

Hepatitis C Seroprevalence Among A Tertiary Hospital Based General Population In Northern India

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ABSTRACT

Hepatitis C is a disease caused by enveloped, RNA virus belongs to Flaviviridae family. It causes inflammation of the liver that lead to liver cirrhosis and finally hepatocellular carcinoma. Its genome consists of open reading frame (ORF) codes for structural and non-structural proteins. The ORF have 5' and 3' UTR regions. Hepatitis C virus is cause of post transfusion hepatitis. Symptoms of HCV include fatigue, dark urine, belly pain, joint pain, itchy skin, sore muscles and jaundice. It is a blood-borne transmitted agent. Use of unsafe therapeutic injections also leads to HCV infection. Diagnosis of HCV infection can be done by various methods like Enzyme Immunoassay (EIA) and Recombinant Immunoblot Assay (RIBA). The aim of this study is to estimate seroprevalence of Hepatitis-C in both sexes and different age groups in hospital based general population. And to study the trends of HCV infections in a tertiary hospital located at Northern India.

A prospective study was conducted for four months (January-April) at Tertiary Hospital in Northern India. Total numbers of 1643 blood samples were screened for the presence of anti-HCV antibodies in patient's serum. Samples were tested by HCV TRI-DOT rapid test. Positive samples were retested by SD BIOLINE HCV rapid test and confirmed by ELISA. Out of 1643, 102 (6.2%) samples were HCV positive. Among seropositive samples, 48 were males (2.9%) and 54 were females (3.2%). HCV seropositivity was shown by 40 IPD patients (2.4%), 52 OPD patients (3.1%) and 10 ICU patients (0.6%). Among departments, patients from Recovery showed (0.6%), General ward (1.0%), Private room (0.2%), Neurology lab (0.1%) and Emergency (0.3%) showed HCV seropositivity. On analyzing age-wise seropositivity, it was found that maximum seropositivity was seen in 30-40 years (2.9%) followed by >55 years (1.4%), 40-55 years (1.2%) and <30 years (0.5%). Analysis of patients on the basis of risk factors showed that 17 had history of surgical operation (1.0%), 18 had history of blood transfusion (1.0%), 6 had history of dental procedure (0.3%), 25 showed history of injecting drug use (1.5%), 21 used contaminated syringes (1.2%) and 15 patients was under haemodialysis (0.9%). Professional health worker should protect themselves while handling infected blood. Counselling and testing should be done for those who are at risk for infection.

Key words: Hepatitis C virus, seropositive, ELISA, risk factors, cirrhosis

1. INTRODUCTION

The term 'Hepatitis' refers to damage to the liver by causing inflammation, cirrhosis and hepatocellular carcinoma (Memon et al., 2002 and Kohli et al., 2014). Inflammation is the local reaction of the body. Hepatitis C virus is an enveloped, spherical, positive stranded

