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# Full Length Research Paper

# Occurrence of antibodies against hepatitis C virus (HCV) among alcoholics

J. A. Ndako\*, O. A. Olabode, G. O. N. Echeonwu, J. Chukwuekezie, C. C. Ebo and E. A. Salihu

Department of Virology, Federal College of Veterinary and Medical Laboratory Technology, Vom, Nigeria.

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Studies have shown that hepatitis C virus (HCV)-infected alcoholics have more severe biochemical and histological evidence of liver disease than anti-HCV-negative patients. One possible mechanism for the increased liver damage is that alcohol may have a stimulatory effect on HCV replication. This study was therefore carried out to investigate the seroprevalence of HCV virus among alcoholics. Two-hundred and seventy (270) alcoholics and fifty (50) control subjects at selected locations in Jos South local government area (LGA), of Plateau State were screened for HCV antibodies using grand rapid diagnostic test strip. Structured questionnaire was employed to obtain demographic data of study subjects. Overall, the prevalence of HCV infection was found to be 45(16.7%) in response to alcoholics while the non-alcoholic (control) subjects recorded 3 (6.0%) positivity, [( $x^2 = 3.765$ ); P > 0.05]. Gender consideration showed that females subjects had a higher prevalence of 25 (9.3%) compared to males with 20 (7.4%) among the alcoholic subjects. The 19 - 30 years age group recorded the highest seroprevalence of 14 (5.2%) to the HCV,  $[(x^2 = 4.757); P > 0.05]$ , while no significant difference was observed among positive subjects screened for serum aminotranferase levels in relation to age and gender. Available evidence from this study indicates that alcoholic consumption is a strong determinant of HCV infection. Drastic measures at creating awareness and the need for routine screening among alcoholics should be given due consideration. However, future HCV studies need to quantify concurrent alcoholic consumption to further our understanding of the total burden of illness from alcohol associated HCV in the community.

**Key words:** Alcoholics, hepatitis c virus, occurrence.

#### INTRODUCTION

The hepatitis C virus (HCV) is a small (50 nm in size) enveloped, single-stranded, positive sense RNA virus in the family *Flaviviridae*. HCV mainly replicates within hepatocytes in the liver. HCV virus is a blood-borne, infectious viral disease (Ryan and Ray, 2004). The infection can cause liver inflammation that is often asymptomatic, but ensuing chronic hepatitis can result later in cirrhosis (fibrotic scarring of the liver) and liver cancer. Studies have shown that HCV accounts for between 75 to 95% post transfusion hepatitis (Zuckerman and Thomas, 1993). Approximately, 170 million people world wide are infected with the disease (Zignego et al., 2006).

**Abbreviations: HCV, Hepatitis C virus; HCC, hepatocellular carcinoma.** 

Prevalence of HCV is considerably higher in developing countries; it reaches about 4 to 6% in selected population in parts of African and Middle East (Campbell et al., 2005). Almost one—third of alcoholics with clinical symptoms of liver disease have been infected with HCV which is four times the rate of HCV infection found in alcoholics who do not have liver disease (Coelho–Little et al., 1995; Mendenhall et al., 1991; Takase et al., 1993).

HCV is transmitted primarily through contaminated blood and less effectively through human bodily secretions. HCV has been detected in saliva, urine, semen and ascetic fluid (Campbell et al., 2006). People with HCV infection often experience mild symptoms and conesquently does not seek treatment (Ryan et al., 2004). Patients infected with HCV, have an 80 - 85% chances that the infection will persist, hence progressing to chronic HCV infection with multiple manifestation. The most common is progressive liver disease associated with inflammation and fibrosis while in some, it progresses to

<sup>\*</sup>Corresponding author. E-mail: ndakoj@yahoo.co.uk.

cirrhosis (Mann's, 2001). Several factors may accelerate the progression of hepatitis C, including older age at the time of infection and excessive alcohol consumption (Poynard et al., 2001).

The diagnosis of hepatitis C is rarely made during the acute phase of the disease because the majority of people infected experience no symptoms during this phase of the disease. Those who experience acute phase symptoms are rarely ill enough to seek medical attention. The diagnosis of chronic phase hepatitis C is also challenging due to the absence or lack of specificity of symptoms until advanced liver disease develops, which may not occur until decades into the disease (Johnson et al., 1993). Hepatitis C testing begins with serological blood tests used to detect antibodies to HCV. Anti-HCV antibodies can be detected in 80% of patients within 15 weeks after exposure, in > 90% within 5 months after exposure and in > 97% by 6 months after exposure. Overall, HCV antibody tests have a strong positive predictive value for exposure to the hepatitis C virus, but may miss patients who have not yet developed antibodies (seroconversion), or have an insufficient level of antibodies to be detectable (Zignego et al., 2006).

