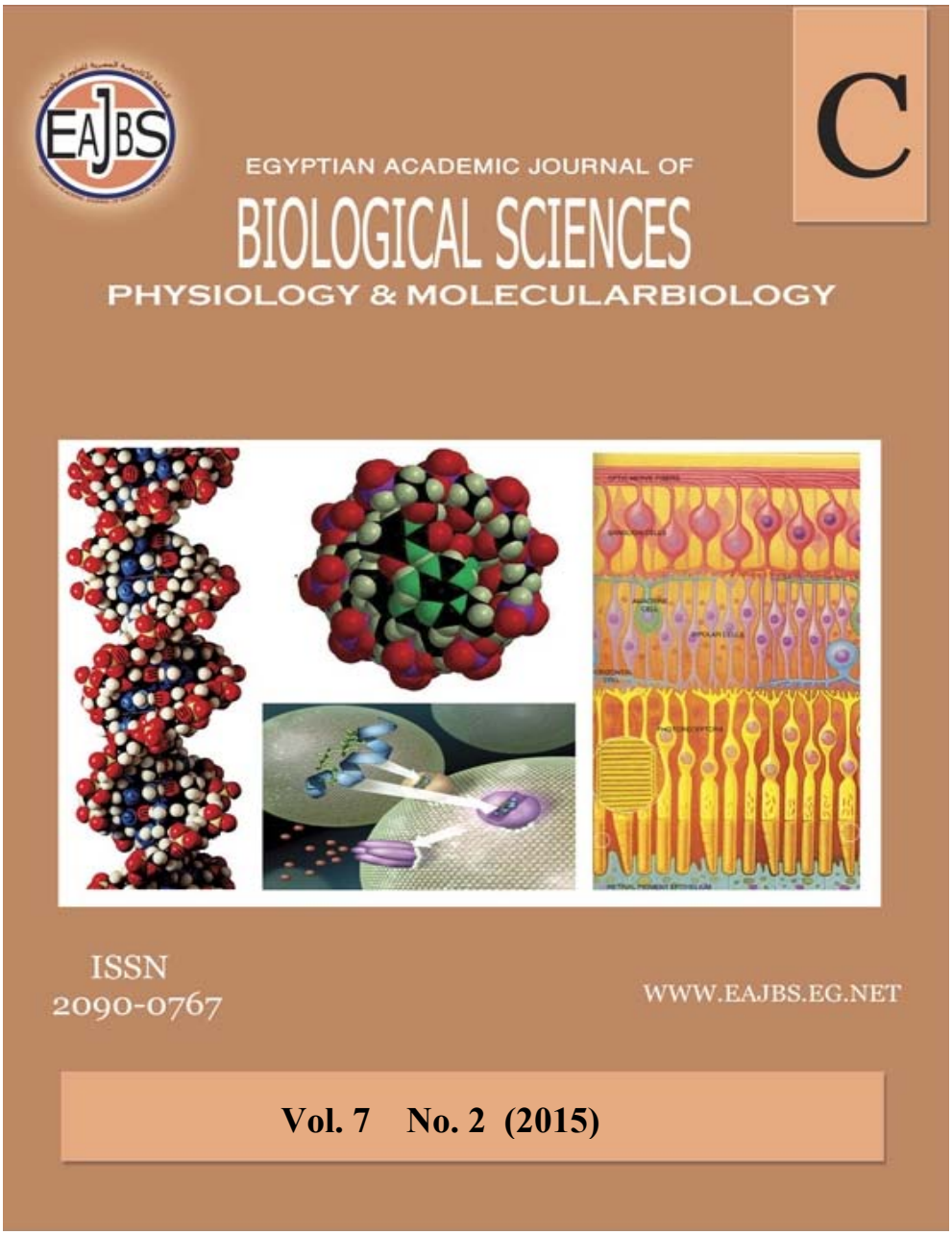

Egyptian Academic Journal of Biological Sciences is the official English language journal of the Egyptian Society for Biological Sciences, Department of Entomology, Faculty of Sciences Ain Shams University .