RNA virus which belongs to Flaviviridae family. It is 50 to 60 nm virus. It is surrounded by an envelope having glycoprotein spikes (Bonkovsky et al., 2001). It can cause infection throughout the life. It is blood-borne infection. This virus passes from blood of an infected person to blood of an uninfected person (Eyre et al. 2014). According to World Health Organization (WHO), 10-24 million people are HCV infected in India. Humans are only natural hosts of HCV. Hepatitis C is known to be as a “silent disease” because it is difficult to detect it in early stage and that is why early treatment of HCV is difficult. In most of the people, virus infection is not resolved naturally. When the liver is not able to remove the virus, the person becomes chronically infected. Chronic infection further causes cirrhosis followed by hepatocellular carcinoma and finally death (Sharma et al., 2010). Harvey J. Alter (Mid 1970s) was first person who worked on virus. Michael Houghton first cloned and identified this virus. They named it as non-A, non-B hepatitis (NANBH). Medical researches took decade to identify virus. Later in April 1989, the name was changed from NANBH to Hepatitis C virus (Weiner et al., 1990). N-terminal region of ORF codes for structural proteins and rest of region codes for non-structural proteins. The ORF have 5′ and 3′ untranslated regions (UTR). These regions have role in RNA replication and polyprotein translation (Thurner et al., 2004). 5′ UTR is conserved region. It consists of four domains, I to IV with pseudoknot and stem-loops (Brown et al., 1992). It consists of 341 nucleotides. First 12 to 30 nucleotides along with domains II, III and IV make internal ribosome entry site (IRES) (Honda et al., 1996). 3′ Untranslated region consist of 225 nucleotides. In HCV infected patient, HCV genotype does not change. The treatment of HCV depends upon strain of genotype present (Chevaliez et al., 2006). Hepatitis C virus is a cause of post transfusion hepatitis (Alter et al., 1999). HCV is compared with ‘viral time bomb’ as it is leading hepatotropic virus. It is cause of acute hepatitis, hepatocellular carcinoma and chronic liver diseases (Ali et al., 2009). Worldwide, 200 million people are infected with HCV (Lavanchey et al., 2009). About 170 million have chronic HCV with 500,000 deaths per year (Brown et al., 2003). According to World Health Organization, 10-24 million people are HCV infected in India (Albanese et al., 2011). In healthy people, HCV seroprevalence ranges from 1.5% to 4% (Irshad et al., 1994). In blood donors, it varies from 0.48% in Vellore to 1.85% in New Delhi (Panigrahi et al., 1997). In patients suspected for acute viral hepatitis, seroprevalence ranges from 3% to 12% (Kar et al., 1997). In HCV infected people, 27% of cirrhosis occurs worldwide. In last 20-40 years, most HCV transmission occurs in young adults and similar rate was found in Australia. Hepatocellular carcinoma (HCC) is one of the most frequent malignancies in Asia. Cancer deaths exceeds in number and is ranked 3rd in Japan and Korea. In Korea, the number of deaths from liver cancer increased from 5,789 in 1983 to 9,966 in 1994. After that, it remains constant at 9,500/100,000 in 2003. In Japan, 80% of HCC cases are caused by HCV infection (Kim et al., 2008). The rate of HCC mortality in Iran increased from 1999 to 2004 (Pourhoseingholi et al., 2010). In United States, incidence of acute hepatitis C has declined phase in 1992 but, since 2003, rates have plateaued (Daniels et al., 2007). In UK, chronic HCV infection and end-stage liver disease have been identified (Mann et al., 2008). World Health Organization (WHO) found that immigrants to the UK may be at risk (Uddin et al., 2010).

2. STATUS OF HCV INFECTION IN INDIA

Hepatitis C is an important pathogen in India which is causing liver disease. In India, there is very high frequency of HCV (60-90%). HCV infection was found to be more common in injecting drug users (IDU) (Basu et al., 2010). In Delhi, HCV prevalence was 0.78% in voluntary blood donors and 1.33% in pregnant women (Irshad et al., 1998). Chadha et al., (1999) reported 0.09% HCV prevalence in rural Maharashtra. In Hyderabad, it was found to be 1.4% in gastroenterology camps (Khaja et al., 2006). 7.89% HCV seroprevalence was

found in Arunachal Pradesh (Phukan et al., 2001). In pregnant women, HCV seroprevalence varies from 0.6% to 1.4% (Parthiban et al., 2009, Kumar et al. 2007, Sood et al., 2012). Pathak et al., (2013) and Khaja et al.,(2006) reported HCV prevalence as 1.4% and 2.02% in two studies from Andhra Pradesh. Another study conducted in West Bengal showed 0.71% positivity by PCR. Study conducted in Lucknow and Chennai showed coinfection of HIV-HCV as 1.61% and 2.2% (Mukhopadhyaya et al., 2008).

3. STATUS OF HCV INFECTION IN PUNJAB

Studies related to seroprevalence of HCV become “eye openers” as it is causing chronic infections (Alter et al., 2007 and Zaghoul et al., 2014). Study conducted by Sood et al., reported 5.2% HCV seroprevalence among families of Punjab. Age-group 40-60 years were highest seroprevalent (Sood et al., 2012). Presence of antiHCV antibodies among injecting drug users was found to be 49% (Panda et al. 2014).Singh et al., (2014) reported highest frequency of HCV (30.04%) in Ludhiana and lowest frequency (0.39% each) Tarn Taran and Ropar. Rural locality of Punjab was more HCV seroprevalent (67.25%) than urban locality (32.75%).

