PREVALENCE OF HEPATITIS B SURFACE ANTIGEN IN TALLAFAR POPULATION

Dr. Abdul-Aziz Ahmed Aziz Assisst. Prof. Medical College , University of Kufa.
Dr. Mohammad Ali Abdul-Ghafoor C.A.B. Physician ,General hospital of Tallafar.

الخلاصة
الا[a] داف:لمعرفة مدى انتشار المستضد السطحي **ب(**HBsAg**)** لألتهاب الكبد الفروسي لدى مواطني مدينة تلعفر.
الطريقة: أجريت [a]هذه الدراسة في مستشفى تلعفر العام ، نينوى ، العراق . خلال الفترة من حزيران٢٠٠٧ ولغاية تموز ٢٠٠٨ . حيث تم التحري عن المستضد السطحي **ب (**HBsAg**)** لألتهاب الكبد الفروسي لدى١٥٩٤ شخصا(١١١٠ ذكر و٤٨٤ انثى).وتم اجراء الفحوصات بواسطة الأنزيم المرتبط بمعايرة المناعة(ELISA).
النتائج: اظهرت النتائج ان ٢٨(١,٧%) شخصا من مجموع ١٥٩٤ اشخاص يحملون المستضد السطحي **ب** للفايرس وبواقع ١٩ ذكر(١,٧%) و٩انثى(١,٨%).
خاتمة:تؤكد نتائج الدراسة ان نسبة انتشار المستضد السطحي **ب** لدى مواطني تلعفر اقل من مستوياتها في الدول المجاورة وان اكثر الاشخاص المعرضين للاصابة [a]هم العاملين في المؤسسات الصحية ومتبرعي الدم.مما يستدعي الا[a]تمام بتعقيم الادوات والاجهزة الطبية وتوخي الحذر في التعامل مع الحالات المصابة وتطعيم العاملين في المؤسسات الصحية.

ABSTRACT
Objective: The present study was designed to assess the prevalence of the hepatitis B surface antigen (HBsAg) among Tallafar population, this information in turn could be used to identify the high risk population which could be offered the hepatitis vaccine.

Methods: The present study was conducted on 1594 subjects (1110 males and 484 females) at Tallafer General Hospital, Nineva, Iraq, from 1st June2007 to 30th July 2008. HBsAg was determined by Enzyme Linked Immunosorbent Method (ELISA).

Results: Approximately 1594 subjects were tested during the period of study. HBsAg was detected in 28 subjects given an overall prevalence of 1.75% .Of the positive cases 19(1.7%) were males and 9 (1.8%) females.
Conclusion: The finding of the present study indicate that the prevalence of the hepatitis B surface antigen (HBsAg) among Tallafar population is less than the surrounding countries. Health care workers and blood donors may act as important reservoirs of hepatitis B virus . Approaches to controlling hepatitis B would be to decrease the carriage rate by vaccination of peoples at risk, adequate sterilization, and by screening blood donors for all marker of hepatitis B virus infection using more sensitive and specific techniques, in order to minimize post-transfusion hepatitis.

INTRODUCTION

The earliest record about Hepatitis B virus (HBV) was published by MacCallum in 1947[1] . However, the virus was not discovered until 1965[2]. In 1970, Dane DS, and others discovered the virus particle by electron microscopy[3]. By the early 1980s the genome of the virus had been sequenced[4] and the first vaccines were being tested[5]. More than 350 million peoples worldwide have been infected with HBV[6] .The prevalence of HBV infection range from less than 2% in low prevalence areas such as the continental United States and Western Europe [7] up to 20% in high prevalence areas such as Africa, China and South East Asia[8,9]. The prevalence of HBsAg in our country is range from 2 to 3% in the normal population and slightly higher among health professional[10]. At particular risk of becoming carrier are infant born to hepatitis B infected women, injection drug abuse, persons with multiple sex partners, and health care workers owing to blood exposure and accidental needle injury[7,11]. Initial infection with HBV is most often asymptomatic but, in 0.1% to 1% of acute cases, it may develop into fulminant hepatic failure, which is often fatal in the absence of liver transplantation[12]. After the acute episode, which may or may not be symptomatic, hepatitis B becomes chronic in 2 to 10% of cases. There is the risk that it may lead to cirrhosis and/or hepatocellular carcinoma. The current challenge is vaccination of at-risk subjects, screening, and management of infected patients in order to prevent complications[13]. The aim of the present study is to assess the prevalence of the HBsAg among Tallafar population, this information in turn could be used to identify the high risk population which could be offered the hepatitis vaccine.

