

Prevalence of Hepatitis-G virus infection in patients with liver diseases in district Larkana, Pakistan

Lubna Qureshi, Nisar Ahmed Shaikh, Ikram Ahmed Tunio, Jaikrishan Ambwani, Nadia Bhatti, Pir Bux Ghumro

Shah Abdul Latif University, Khairpur, Surgical Unit II Teaching Hospital, Department of Pathology and Urology, Chandka Medical College, Larkana, Pakistan

Objective: To investigate the Hepatitis-G virus (HGV) infection prevalence in patients with liver diseases and the correlation of HGV with HBV, HCV and HDV.

Methodology: In this cross-sectional study, a total of 196 patients were recruited from August 2018 to January 2019. From blood samples, serum was separated and tested for HGV by using ELISA Kit.

Results: Out of 196 patients, 123 were males and 73 females. Mean age was 40 years. We found 47(23.9%) positive for Hepatitis-B infection, 65(33.2%) positive for Hepatitis-C infection, 28(14.3%) positive for Hepatitis-D infection, 37(18.9%) with cirrhosis of liver and 19(9.7%) with carcinoma of liver. Among them, 7(3.6%) were positive for HGV. HGV was co-related with

Hepatitis-C (5 cases) and Hepatitis-D (2 cases) and there was no case found for HGV positive in patients with carcinoma of liver, cirrhosis of liver and Hepatitis-B. Male were more prone than female and common in married and in young ages. Those patients who are more prone to blood transfusion and blood products were at high risk of exposure such as thalassemia, hemophilia and persons with liver transplantation and I/V drug abusers and on hemodialysis.

Conclusion: Prevalence of HGV was 3.6% in liver disease and more prone in male with younger ages. It was also correlated with HCV and HDV. (Rawal Med J 202;45:755-757).

Keywords: Prevalence, co-infection, cirrhosis, hepatitis G.

INTRODUCTION

Hepatitis-G virus (Human Pegivirus) was found in 1995 in primates like Lemurs, Tarsiers, lorises, Apes & Monkeys.¹ This recently identified HGV virus is related with flaviviridae family which is RNA positive-stranded related to Hepatitis C virus.^{1,2} The range of HGV prevalence worldwide is in most of blood donors from 0.9% up to 14.6%.³ HGV is currently increasing all over the world and it has infected about one third of whole population.⁴ Those individuals who received the blood transfusion are with the risk of parenteral exposure are found a large number of incidences of HGV.^{4,5} Around 10 to 25% population of Hepatitis C may have HGV.

Patients with hemophilia, thalassemia, persons with liver transplant and those exposed to blood product & blood transfusion are at high risk.⁶⁻⁸ There is probability for decrease of HGV infection after sometime.⁹ Patients of carcinoma of liver have V and HCV.¹⁰ Large number of CLD individuals and HGV infected patients had no past history of intra venous exposure so there may be other routes are

also involved in spread of HGV.¹¹⁻¹³ HGV pathogenicity and occurrence in various liver diseases have been studied.^{14,15} It may share route with HCV, as this is more common in the Western and Japanese populations.^{16,17} The objective of this study was to investigate the HGV infection prevalence in patients with liver diseases and its correlation with HBV, HCV and HDV.

METHODOLOGY

This cross-sectional study was carried out from August 2018 to January 2019 after obtaining permission from Ethical committee SMBBMU Larkana. Blood samples of 196 cases with different liver diseases had samples of blood collected. A structured and pre-tested questionnaire was filled by the patients after verbal informed consent. From each patient 5ml blood collected and at (3000-5000 r/m) centrifuged for 05 to 10 minutes and serum was separated then freeze at -20°C for further procedure. Blood samples were tested for HGV by ELISA Kits by ABNOVA®.

ELISA KIT by ABNOVA® clinical sensitivity assay has been calculated by a panel of samples obtained from 560 Hepatitis G positive individuals confirmed positive by HGV RT-PCR and was determined to be 100%.²¹ The specificity was evaluated in a panel of samples from 1200 healthy individuals and no false positive results were observed indicating 100% specificity.

Statistical Analysis was performed using SPSS version 20. Like age and gender of individuals, the standard deviation and mean was calculated for quantitative data. The cases were compared by the Chi-square test

RESULTS

Out of 196 study participants, 123 were males and 73 females with mean age of 40 years. The infected patients with different hepatic illnesses comprising of 47(23.9%) cases with HBV infection, 65(33.2%) cases presented with HCV infection, 28(14.3%) with HDV infection, 37(18.9%) cirrhosis of liver and 19(9.7%) with carcinoma of liver (Fig. 1). Patients with different liver diseases, seven (3.6%) patients out of 196 were detected positive of HGV. HGV was correlated with HCV in five cases and HDV in two cases. There were no cases of HGV found in the patients with carcinoma of liver, cirrhosis of liver and HBV positive persons.