The symptoms of hepatitis C include fatigue, dark urine, belly pain, joint pain, itchy skin, sore muscles, and jaundice. People with chronic hepatitis C will have cirrhosis in which tissue of liver changes to fibrous tissue and ultimately scar-like hardening and liver stops functioning. The symptoms of cirrhosis are shrinkage of muscles, bleeding from enlarged veins in digestive tract, redness of palms of hands, clusters of blood vessels, damage to brain and nervous system.

4. MATERIAL AND METHODS

A prospective study was conducted for four months (January-April) in the department of Clinical Microbiology at Tertiary Hospital in Northern India. Total numbers of 1643 blood samples were collected from individuals who attended outdoor patient and indoor patient departments of Tertiary hospital. Samples were kept in clean, dry and sterile red vials. Proper labelling was done with patient’s full name, sex, age, areas and date of collection. Vials containing sample were centrifuged at 10,000 r.p.m. for 15 minutes. Sera were used for screening of anti-HCV antibodies. A total of 1643 blood samples were screened for the presence of anti-HCV antibodies in the patient’s serum. There were two methods used for screening of HCV seropositivity Rapid test methods and Enzyme linked immunosorbent assay (ELISA).

The 4th Generation HCV TRI-DOT kit is used for qualitative detection of antibodies to Hepatitis C Virus in serum. All the samples were tested by HCV TRI-DOT kit method. Further, HCV positive samples were retested by another rapid method i.e. SD-BIOLINE HCV method. Finally, all positive samples were confirmed by ELISA.