Physiology & molecular biology journal is one of the series issued twice by the Egyptian Academic Journal of Biological Sciences, and is devoted to publication of original papers that elucidateimportant biological, chemical, or physical mechanisms of broad physiological significance.

www.eajbs.eg.net

Citation :*Egypt.Acad.J.Biolog.Sci. (C.physiology and Molecular biology) Vol.7(2)pp1-9(2015)*

Egypt. Acad. J. Biolog. Sci., 7(2): 1 -9 (2015)

Egyptian Academic Journal of Biological Sciences
C. Physiology & Molecular Biology
ISSN 2090-0767
www.eajbs.eg.net

Immunoassay of anti-HCV and seroepidemiological surveillance of hepatitis C virus infection

Sarah Y. Ahmed[1,2], Sabry A.A. El-Sayed[3], Wajed Al Shammari [2]

1- Bacteriology, Mycology and Immunology Dept., Faculty of Veterinary Medicine, Zagazig University, P.O. Box 44511, Zagazig, Egypt.
2- Department of Clinical Laboratory Sciences, College of Applied Medical Science, University of Hail, Hail 2440 Saudi Arabia.
3- Department of Nutrition and Clinical Nutrition, Faculty of Veterinary Medicine, Zagazig University, P.O. Box 44511, Zagazig, Egypt.

ARTICLE INFO

Article History
Received: 30/7/2015
Accepted:25/8/2015

***Keywords*:**
Seroprevalence
HCV
Anti-HCV
ELISA
Hail

ABSTRACT

Infection with hepatitis C virus (HCV) is a common global cause of chronic liver disease, which is also true in Saudi Arabia. HCV prevalence in Saudi Arabia varies in different provinces being highest in the Western and Southern provinces. Most of the studies among blood donors documented a decrease in HCV prevalence, probably due to increase awareness and improved socioeconomic status. The present study was designed to investigate the prevalence of hepatitis C infection among general population of Hail region, Saudi Arabia and to determine titer of anti-HCV in patient serum. A total of 100 participants in he study survey with average age of 18-63 years, were preformatted a questionnaire including the demographic data, socio-economic status and medical history. Three mL of venous blood were collected from 37 participants, in a container with strict aseptic precautions. Collected serum samples were used for serological evaluation of anti-HCV infection, by using ELISA. Among the participants in the study survey ,HCV positivity percentage was found to be (1%) (One /100) in our study. Thirty seven serum samples were examined for anti-HCV, thirty of them were positive with titer ranged from (10.512 to 1859.395 IU/ml), while seven samples were negative, In conclusion, the obtained data revealed, low prevalence of HCV in Hail region, Saudi Arabia.

INTRODUCTION

Hepatitis C virus (HCV) is the most common blood borne infection in worldwide (Alter,1999). Chronic HCV can lead to hepatic fibrosis, cirrhosis and hepatocellular carcinoma (HCC) and is the leading cause of liver transplantation nationwide (Lauer and Walker, 2002). HCV is the major cause of post-transfusion and community-acquired non-A, non-B hepatitis (NANBH), HCV is a single-stranded, positive-sense RNA virus of the family *Flaviviridae* (Kuo *et al*.,1989, Alter *et al*.,1992). There are six HCV genotypes and more than 50 (as many as 90) subtypes (Alter *et al*.,1992).