Starting HCV therapy early in the disease stage may present a better opportunity to achieve undetectable HCV viral load or a sustained virological response. Response to HCV treatment may be better in early disease, due in part to perhaps less mutations and damage to the liver having occurred and because the person's immune system may be able to be more responsive to the virus and to treatment. Heavy alcohol use can be detrimental to HCV-infected patients' long-term response to interferon therapy. It is likely that alcohol affects HCV treatment effectiveness both because drinking tends to interfere with patients' adherence to therapy and because alcohol interferes with interferon therapy's antiviral actions (Watanabe et al., 2003; Scott et al., 2006). There is no vaccine against HCV, majorly due to the high mutability of the HCV genome which enables it escapes detection by the antibodies, and this mutating ability of the virus makes vaccine development difficult. Lack of knowledge of any protective immune response following HCV infection also impedes vaccine research. In the absence of a vaccine, all precautions to prevent infection must be taken.

This study was embarked upon to determine the prevalence of HCV antibody among alcoholic subjects at selected locations in Jos South local government area (LGA) of Plateau State. The study is also aimed to further create awareness to the public and other health care providers on the importance of HCV screening among the general public.

## **MATERIALS AND METHODS**

#### Study area

This cross-sectional study was conducted in Vwang community in

Jos South local government area of Plateau State. The choice of this area is based on the life style of which includes high alcohol intake, especially the local brews who are mostly farmers.

#### Study population

The Vwang community is approached through the village 'head' who later informed his subjects to participate in the study. Only those between the ages of 15 years and above were asked to participate.

#### Questionnaire

The questionnaire administered to the subjects was designed to collect socio— demographic information about the participants and factors that might predispose them to the infectious agents. All the subjects screened completed an informed consent form.

#### Sample collection

From all the patients that completed the questionnaire, 3 ml of blood was collected from each patient via venous puncture technique using sterile syringe after swabbing the area with 70% alcohol. The serum was separated by centrifugation into a cryovial tube and stored at -20 °C in the deep freezer prior use.

#### Reagents/chemicals

The HCV one step hepatitis C virus test strip is a rapid chromatographic immunoassay for the qualitative detection of antibody to hepatitis C virus in serum or plasma. The test strip is a qualitative, membrane based immunoassay for the detection of antibody to HCV in serum or plasma. The membrane is coated with recombinant HCV antigen on the test line region of the strip. When testing the serum or plasma specimen reacts with the protein coated particles. The mixture migrates upward on the membrane chromatographically by capillary action to react with recombinant HCV antigen on the membrane and generate a colored line. The presence of this colored line indicates a positive result, while its absence indicates a negative result.

#### Statistical analysis

The data obtained was analyzed using the chi-square test method.

### **RESULTS**

Table 1 shows the distribution of HCV among alcoholics and non alcoholic subjects. Highest prevalence of 45 (16.7%) was recorded out of a total of 270 alcoholics screened, while 3 (6.0%) was recorded among the non alcoholic (control) subjects screened.

From Table 2, the highest prevalence of 14 (5.2%) was recorded among subjects aged 19 - 30 years, while the lowest prevalence of 4 (1.5%) was recorded among those aged 51 - 60 years. Table 3 shows the prevalence of HCV in relation to gender. Highest prevalence of HCV in alcoholics was obtained among the female subjects with 25 (9.3%) while male subjects recorded 20 (7.4%).

Table 1. Overall distribution of HCV infection amongst subjects (alcoholics and non-alcoholics) screened.

Status of subjects screened.	No. of	No. of	No. of
	Subjects screened	Positive subjects	Negative subjects
Alcoholics	270 (84.4%)	45 (14.1%)	225 (70.3%)
Non alcoholics (control group)	50 (15.6%)	3 (0.90%)	47 (14.7%)
Total	320 (100.0%)	48 (15.00%)	272 (85.00%)

 $<sup>\</sup>chi^2 = 3.765$ , df = 1, P value = 0.05.

Table 2. Age distribution of HCV infection amongst (alcoholics) subjects screened.

Age range	Total No. of subjects screened	Total No. of positive subjects	Total No. of negative subjects
19 - 30	120	14 (7.2%)	106 (32.1%)
31 - 40	71	12(5.2%)	59 (22.6%)
41 - 50	50	10 (3.2%)	40 (16.7%)
51 - 60	15	4 (0.6%)	11 (6.8%)
61 - 70	14	5 (0.3%)	9 (1.6%)
Total	270	45 (16.7%)	225 (83.3%)

 $<sup>\</sup>chi^2 = 4.757$ , df = 5, P value = 0.446.

Table 3. Distribution of HCV prevalence based on gender amongst (alcoholics) subjects screened.