4. RESULT

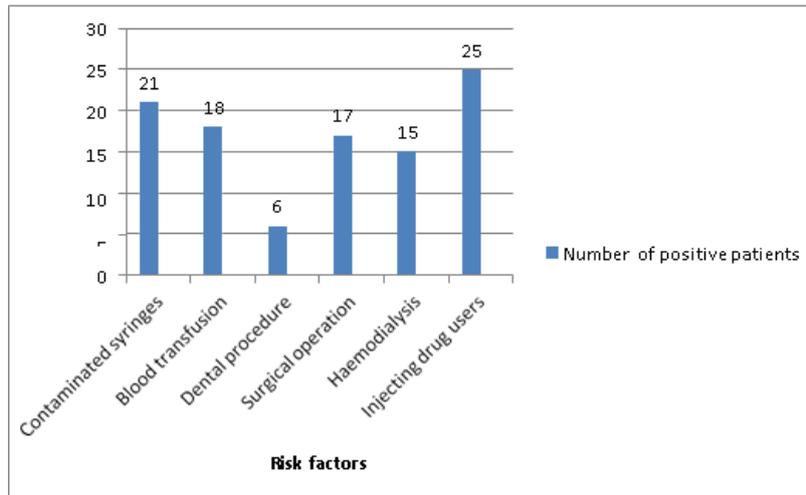


Table 1: Risk factors for HCV infection

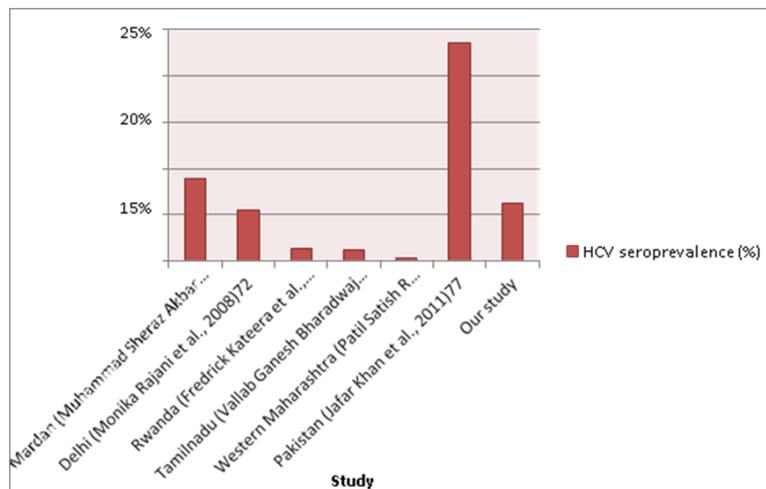


Table 2: HCV seroprevalence in hospital-based population

5. DISCUSSION

A total of 1,643 patients were screened in the present study which was conducted in a tertiary hospital in Northern India for four months. Sera were screened for the presence of anti-HCV antibodies. Out of 1,643 samples, 102 (6.2%) were HCV positive. All HCV positive samples (102) were tested with HCV TRI-DOT kit and retested with SD-BIOLINE HCV kit. All positive samples were confirmed by ELISA. Among seropositive samples, 48 were males (2.9%) and 54 were females (3.2%). Females were more HCV prevalent. Samples were collected from different wards .i.e. IPD (indoor patient department), OPD (outdoor patient department) and ICU (intensive care unit). HCV seropositivity was shown by 52 OPD patients (3.1%), 40 IPD patients (2.4%) and 10 ICU patients (0.6%). Samples collected from OPD showed increased HCV infection rates (3.1%) while samples collected from ICU showed less HCV infection rates (0.6%). There were many departments in hospital named as Recovery, General ward, Private room, Neurology lab and Emergency. The seropositivity of HCV was higher in patients of General ward (1.0%) and lower in Neurology lab (0.1%).

6. CONCLUSION

Hepatitis C is a virus that infects the liver by causing liver inflammation and damage. It is spread by exposure to contaminated blood, sharing of needles, sex with infected person and from mother to child during birth. According to Centre for Disease Control and Prevention

(CDC), about 17,000 new hepatitis C cases were seen every year. Out of which 75 to 85% of cases become chronically infected.

The seroprevalence of HCV is 6.2% in our study. Such high HCV prevalence is due to increase in number of injecting drug users in Northern India. There should be counselling for young generation in order to reduce HCV infection. Primary prevention activities and secondary prevention activities should be acquired to decrease the risk for HCV infection. Some important factors should be considered such as suitable diagnostic tests and public health awareness. Counselling and testing of persons should be done for those who are at risk for infection or who practice high-risk behaviours. Healthcare workers should carry out history about use of drugs or unprotected sex. Drug users should be advised to go for substance-abuse treatment, stop taking drugs, use sterilized syringes and dispose syringes in safely manner. Safety precautions are needed to be taken in haemodialysis settings. Staff should use gloves while handling blood, body fluids, excretions, secretions and contaminated equipment. Persons with high ALT (alanine aminotransferase) and AST (aspartate aminotransferase) levels should be checked daily for HCV infection. Patient with history of blood transfusion or organ transplantation should be screened for HCV infection. Information regarding transmission of HCV should be provided to public such as nature of hepatitis C, benefits of early detection, steps to be taken if HCV positive, screening tests and drug-treatment centres. Persons with HCV positive should be advised to prevent themselves from further liver disorders, stop drinking alcohol and not to take new medicines without consulting doctor. The data of current study will be helpful in taking prevention and control measures against HCV infection.