These genotypes can differ by up to 50% of their nucleotide sequences, and the virus has a high propensity to mutate. In a large population-based study, 1.8% of a large household-based sample was positive for anti-HCV antibody (2.3% in adults 20 years or older), which would translate into an estimated 3.9 million infected persons in the U.S (Alter *et al*., 1999). 74% of them had viremia, indicating chronic infection (an estimated 2.7 million). Studies have reported a higher prevalence of anti-HCV antibodies, with the highest rates consistently in intravenous drug users (Alter,1997 and Bell *et al*.,1990). The incidence of HCV infection has fallen since the 1990s. The yearly incidence of HCV infection was estimated to average 230,000 cases per year in the 1980s, but by 2001 had declined to 25,000 cases per year. In a French population-based study, 24% (17/72) were aware that they were seropositive for HCV infection (Dubois *et al*.,1997). HCV infection is a leading cause of complications from chronic liver disease in the United States. HCV-related end-stage liver disease is now the most common indication for liver transplantation among American adults, accounting for over 30% of cases, and there was a 5-fold increase in the number of patients with HCV who underwent liver transplantation between 1990 and 2000 (Busch, 2001; Kim, 2002; Seeff *et al*.,1992; Seeff *et al*.,1994). Chronic HCV infection can also cause morbidity in the absence of cirrhosis or other serious complications. HCV infection without cirrhosis is associated with significantly worse quality of life measures and symptoms (primarily fatigue) compared to the general population, though confounding factors such as intravenous drug use and other co-morbid conditions have not been well-controlled in studies(Kenny-Walsh,1999; Koff, 1999 and Foster *et al*.,1998). One study in women with a low prevalence of intravenous drug use, however, found high levels of psychological distress and impaired quality of life in women with anti-HCV antibodies that did not correlate to the presence or absence of chronic infection (Coughlan *et al*., 2002). Hepatitis C virus (HCV) has ability to persist in most immunocompetent adults. Although HCV activates an innate immune response, it employs an elaborate set of mechanisms to evade interferon (IFN)-based antiviral immunity. By comparing innate and adaptive immune responses to HCV with those to hepatitis A and B viruses, prolonged innate immune activation by HCV impairs the development of successful adaptive immune responses (Su and Barbara,2014). Hepatitis C virus have the capacity to generate a strong cellular immune response against the virus and avoid persistent infection, and perhaps do so repeatedly after re-exposure(Lisa *et al*.,2006).HCV-specific antibodies generally develop 2–8 weeks after exposure in humans and remain throughout the course of chronic infection. Antibody levels are generally low, with limited affinity maturation or isotype switching (Chen *et al*.,1999). In those who clear the virus, antibody levels gradually diminish in titer, with approximately 50% of individuals having no detectable antibodies by 20 years after primary infection (Takaki *et al*., 2000 and Villano *et al*., 1999). Although this humoral immunity may be part of an effective immune response in the minority of individuals who clear the primary infection, antibody generation is not an absolute requirement for viral clearance(Abe *et al*.,1992; Farci *et al*.,1996; Mehta *et al*., 2002). Studies have shown that individuals with hypogammaglobulinaemia clear the virus in proportions similar to the general population (Bjoro *et al*.,1994; Razi *et al*.,2001 and Chapel *et al*., 2001), and recovery does not correlate to anti-HCV

antibody titers or to the levels of antibodies directed to the envelope glycoprotein's, E1 and E2 (Bassett *et al*., 1998; Kobayash *et al*.,1997 and Grellier *et al*.,1997), although it should be noted that these studies did not assess neutralizing antibodies.

MATERIAL AND METHODS

Study setting and design

This study was carried out over a Five-month period at a Maternity hospital and King Khalid hospital in Hail region, Saudi Arabia.

Participants:

A total of 100 participants in the agegroup of 18-63 years, attending the clinic at Maternity and King Khalid hospital, Hail, Saudi Arabia,between October 2013 to February 2014 were studied. The approval of the institute's was obtained priorto the sample collection; informed writtenconsent was obtained from all the participants. Detailed clinical historyand conventional laboratory investigations wereconducted.

Questionnaire:

A preformatted questionnaire including the demographic data, socio-economic status and medical history wascompleted during patients follow-up.The questionnaires included questions on Age, gender, Family income, occupation and educational level and patient history.

Statistical analysis:

Statistical analysis was performed by using SPSS for Windows version 12.0 (SPSS INC., Chicago, III., USA). Data was presented as mean ± SD. Chi-square analysis μ2 was used in findings on comparison of HCV infection positivity according to individual characteristics. Evaluation was carried out at the 95-99% confidence interval and P< 0.05 was considered statistically significant.

Detection of anti-HCV by using ELISA technique:

Enzyme-linked immunoassay (ELISA) or enzyme immunoassay (EIA), detects antibodies against recombinant HCV antigens. "First generation" ELISA tests used a single antigen; later-generation tests added additional antigens (39,41).

Serum samples:

Three mL of venous blood were collected from 37 participants, in a container with strict aseptic precautions. The serum was used for serological evaluation of ani-HCV infection.

ELISA kits for detection of anti-HCV(Monolisa™ Anti-HCVPLUSV ersion 2)

An indirect immunoenzymatic technique allowing the detection of the antibodies associated with an infection by Hepatitis C virus in patient serum. Monolisa™ Anti-HCV PLUS Version 2 is based upon the use of a solid phase prepared with purified antigens, ٣recombinant proteins produced by *E.coli* from clones selected in the non structural area (NS3 and NS4) andin the structural area of the hepatitis C virus genome.

RESULT

I-Incidence of HCV in Hail region February to December 2013

During February to December 2013, (165 blood donors) were enrolled in this study,79% out of them are positive for HCV Assay, As show in **Figure (1)**.