Gender	No. of subjects screened	No. of Positive subjects	No. of Negative subjects
Males	114	20 (7.4%)	139 (51.5%)
Females	111	25 (9.3%)	131 (48.5%)
Total	225	45 (16.7%)	270 (100.0%)

 $<sup>\</sup>chi^2 = 0.734$ , df = 1, P value = 0.391.

Table 4. Risk factors based on clinical history of (alcoholic) subjects screened.

Risk factors	Total No. of subjects	Total No. positive	Total No. negative
History of transfusion with blood	50	4 (1.5%)	46 (17.0%0
History of Surgical operation	30	2 (0.7%)	28 (10.4%)
Total	80 (29.6%)	6 (2.2%)	74 (27.4%)

 $<sup>\</sup>chi^2 = 0.048$ , df = 1, P value = 0.826.

Risk factors put into consideration showed that subjects with history of blood transfusion recorded 4 (1.5%) prevalence out of the 50 responses obtained in that regard.  $X^2$  analysis indicated no significant difference between transfused subjects and those not transfused [with P value of 0.826 that is, (P > 0.05)], similarly risk factors based on subjects with history of surgery showed a record of 2 (0.7%) prevalence out of the 30 alcoholic subjects considered (Table 4).

The serum aminotransferase level recorded was based on the 45 (16.7%) HCV positive subjects. Of these, 29 (10.7%) showed a slight elevation of liver enzyme (transaminases), while 16 (5.9%) recorded normal

enzyme level, out of which, subjects aged 19 - 30 years recorded 9 (3.3%) while those aged 51 - 60 had 3 (1.1%) with elevated enzyme level as evidenced in Table 5.

The serum aminotransferase level recorded among the male positive subjects showed an elevation of liver enzyme among 14 (5.2%) subjects, compared to female subjects with 17 (6.3%) that recorded an abnormal enzyme level as evidenced in Table 6.

#### **DISCUSSION**

HCV is one of the major causes of chronic liver disease

Table 5. Determination of serum transaminase level (%) on positive subjects in r	elation
to age distribution.	

Age	Total non of subjects	Total No. abnormal	Total No. normal
19 - 30	14	9 (3.3%)	5 (1.9%)
31 - 40	12	8 (2.9%)	4 (1.5%)
41 - 50	10	6 (2.2%)	4 (1.5%)
51 - 60	4	3 (1.1%)	1 (0.4%)
61 - 70	5	3 (1.1%)	2 (0.7%)
Total	45	29 (10.7%)	16 (5.9%)

 $\chi^2$  = 3.835, df = 12, P value = 0.986.

**Table 6.** Determination of serum transaminase level on positive subjects in relation to gender distribution.

Gender	Total No. subject	Total No. abnormal	Total No. normal
Males	20	14 (5.2%)	9 (3.3%)
Females	25	17 (6.3%)	8 (2.9%)
Total	45	31 (11.5%)	17 (6.2%)

 $\chi^2$  = 1.376, df = 3, P value = 0.711.

carcinoma (HCC) and hepatocellular worldwide (Campbell et al., 2006). Numerous risk factors contribute to HCV acquisition and multiple risk factors may be present in a single individual. In the present study, anti-HCV antibodies were detected in 16.7% among alcoholics. However, a very similar prevalence (16.0%) was found in a recent review of published data involving 799 unselected individuals with alcohol abuse, (Heintges and Wands, 1997). Previous studies conducted in Brazil also found anti- HCV antibodies in 12 to 16% of unselected alcoholic patients (Cavazzola et al., 1994). In contrast, serum markers of HCV infection were detected in up to 36% of Brazilian patients with alcoholic cirrhosis (Strauss et al, 1992). In a similar work done by Robert et al. (2003), a prevalence of 2 - 14% was reported. This figure obtained in this study is therefore alarming and calls for concern.

The 19-30 years age group recorded the highest infection rate in the communities studied. This is in agreement with the work of Amina et al. (2004) who stated that subjects with 20-24 years mean age group had the highest HCV transmission prevalence rate. One could argue that the average age of the positive subjects in this study is relatively high, since younger people are more predisposed to several social lifestyles.

Females are more infected than males in this study. The rate of infection among these subjects based on gender as shown on Table 3 shows a P-Value of 0.391 which also shows no significant difference; this however does not correspond with previous result which said that men have HCV infection than women (Poynard et al., 2001). However, from this research work, women have the highest prevalence, which may due to the fact

that women in these localities consume the local brew as stimulants before embarking on their activities on the farm. Other factors like manicuring, sharing of sharp objects like razor and nail files, could be other possible predisposing factors.