7. REFERENCES

- [1] Albanese, E., Liu, Z., Acosta, D., Guerra, M., Huang, Y., Jacob, K. S., ... &Uwakwe, R. (2011). Equity in the delivery of community healthcare to older people: findings from 10/66 Dementia Research Group cross-sectional surveys in Latin America, China, India and Nigeria. *BMC health services research*, 11(1), 153.
- [2] Ali, S. A., Donahue, R. M., Qureshi, H., &Vermund, S. H. (2009). Hepatitis B and hepatitis C in Pakistan: prevalence and risk factors. *International journal of infectious diseases*, 13(1), 9-19.
- [3] Alter, M. J., Kruszon-Moran, D., Nainan, O. V., McQuillan, G. M., Gao, F., Moyer, L. A., ... & Margolis, H. S. (1999). The prevalence of hepatitis C virus infection in the United States, 1988 through 1994. *New England journal of medicine*, 341(8), 556-562.
- [4] Alter, M. J. (2007). Epidemiology of hepatitis C virus infection. *World journal of gastroenterology: WJG*, 13(17), 2436.
- [5] Basu, D. (2010). Overview of substance abuse and hepatitis C virus infection and co-infections in India. *Journal of Neuroimmune Pharmacology*, 5(4), 496-506.
- [6] Bonkovsky, H. L., & Mehta, S. (2001). Hepatitis C: a review and update. *Disease-a-Month*, 47(12), 610-647.
- [7] Brown, E. A., Zhang, H., Ping, L. H., & Lemon, S. M. (1992). Secondary structure of the 5' nontranslated regions of hepatitis C virus and pestivirus genomic RNAs. *Nucleic acids research*, 20(19), 5041-5045.
- [8] Brown Jr, R. S., &Gaglio, P. J. (2003). Scope of worldwide hepatitis C problem. *Liver transplantation*, 9(11), S10-S13.
- [9] Chadha, M. S., Tungatkar, S. P., &Arankalle, V. A. (1999). Insignificant prevalence of antibodies to hepatitis C in a rural area of western Maharashtra. *Indian journal of gastroenterology: official journal of the Indian Society of Gastroenterology*, 18(1), 22-23.

- [10] Chevaliez, S., & Pawlotsky, J. M. (2006). HCV genome and life cycle. In *Hepatitis C viruses: genomes and molecular biology*. Horizon Bioscience.
- [11] Daniels, D., Grytdal, S., & Wasley, A. (2009). Surveillance for acute viral hepatitis—United States, 2007. *Morbidity and Mortality Weekly Report: Surveillance Summaries*, 58(3), 1-27.
- [12] Eyre, N. S., Helbig, K. J., & Beard, M. R. (2014). Current and future targets of antiviral therapy in the hepatitis C virus life cycle. *Future Virology*, 9(11), 947-965.
- [13] Honda, M., Ping, L. H., Rijnbrand, R. C., Amphlett, E., Clarke, B., Rowlands, D., & Lemon, S. M. (1996). Structural requirements for initiation of translation by internal ribosome entry within genome-length hepatitis C virus RNA. *Virology*, 222(1), 31-42.
- [14] Irshad, M., Joshi, Y. K., Acharya, S. K., & Tandon, B. N. (1994). Prevalence of hepatitis B virus infection in healthy persons in North India. *National Medical Journal of India*, 7, 210-210.
- [15] Irshad, M., & Agarwal, S. K. (1998). HCV infection in Delhi, India. *Hepatology research*, 11(2), 129-132.
- [16] Kar, P., Budhiraja, S., Narang, A., & Chakravarthy, A. (1997). Etiology of sporadic acute and fulminant non-A, non-B viral hepatitis in north India. *Indian journal of gastroenterology: official journal of the Indian Society of Gastroenterology*, 16(2), 43-45.
- [17] Khaja, M. N., Madhavi, C., Thippavazzula, R., Nafeesa, F., Habib, A. M., Habibullah, C. M., & Guntaka, R. V. (2006). High prevalence of hepatitis C virus infection and genotype distribution among general population, blood donors and risk groups. *Infection, Genetics and Evolution*, 6(3), 198-204.
- [18] Kim, S. R., Kudo, M., Hino, O., Han, K. H., Chung, Y. H., & Lee, H. S. (2008). Epidemiology of hepatocellular carcinoma in Japan and Korea. *Oncology*, 75(Suppl. 1), 13-16.
- [19] Kohli, A., Shaffer, A., Sherman, A., & Kottlilil, S. (2014). Treatment of hepatitis C: a systematic review. *Jama*, 312(6), 631-640.
- [20] Kumar, A., Sharma, K. A., Gupta, R. K., Kar, P., & Chakravarti, A. (2007). Prevalence & risk factors for hepatitis C virus among pregnant women. *Indian Journal of Medical Research*, 126(3), 211.
- [21] Lavanchy, D. (2009). The global burden of hepatitis C. *Liver international*, 29, 74-81.
- [22] Mann, A. G., Trotter, C. L., Adekoyejo Balogun, M., & Ramsay, M. E. (2008). Hepatitis C in ethnic minority populations in England. *Journal of viral hepatitis*, 15(6), 421-426.
- [23] Memon, M. I., & Memon, M. A. (2002). Hepatitis C: an epidemiological review. *Journal of viral hepatitis*, 9(2), 84-100.
- [24] Mukhopadhyaya, A. (2008). Hepatitis C in India. *Journal of biosciences*, 33(4), 465-473.
- [25] Panigrahi, A. K., Panda, S. K., Dixit, R. K., Rao, K. V., Acharya, S. K., Dasarathy, S., & Nanu, A. (1997). Magnitude of hepatitis C virus infection in India: prevalence in healthy blood donors, acute and chronic liver diseases. *Journal of medical virology*, 51(3), 167-174.
- [26] Parthiban, R., Shanmugam, S., Velu, V., Nandakumar, S., Dhevahi, E., Thangaraj, K., ... & Thyagarajan, S. P. (2009). Transmission of hepatitis C virus infection from asymptomatic mother to child in southern India. *International Journal of Infectious Diseases*, 13(6), e394-e400.
- [27] Pathak, S., & Chandrashekhar, M. (2013). Transfusion transmissible infections—seroprevalence among blood donors in a tertiary care hospital of Delhi. *Asian journal of transfusion science*, 7(2), 116.