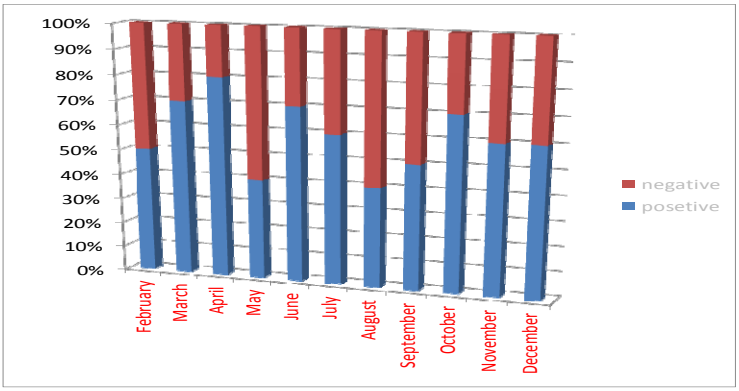

Fig. 1: Incidence of HCV infection in Hail region.

Figure (1): Incidence of HCV in Hail region February to December 2013.

II- Seroprevalence of TORCH in Hail region

Among the participants, HCV positivity percentage was found to be (1%) (One /100) in our study. It was found that, the average age of the patient was (18 to 63 years old), the majority came from Hail city, as shown in Table (1).

Table 1: Comparison of demographic characteristics and HCV positivity.

Demographic and socioeconomic characteristics		**HCV infection + (n=1) N0.(%)[1]**	**HCV infection – (n=99) N0.(%)[2]**	χ^2	**P***
Age	18-25 26-30 31-35 36-40 41-45 46-63	0(0%) 0(0%) 0(0%) 0(0%) 1(1%) 0(0%)	37(37%) 22(22%) 15(15%) 12(12%) 6(6%) 7(7%)	13.42	0.02
Gender	Male Female	1(1%) 0(0%)	70(70%) 29(29%)	0.41	0.52
Family situation	Unmarried married	0(0%) 1(1%)	66(66%) 33(33%)	1.96	0.16
Occupation	Job No job	1(1%) 0(0%)	41(41%) 58(58%)	1.4	0.24
Educational level	Primary high school university	1(1%) 0(0%) 0(0%)	15 (15%) 18(18%) 67(67%)	5.4	0.06

1) Percentage in HCV (+)s.
2) Percentage in HCV (-)s.
*Percentage statistically significant difference (P>0.05).

Comparison of Knowledgement about HCV infection and HCV positivity

Obtained data revealed that, only 1% of participants know about *HCV* and methods of infection, as shown in Table (2).

Table 2: Comparison of Knowledgement about HCV infection and HCV positivity.

Knowledgement about HCV infection[1)]		**HCV infection+ (n=1) no. (%)[2]**	**HCV infection – (n= 99) no. (%)[3]**	χ^2	**P***
I know about *HCV* Infection		1(1%)	29(29%)	2.36	0.12
My Knowledgement about HCV infection from	Journal Physician T.V.	0(0%) 1(1%) 0(0%)	17(17%) 34(34%) 27(27%)	1.27	0.53
I Know about method of HCV infection		1(1%)	76(76%)	0.30	0.58

1) Ones who answered yes in Knowledgement about HCV infection were considered.
2) Percentage in HCV (+)s.
3) Percentage in HCV (-)s.
*Percentage statistically significant difference (P>0.05).

Comparison of Knowledgement about symptoms of HCV infection

Among the participants, only 1% of them know about symptoms of chronic phase of HCV infection, as shown in Table (3).

Table 3: Knowledgement about symptoms of HCV infection.

Symptoms of HCV developed[1)]	HCV infection (+) No=1 (%)[2)]	HCV infection (–) No=99 (%)[3)]	χ2	P*
Acute phase: tiredness and jaundice (yellowish of the skin and eye), fever and headache.	0(0%)	65(0%)	1.88	0.17
Chronic phase: Jaundice, fatigue, abdominal pain, joint pain, developed to liver cirrhosis and cancer	1(1%)	35(0%)	1.79	0.18

1) Ones who answered yes in Knowledgement about HCV infection were considered.
2) Percentage in HCV (+)s.
3) Percentage in HCV (-)s.
*Percentage statistically significant difference (P>0.05).