Risk factors put into consideration showed that subjects with history of blood transfusion recorded 4(1.5%) prevalence out of the 50 responses obtained in that regard.  $X^2$  analysis indicated no significant difference between transfused subjects and those not transfused [with P value of 0.826 that is, (P > 0.05)]. This result agrees with the research done by Alter (1999), where he found that HCV infection can easily be trans-mitted through blood transfusion and that surgical operation can be a predisposed fact when equipment used are not probably sterilized. With these alarming facts and estimates, there are needs for routine screening to enable early diagnosis and treatment of the virus and to further prevent transfusion of infected blood and blood products.

#### Conclusion

Excessive alcohol consumption among patients infected with chronic hepatitis C is likely to result in more severe liver injury, promoting cirrhosis and increasing the risk for development of liver cancer (specifically, hepatocellular carcinoma). Although the mechanisms by which chronic hepatitis C progresses to more severe liver disease in alcoholic patients have not been clearly established. They may include an alcohol—induced increase in viral replication, rapid mutation of HCV leading to greater viral complexity, increased liver—cell death and inflammatory

response, suppression of immune responses, accumulation of fat in the liver and accumulation of excess iron in body tissues. Due to the unavailability of vaccine for HCV infection at the moment coupled with the cost effect of such drugs, a preventive measures should be vigorously pursued as part of public awareness.

The basic means of preventing HCV transmission is by ensuring that infectious materials from infected persons or carriers are kept safe from getting in contact with persons susceptible to HCV infection. Adequate precaution should be taken when handling people known or presumed to be infected since their blood and body fluid can establish a state of infection. Promotion of behavioural changes among the general public should be encouraged. Health care workers are urged to ensure safe injection practices. Risk reduction counseling is encouraged especially among the alcoholic.

#### **REFERENCES**

- Alter MJ (1999). Hepatitis B virus infection in the United States. J. Hepatol., 31(1): 88-91.
- Campbell J, Hagan H, Latka M, Garfein R, Golub E, Coady M, Thomas D, Strathdee (2006). High prevalence of alcohol use among hepatitis C virus antibody positive injection drug users in three US cities." Drug Alcohol Depend 81(3): 259-65.
- Cavazzola LT, Arruda CA, Reckziegel R, Gr"uber AC, Galperim B, Maia CR, Barros SGS (1994). Frequency of positive antibodies against the hepatitis "C" virus in alcoholics in Brazil–A pilot study (abstract). Hepatol. 19: 511.
- Heintges T, Wands JR (1997). Hepatitis C virus: epidemiology and transmission. Hepatology, 26: 521-526.
- Johnson R, Gretch Ds, Yamabe H, Hart J, Bacchi C, Hartwell P, Couser W, Corey L, Wener M, Alpers C (1993). Membranoproliferative glomerulonephritis associated with hepatitis C virus infection. N. Engl. J. Med. 328(7): 465-470.

- Poynard T, Bedossa P, Opolon P (2001). Natural history of liver fibrosis progression in patients with chronic hepatitis C. The OBSVIRC, METAVIR, CLINIVIR, and DOSVIRC groups. Lancet., 349: 825–832.
- Ryan KJ, Ray CG (2004). Sherris Medical Microbiology, 4th ed., McGraw Hill, pp. 551–552.
- Scott J, McMahon B, Bruden D, Sullivan D, Homan C, Christensen C, Gretch D (2006). "High rate of spontaneous negativity for hepatitis C virus RNA after establishment of chronic infection in Alaska Natives". Clin. Infect. Dis., 42 (7): 945-52.
- Strauss E, Sá MFG, Gayotto LCC, Takada N, Takada A (1992). Relationship of serum gammaglobulin levels with hepatitis C markers in alcoholic cirrhosis and hepatosplenic schistosomiasis [abstract]. Hepatol., 16: 594.
- Takase S, Takada N, Sawada M, (1993). Relationship between alcoholic liver disease and HCV infection. Alcoh., 1: 77–84.
- Watanabe H, Saito T, Shinzawa H, Okumoto K, Hattori E, Adachi T, Takeda T, Sugahara K, Ito J, Saito K, Togashi H, Suzuki R, Hayashi M, Miyamura T, Matsuura Y, Kawata S (2003). "Spontaneous elimination of sesrum hepatitis C virus (HCV) RNA in chronic HCV carriers: a population-based cohort study". J. Med. Virol., 71(1): 56-61.
- Zignego AL, Ferri C, Pileri SA, Caini P, Bianchi FB (2006). For the Italian Association of the Study of Liver (A.I.S.F.) Commission on Extrahepatic Manifestations of HCV infection. "Extrahepatic manifestations of Hepatitis C Virus infection: A general overview and guidelines for a clinical approach". Dig. Liver Dis.: E-publication.
- Zuckerman AJ, Thomas HC (1993). Viral hepatitis, Scientific basis and clinical management. pp. 229-326.