Fig 1. Percentages of patients with different liver diseases.

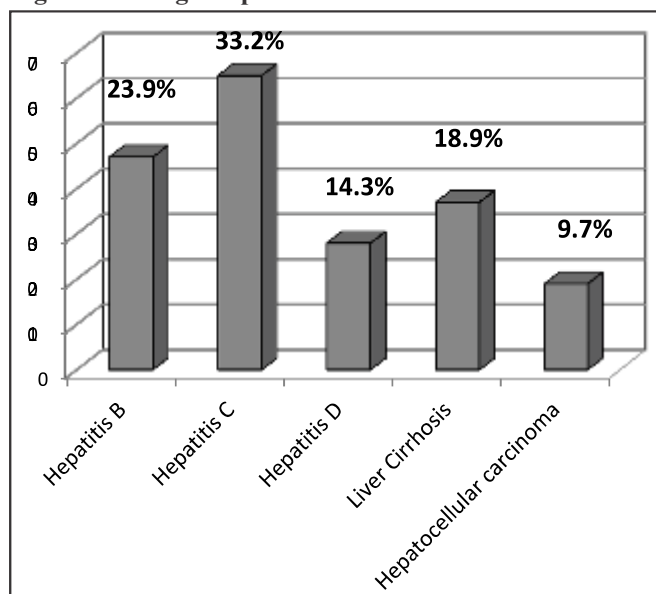


Figure 2. Distribution of Hepatitis-GV positive cases according to age.

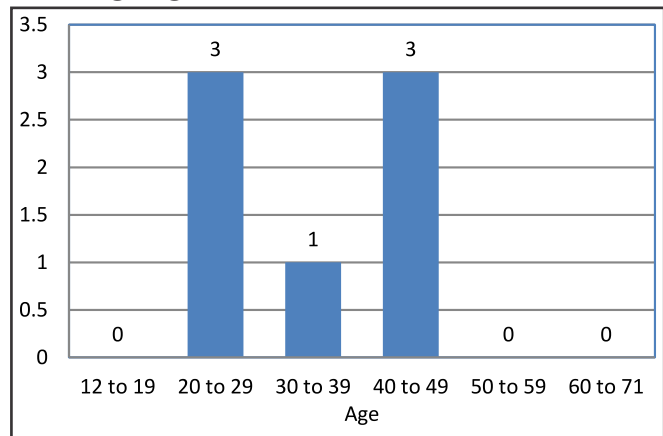


Table. The prevalence of Hepatitis-G virus infection in various geographical areas.

S. No.	Geographical Area	Prevalence of HGV	Year
1.	Africa	10.0% to 20.0%	1998
2.	Australia	4.0%	1996
3.	Brazil	7.1%	2002
4.	Colombia	6.1%	1998
5.	Hungary	5.5%	1999
6.	India	6.0%	2007
7.	Iran	4.8%	2009
8.	Japan	5.0%	2005
9.	Pakistan	2.3%	1999
10.	USA	1.6%	1996

Among positive cases, 5 were male and 2 female. Among positive cases, only one patient was unmarried and 6 were married. Age group from 20 to 49 was most affected, it shows the young age people are more prone to the HGV than others (Fig. 2).

DISCUSSION

Our result strongly validates the spread of HGV through infected blood. Etiology of this spread is not known cryptogenic CLD and in hemodialysis patients.⁹⁻¹¹ Patients with hemodialysis HGV positive patients did not generally present with liver diseases.¹¹ With interferon the HGV infection usually does not change the response in HCV infected patients.^{10,12} This data provides evidence that HGV co-infection induced in CLD patients there is no important role which can cause HBV and HCV. Compared to other countries, HGV

prevalence in Pakistan is on low side (Table).

Furthermore, there is no any evidence found by the researchers that show the HGV pathogenicity and co-infection with HCV cannot create severe hepatitis than with HCV itself, while HGV turns chronic regularly, patients who are infected with only HGV the appearance of chronic hepatitis is absent in those.^{13,14} In current study, the incidence of HGV in the patients with liver diseases was associated to HDV including HCV which was elucidated in another study.¹⁵ Consequently, HGV does not seem to be associated with cirrhosis of liver, hepatocellular carcinoma and HBV in our geographical area.^{16,17}

The occurrence of HGV infection of 3% in blood donors, HCV in 7%, HBV in 8%, in alcoholic liver disease 2%, in hepatocellular carcinoma 4% and in cryptogenic chronic liver disease 8% is comparable to our study.¹⁰ HGV prevalence can be controlled/reduced by HGV screening test among those who are positive with HDV and HCV.