Comparison of Knowledgement about serological and molecular tests for detection of HCV infection:

Obtained results illustrated that, only 1% of participants Know and conduct serological test for detection of HCV, as shown in Table (4).

Table 4: Knowledgement about serological and molecular tests for detection of HCV infection.

Serological test for detection of HCV[1)]	HCV infection + No=1 (%)[2)]	HCV infection - No=99 (%)[3)]	χ^2	P*
I Know and conduct test for detection of HCV.	1(1%)	23(23%)	3.19	0.07
I conduct HCV ELISA test for detection of anti HCV in serum.	1(1%)	23(23%)	3.19	0.07
I conduct PCR (Polymerase chain reaction) for detection of HCV genes.	1(1%)	0(0%)	100	0.00

1) Ones who answered yes in Knowledgement about serological and molecular tests for detection of HCV infection were considered.
2) Percentage in HCV (+)s.
3) Percentage in HCV (-)s.
*Percentage statistically significant difference (P>0.05).

Detection of anti-HCV in patients serum by using ELISA Kits:

Thirty seven serum samples were examined for anti-HCV, thirty of them were positive withtiter ranged from (10.512 to 1859.395 IU/ml), while seven samples were negative, as shown in Table (5) and Fig. (2).

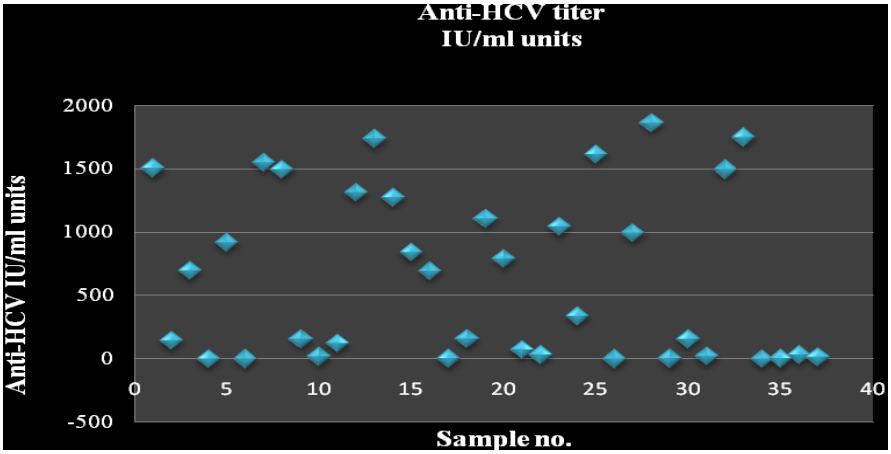

Fig. 2: Titer of anti-HCV in patient serum.

Table 5: Anti-HCV titer in patients serum.

Sample number	Sex	Anti-HCV titer IU/ml units	Result
1	F	1502.087	positive
2	F	141.504	positive
3	F	694.401	positive
4	F	-2.309	negative
5	F	915.564	positive
6	F	-1.026	negative
7	F	1548.256	positive
8	F	1492.050	positive
9	F	155.242	positive
10	F	17.424	positive
11	F	120.367	positive
12	F	1311.389	positive
13	F	1734.939	positive
14	F	1271.242	positive
15	F	838.034	positive
16	F	691.593	positive
17	F	0.000	negative
18	F	156.755	positive
19	F	1104.633	positive
20	F	786.958	positive
21	F	69.500	positive
22	F	29.963	positive
23	F	1042.405	positive
24	F	334.436	positive
25	F	1612.491	positive
26	F	-0.770	negative
27	F	992.222	positive
28	F	1859.395	positive
29	F	-1.539	negative
30	F	154.336	positive
31	F	17.936	positive
32	F	1494.058	positive
33	F	1748.991	positive
34	F	-2.052	negative
35	F	-1.539	negative
36	F	29.707	positive
37	F	10.512	positive