- [28] Phukan, A. C., Sharma, S. K., Das, H. K., & Mahanta, J. (2001). HCV activity in an isolated community in north east India. *Indian journal of pathology & microbiology*, 44(4), 403-405.
- [29] Pourhoseingholi, M. A., Fazeli, Z., Zali, M. R., & Alavian, S. M. (2010). Burden of hepatocellular carcinoma in Iran; Bayesian projection and trend analysis. *Asian Pac J Cancer Prev*, 11(4), 859-62.
- [30] Sharma, S. D. (2010). Hepatitis C virus: molecular biology & current therapeutic options. *Indian J Med Res*, 131(1), 17-34.
- [31] Singh, P., Kaur, R., & Kaur, A. (2014). Frequency distribution of Hepatitis C virus in different geographical regions of Punjab: Retrospective study from a tertiary care centre in North India. *Journal of natural science, biology, and medicine*, 5(1), 56.
- [32] Sood, A., Midha, V., Bansal, M., Sood, N., Puri, S., & Thara, A. (2012). Perinatal transmission of hepatitis C virus in northern India. *Indian Journal of Gastroenterology*, 31(1), 27-29.
- [33] Sood, A., Sarin, S. K., Midha, V., Hissar, S., Sood, N., Bansal, P., & Bansal, M. (2012). Prevalence of hepatitis C virus in a selected geographical area of northern India: a population based survey. *Indian Journal of Gastroenterology*, 31(5), 232-236.
- [34] Thurner, C., Witwer, C., Hofacker, I. L., & Stadler, P. F. (2004). Conserved RNA secondary structures in Flaviviridae genomes. *Journal of General Virology*, 85(5), 1113-1124.
- [35] Uddin, G., Shoeb, D., Solaiman, S., Marley, R., Gore, C., Ramsay, M., ... & Thomas, H. C. (2010). Prevalence of chronic viral hepatitis in people of south Asian ethnicity living in England: the prevalence cannot necessarily be predicted from the prevalence in the country of origin. *Journal of viral hepatitis*, 17(5), 327-335.
- [36] Weiner, A. J., Kuo, G., Lee, C., Rosenblatt, J., Choo, Q. L., Houghton, M., ... & Saracco, G. (1990). Detection of hepatitis C viral sequences in non-A, non-B hepatitis. *The Lancet*, 335(8680), 1-3.
- [37] Zaghoul, H., & El-shahat, M. (2014). Recombinase polymerase amplification as a promising tool in hepatitis C virus diagnosis. *World Journal of Hepatology*, 6(12), 916.