in chronic liver patients and high-risk groups and that other factors such as chronic hepatitis B virus infection, alcohol consumption, and obesity may be more important contributors to liver inflammation and damage.

The study found that among 221 participants tested, 97 (43.9%) tested positive for HGV RNA, which is a relatively high prevalence compared to previous studies e.g., a study in 2010 showed HGV-RNA prevalences in chronic hepatitis B and C cases was 28.6% and 30.8%, respectively(7).

The study found no statistically significant difference in the levels of liver function enzymes (LFEs), ALT, AST, ALP, and total bilirubin, between high-risk groups and chronic liver patients with or without GBV-C/HGV RNA. This is consistent with previous studies that have found no association between HGV RNA and liver inflammation or damage(16).

The study found non-significant differences in the levels of LFEs in different age groups of participants. This contrasts with some previous studies of the association between age and HGV RNA prevalence(17, 18).

The study suggests that HGV RNA may not significantly threaten liver health in chronic liver patients and high-risk groups based on liver function enzyme levels. This is in line with earlier research that did not discover any connection between HGV RNA and inflammation or damage to the liver(16).

Conclusion

In conclusion, the study by Chivero and Stapleton (2015) adds to the existing literature by providing further insights into the prevalence and impact of HGV RNA on liver function in chronic

liver patients and high-risk groups (19). While our research found a relatively high prevalence of RNA, the results suggest that Viral RNA may not contribute significantly to liver inflammation and damage, based on liver function enzyme levels. Significant ramifications for the management and treatment of chronic liver disease result from these findings.

Additionally, several earlier investigations have not discovered any appreciable variations in liver enzyme levels between HGV-positive and HGV-negative cohorts. For instance, Sathar et al.'s 2000 study(20) examined patients with chronic hepatitis C and observed no discernible variations in AST, ALT, or γ-GTP levels according to HGV status. Similarly, Reshetnyak et al.'s 2008 meta-analysis, which included data from 178 cases, did not discover any proof that co-infection with HGV significantly affects aminotransferase levels in individuals with chronic hepatitis C(17). Nevertheless, the presence of HGV RNA by itself might not indicate illness or active viral replication. One drawback is that PCR-based assays are unable to distinguish between virus fragments or propagating versus dormant viruses(17). To gain a deeper understanding of the pathophysiology of HGV and its effects on liver function over time, more longitudinal studies Assessing antigens and viral load are required(16, 17). Furthermore, variations in the research populations, co-infection statuses, and other variables might affect the outcomes. Further extensive controlled investigations are required to clarify the hepatotoxicity of HGV(13, 14).

We concluded that, among chronic liver patients or high-risk populations, HGV might not be a quiet threat to liver health and function based on the current study results. The absence of significant variations in liver enzyme levels according to HGV-RNA positive suggests that the virus might not be the only factor contributing to increased liver damage or inflammation. Therefore, based on this evaluation of common liver function biomarkers, HGV does not seem to adversely affect liver health or substantially contribute to the development of liver disease. In conclusion, lack of liver enzyme rise should not rule out the possibility of subtle liver effects; present evidence suggests HGV is not immediately hepatotoxic, while further research is necessary to understand its clinical importance in different patient populations.

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