DISCUSSION

Incidence of HCV infection blood donors in Hail region was 79%.Hepatitis C virus prevalence rates are estimated to range between 5.5% in Africa, 4.6% in the Eastern Mediterranean region, 4% in the Western Pacific region, 2% in South East Asia, 1.7% in the United States of America (USA), and 1% in Europe (Shobokshi *et al*.,2003). There has been very high prevalence rate of HCV reported in Egypt in the past 28% (Sy and Mazen, 2006). The study showed 1.3% seroprevalence of HCV Ab among general population in central region of Yemen (Ibb city). The frequency of HCV Ab seropositivity was found to be higher than that reported in southern part of Yemen (Aden city) 0.6% and lower than northern part of Yemen (Sana'a city) 2.3% and southern islands (Socotra island) 5.1%(Bajubair *et al*., 2008). Such differences in prevalence rates may be explained by differences in health resources and educational levels in different regions in Yemen or due to methodological differences between studies.(Mahaba *et al*. 1999) described the prevalence of HCV in 8862 subjects

from the Hail region, Saudi Arabia. The overall prevalence was found to be 5.1%, with a very high prevalence among Egyptian expatriates (26%).

The seroprevalence of HCV positivity in Hail region was 1%, with average age of patient (18 to 63 years old) and these result was agreed with (Mahdi *et al*., 2000 and Madani, 2007) who mentioned that, prevalence assessed from Saudi blood donor screening centers indicates HCV infection rates of 0.4-1.1%. Declines in HCV prevalence rates were also noted in the blood bank database of King Khalid University Hospital in Riyadh, from 0.58% in 1996 to 0.08% in 2006. A summary report compiled by the WHO mentions 437,292 official reports of HCV infections among persons living in the KSA, giving an estimated prevalence of about 1.8% (WHO,2009).

The obtained data revealed that titer of anti- HCV in patient's serum was ranged from (10.512 to 1859.395 IU/ml). (Khayriyyah *et al*., 2013) mentioned that the number of persons with anti-HCV in the world has increased from an estimated 122 million (P: 2.3%, 95% UI: 2.1%-2.5%) in 1990 to an estimated 184 million (P: 2.8%, 95% UI: 2.6%-3.1%) in 2005. However, given the cross-sectional nature of prevalence data, this global rise in prevalence and changes observed in East Asia, Western Europe, and West sub-Saharan Africa may reflect changes in compositional data or global shifts in age patterns rather than changes in disease epidemiology.

CONCLUSION

In conclusion, the results of the present study hint that, seroprevalence of HCV positivity in Hail region was 1%, the relatively low prevalence of HCV in our study group may well be due to an improvement in the control the testing for infectious diseases in the home countries of the expatriates.

ACKNOWLEDGMENTS

The authors would like to thank the University of Hail for supporting this research. The authors also express gratitude to Dr. Eyad Al Shammari, Dean of applied medical science college for his encouragement.

CONFLICT OF INTEREST

The author has no commercial relationships and no potential conflicts of interest.

REFERENCES

Abe K, Inchauspe G, Shikata T, Prince AM.(1992): Three different patterns of hepatitis C virus infection in chimpanzees. *Hepatology*, 15: 690–95.

Alter HJ. (1995): To C or not to C: these are the questions. Blood.85(7):1681–95.

Alter MJ, Kruszon-Moran D, Nainan OV. *et al*.(1999): The prevalence of hepatitis C virus infection in the United States, 1988 through 1994. N Engl J Med. 341(8):556–62.

Alter MJ, Margolis HS, Krawczynski K. *et al*. (1992): The natural history of community-acquired hepatitis C in the United States. The Sentinel Counties Chronic non-A, non-B Hepatitis Study Team. N Engl J Med.327(27):1899–905.

Alter MJ. (1997):Epidemiology of hepatitis C. Hepatology. 26(3 Suppl 1):62S–65S.

Bassett SE, Brasky KM, Lanford RE.(1998): Analysis of hepatitis C virus-inoculated chimpanzees reveals unexpected clinical profiles. *J. Virol.* 72: 2589–99.

Bajubair, M. A.; A. A. Elrub, and G. Bather, (2008): "Hepatic viral infections in Yemen between 2000–2005,"Saudi Medical J. 29(6):871–874.

Bell J, Batey RG, Farrell GC. *et al*.(1990): Hepatitis C virus in intravenous drug users. Med J Aust. 153(5):274–6.

Bjoro K, Froland SS, Yun Z, Samdal HH, Haaland T.(1994): Hepatitis C infection in patients with primary hypogammaglobulinemia after treatment with contaminated immune globulin. *N. Engl. J. Med.* 331: 1607–11.

Busch MP.(2001):Insights into the epidemiology, natural history and pathogenesis of hepatitis C virus infection

from studies of infected donors and blood product recipients. Transfus Clin Biol. 8(3):200–6.