CONCLUSION

The prevalence of HGV was 3.6% in patients with liver disease and was more prone in male with young ages and also correlated with HCV and HDV.

Author Contributions:

Conception and design: Lubna Naz
Collection and assembly of data: Nisar Ahmed Shaikh
Analysis and interpretation of data: Ikram Ahmed Tunio
Drafting of the article: Lubna Naz
Critical revision of article for important intellectual content: Jai Krishan Ambwani
Statistical expertise: Nadia Bhatti
Final approval and guarantor of the article: Pir Bux Ghumro
Corresponding author email: Nisar Ahmad Sheikh: drnisarshaikh@yahoo.com

Conflict of Interest: None declared

Rec. Date: Feb 26, 2020 Revision Rec. Date: Jul 8, 2020 Accept Date: Sept 12, 2020

REFERENCES

1. Stapleton JT, Fong S, Muerhoff AS, Bukh J, Simmonds P. The GB viruses: a review and proposed classification of GBV-A, GBV-C (HGV), and GBV-D in genus Pegivirus within the family Flaviviridae. *J Gen Virol*. 2011;92:233-8.
2. Karayiannis P, Pickering J, Zampino R, Thomas HC. Natural history and molecular biology of hepatitis G virus/GB virus C. *J Clin Virol*. 1998;15;10:103-10.
3. Konomi N, Miyoshi C, Zerain CL, Li TC, Arakawa Y, Abe K. Epidemiology of hepatitis B, C, E, and G virus infections and molecular analysis of hepatitis G virus isolates in Bolivia. *Clin Microbiol Infect*. 1999;37:3291-5.
4. Feng Y, Zhao W, Feng Y, Dai J, Li Z, Zhang X, et al. A novel genotype of GB virus C: its identification and predominance among injecting drug users in Yunnan, China. *PloS One*. 2011;6:56-7.
5. Muerhoff AS, Leary TP, Sathar MA, Dawson GJ, Desai SM. African origin of GB virus C determined by phylogenetic analysis of a complete genotype 5 genome from South Africa. *J Gen Virol*. 2005;86:1729-5.
6. Idilman R, Ustun C, Aslan O, Zcan M, Arat M, Bozkaya H, et al. The Incidence of Hepatitis G Virus in patients with hematological malignancies: the relationship to the number of blood and blood products transfusions. *Turk J Haematol*. 2000;17:67-1.
7. Pavlova BG, Heinz R, Selim U, Tuchler H, Pittermann E, Eder G. Association of GB virus C (GBV-C)/hepatitis G virus (HGV) with hematological diseases of different malignant potential. *J Med Virol*. 1999;57:361-6.
8. Keresztes K, Takacs M, Horanyi M, Miltenyi Z, Illes A. HCV and HGV Infection in Hodgkin's Disease. *Pathol Oncol Res*. 2003;9:222-5.
9. Chopra S. GB virus (hepatitis G) infection. Accessed on. 2010;18:35-6.
10. Ahmed QM. Hepatitis G virus (HGV): where we stand and what to do. *International Int J Immunol Stud*. 2011;1:255-3.
11. Selim H, El Barrawy M, Mohamed O, Gamal El-Din M. Hepatitis G Virus Infection in Patients with Hepatitis C. *Publ Health*. 2010;40:563-8.
12. Mibagheri SA. Hepatitis G virus: a newly diagnosed infectious agent. *Med J* 1999;1:60-2.
13. Oliveira LA, Martins R, Carneiro MA, Teles SA, Silva SA, Cardoso DD, et al. Prevalence and genotypes of GB virus C/hepatitis G virus among blood donors in Central Brazil. *Mem Inst Oswaldo Cruz*. 2002;97:953-7.
14. Corwin AL, Hyams KC, Kim JP, Wages J, Doss R, Sulaiman A, et al. Evidence of worldwide transmission of hepatitis G virus. *Am J Trop Med Hyg*. 1997;57:455-6.
15. Ross RS, Viazov S, Schmitt U, Schmolke S, Tacke M, Ofenloch-Haehnle B, et al. Distinct prevalence of antibodies to the E2 protein of GB virus C/hepatitis G virus in different parts of the world. *J Med Virol*. 1998;54:103-6.
16. Kao JH, Chen W, Chen PJ, Lai MY, Lin RY, Chen DS. GB virus-C/hepatitis G virus infection in prostitutes: Possible role of sexual transmission. *J Med Virol*. 1997;52:381-4.
17. Wang HL, Jin DY. Prevalence and genotype of hepatitis G virus in Chinese professional blood donors and hepatitis patients. *Int J Infect Dis*. 1997;175:1229-3.