Chapel HM, Christie JM, Peach V, Chapman RW.(2001): Five-year follow-up of patients with primary antibody deficiencies following an outbreak of acute hepatitis C. *Clin. Immunol.* 99: 320–24.

Chen M, Sallberg M, Sonnerborg A *et al*. (1999):Limited humoral immunity in hepatitis C virus infection. *Gastroenterology*. 116: 135–43.

Coughlan B, Sheehan J, Hickey A. *et al*. (2002): Psychological well being and quality of life in women with an iatrogenic hepatitis C virus infection. Br J Health Psychol. 2002;7:105–116.

Dubois F, Desenclos JC, Mariotte N. *et al*. (1997): Hepatitis C in a French population-based survey, 1994: seroprevalence, frequency of viremia, genotype distribution, and risk factors. The Collaborative Study Group. Hepatology. 25(6):1490–6.

Farci P, Shimoda A, Wong D. *et al*. (1996): Prevention of hepatitis C virus infection in chimpanzees by hyperimmune serum against the hypervariable region 1 of the envelope 2 protein. *Proc. Natl Acad. Sci. USA*.93: 15 394–9.

Foster GR, Goldin RD, Thomas HC.(1998): Chronic hepatitis C virus infection causes a significant reduction in quality of life in the absence of cirrhosis. Hepatology.1998;27:209–212.

Grellier L, Brown D, Power J, Dusheiko G.(1997): Absence of anti-envelope antibodies and clearance of hepatitis C virus in a cohort of Irish women infected in 1977. *J. Viral Hepat.*4: 379–81.

Kenny-Walsh E.(1999): Clinical outcomes after hepatitis C infection from contaminated anti-D immune globulin. Irish Hepatology Research Group. N Engl J Med. 340(16):1228–33.

Khayriyyah Mohd Hanafiah, Justina Groeger, Abraham D. Flaxman, and Steven T. Wiersma, (2013): Global Epidemiology of Hepatitis C Virus Infection: New Estimates of Age-Specific Antibody to HCV Seroprevalence. Hepatology, 57(4):1333-1342.

Kim,W. R. (2002): The burden of hepatitis C in the United States. Hepatology. 2002; 36:S30–S34.

Kobayashi M, Tanaka E, Matsumoto A, Ichijo T, Kiyosawa K. (1997): Antibody response to E2/NS1 hepatitis C virus protein in patients with acute hepatitis C. *J. Gastroenterol. Hepatol.* 12: 73–6.

Koff RS.(1999):Impaired health-related quality of life in chronic hepatitis C: the how, but not the why. Hepatology. 29(1):277–279.

Kuo G, Choo QL, Alter HJ. *et al*.(1989): An assay for circulating antibodies to a major etiologic virus of human non-A, non-B hepatitis. Science. 244 (4902): 362–374.

Lauer G.M. and Walker B. D. (2001): Hepatitis C virus infection. N Engl J Med 2001; 345: 355.

Lisa N Elliott, Andrew R Lloyd, John B Zieglerand Rosemary A Ffrench (2006): *Protective immunity against hepatitis C virus infection.* Immunology and Cell Biology.84: 239–249.

Madani T. A. (2007): Hepatitis C virus infections reported in Saudi Arabia over 11 years of surveillance. Ann Saudi Med. 27:191–4.

Mahaba H, el-Tayeb Ael-K, Elbaz H (1999): The prevalence of antibodies to hepatitis C virus in Hail region, Saudi Arabia. J Egyp Pub Health Assoc 74: 69-80.

Mehdi SR, Pophali A, Al-Abdul Rahim KA. (2000): Prevalence of hepatitis B and C and blood donors. Saudi Med J. 21: 942–947.

Mehta SH, Cox A, Hoover DR *et al*. (2002): Protection against persistence of hepatitis C. *Lancet*; 359: 1478–83.

Razvi S, Schneider L, Jonas MM, Cunningham-Rundles C. (2001): Outcome of intravenous immunoglobulin-transmitted hepatitis C virus infection in primary immunodeficiency. *Clin. Immunol.* 101: 284–8.

Seeff LB, Buskell-Bales Z, Wright EC. *et al*. (1992): Long-term mortality after transfusion-associated non-A, non-B hepatitis. N Engl J Med. 327:1906–1911.

Seeff LB. (1994): Mortality and morbidity of transfusion-associated type C hepatitis: an NHLBI multi-center study [abstract] Hepatology. 20(4):204A.

Shobokshi, O.A.; F. E. Serebour, A. Z. Al Drees, A. H. Mitwalli, A. Qahtani, and L.

I. Skakni, (2003): "Hepatitis C virus seroprevalence rate among Saudis," Saudi Medical J. 24(2): S81–S86.

Su-Hyung Park and Barbara Rehermann (2014): Immune Responses to HCV and Other Hepatitis Viruses. Immunity, Vol 40(1):13–24

Sy. T. and M. Mazen Jamal (2006): "Epidemiology of hepatitis C virus (HCV) infection," International J. Medical Sci., 3(2):41–46.

Takaki A, Wiese M, Maertens G *et al*. (2000): Cellular immune responses persist and humoral responses decrease two decades after recovery from a single-source outbreak of hepatitis C. *Nat. Med.* 6: 578–82.

Villano SA, Vlahov D, Nelson KE, Cohn S, Thomas DL.(1999): Persistence of viremia and the importance of long-term follow-up after acute hepatitis C infection. *Hepatology*. 29: 908–14.

WHO. World Health Organization Regional Committee for the Eastern Mediterranean; (2009): The growing threats of hepatitis B and C in the Eastern Mediterranean Region: A call for action.. [Last accessed on 2012 Oct 10]. Available from http://applications.emro.who.int/docs/EM_RC56_3_en.pdf .

ARABIC SUMMARY

الكشف المناعى للأجسام المضادة لفيروس الإلتهاب الكبدى الوبائى سى والإنتشار السيرولوجى والمصلى للإصابة بالألتهاب الكبدى

سارة يوسف [١،٢]، صبرى السيد [٣]، واجد الشمرى [٢]

١- قسم البكتريولوجيا والفطريات والمناعة،كلية الطب البيطرى-جامعة الزقازيق-صندوق بريد ٤٤٥١١ الزقازيق-مصر

٢- قسم المختبرات-كلية العلوم الطبية التطبيقية- جامعة حائل-صندوق بريد ٢٤٤٠ –حائل-المملكة العربية السعودية

٣- قسم التغذية والتغذية الإكلينيكية،كلية الطب البيطرى-جامعة الزقازيق-صندوق بريد ٤٤٥١١ الزقازيق-مصر.

الإصابة بفيروس الإلتهاب الكبدى الوبائى سى شائع فى بلدان العالم ويسبب التهاب الكبد المزمن الموجود فى المملكة العربية السعودية. نتشار فيروس التهاب الكبدى سى فى السعودية منتشر فى محافظات المملكة السعودية فى الغرب والجنوب. معظم الدراسات بين متبرعى الدم سجلت نقص فى إنتشار عدوى فيروس الإلتهاب الكبدى سى فى المملكة العربية السعودية، والتى من المحتمل أن تكون بسب التحسن فى الأوضاع الإجتماعية والإقتصادية. صممت هذه الدراسة للكشف عن إنتشار فيروس الإلتهاب الكبدى سى بين سكان منطقة حائل- المملكة العربية السعودية. لتحديد مستوى الأجسام المضادة لفيروس التهاب الكبدى سى فى مصل المصابين تم تعبئة إستبيان بواسطة ١٠٠ شخص متوسط أعمارهم من ١٨-٦٣ سنة. وتضمن الإستبيان أسئلة عن الوضع المادى والإجتماعى والحالة الصحية والمرضية. تم تجميع ٣مل من دماء ٣٧ شخص وتم إجراء الإختبارات السيرولوجية لتقييم مستوى الأجسام المضادة لفيروس التهاب الكبدى سى بإستخدام جهاز الإليزا. وتبين أنه توجد حالة واحدة مصابة من بين ١٠٠ حالة تم فحصها فى الدراسة. ومن بين ٣٧ عينة مصل تم فحصها تم كشف ٣٠ عينة تحتوى على الأجسام المضادة لفيروس التهاب الكبدى سى بمعدل يتراوح من ١٠٥١٢-1859.395وبينما توجد ٧ عينات مصل لم يكتشف فيها الأجسام المضادة لفيروس الالتهاب الكبدى سى. وأظهرت النتائج وجود معدل منخفض من الإصابة باللإلتهاب الكبدى سى فى منطقة حائل-المملكة العربية السعودية.