MATERIALS & METHODS

The present study was conducted on 1594 subjects (1110 males and 484 females) at Tallafer General Hospital, Nineva, Iraq, from 1st June2007 to 30th July 2008.Their ages range from 20 -55 years. The total population included in the study were divided into the following groups:

Group I: included 280 subjects(186 males and 94 females) who admitted to the medical ward or out patient clinic of Tallafar General Hospital as suspected cases of chronic liver disease .

GroupII: included 305 subjects (222 males and 83 females) working in laboratories , blood bank, and health care worker in Tallafar General hospital .

GroupIII: included 679 subjects (407 males and 272 females)who attending public health laboratories for routine viral examination prior to marriage or cardiac catheterization.

GroupIV: included 330 blood donor subjects(295males and 35 females).

For each subject participate in the study, information on demographic data, past medical history (accident, surgical operation, blood donation, blood transfusion) were collected. Five ml of venous blood was obtained from a suitable forearm vein into plain tube, the sera then separated and kept in capped plastic tubes in deep freeze (-20 ^{o}C) until analysis. HBsAg was determined by Enzyme Linked Immunosorbent Method (ELISA) utilizing kits provided by Abbott GMbH Diagnostika, Wies baden, Germany.

RESULTS

The prevalence of HBsAg among group I, group II, group III and groupIV are shown in Table 1.Approximately 1594 subjects were tested during the period of study. HBsAg was detected in 28 subjects given an overall prevalence of 1.75% . Of the positive cases 19(1.7%) were males and 9 (1.8%) females.

Table 1: Prevalence of HBsAg in male and female subjects of different groups included in the study.

Study groups	Number of subjects			Number of +ve HBsAg			Percent (%)
	Male	Female	Total	Male	Female	Total	
group I	186	94	280	5	3	8	2.85
group II	222	83	305	6	3	9	2.95
group III	407	272	679	3	2	5	0.74
group IV	295	35	330	5	1	6	1.81
Total	1110	484	1594	19	9	28	1.75

Concerning the prevalence of HBsAg in different age groups the study show higher prevalence in younger age groups in comparison with old age groups as shown in table 2.

Table 2: Prevalence of HBsAg in different age groups

Age group (years)	Number of subjects			Number of +ve HBsAg			Percent (%)
	Male	Female	Total	Male	Female	Total	
10-20	110	67	177	2	0	2	1.13
20-30	266	119	385	6	4	10	2.59
30-40	361	128	489	7	4	11	2.25
40-50	300	92	392	3	1	4	1.02
50-60	113	38	151	1	0	1	0.76

DISCUSSION

HBsAg is the earliest serologic marker to appear in subject with HBV infection, it produced by infected liver cells and can be detected in the serum before the onset of symptoms. The level of HBsAg start to decline after the acute onset of illness and usually undetectable after 3-6 months. Persistance of HBsAg beyond 6 months indicates continued viral replication and sign of chronicity[12]. The present study showed that the prevalence rate in the studied population was only 1.75% . The prevalence of HBsAg was more common in females (1.85%) than males(1.71%) , it was also more in younger age group than older, this is presumably due to lesser contact with the disease. In general the overall prevalence (1.75%) was lower than that of other developing countries. The prevalence of HBsAg range from (9.8%) in Saudia Arabia to 20% in some countries of Asia and Africa[14,15]. The seroprevalence of HBV varied widely among different countries depending on modes of transmission, human behavior, and geographically difference, age , sex, population samples, the sensitivity of the tests used to detect HBsAg, social and religious background[16,17,18]. Concerning the community of Tallafar where the study conducted the peoples are strictly follow the

order and behavior of Islam religion concerning drug addiction and other prohibited behaviors. The study also showed high prevalence among patients with suspected chronic liver disease, this finding appear to be logical since viral hepatitis is on of the main precursor of chronic liver disease[19,20]. The peoples who are at high risk for disease include health professional specially those who are working in private and official laboratories, blood bank workers, hemodialysis group ,physician and nurses[17]. The main route of infection include accidental inoculation with infected blood, needle stick , contact of samples with broken skin, dental extraction using contaminated equipment, penetrating injuries when removing metal foil caps from vials of freeze-dried material, and use of syringes and needles for dispensing[16]. Given its prevalence and capacity for chronicity , hepatitis B is now one of the leading cause of chronic liver disease. These facts put the physicians , health care worker and health authorities face to face with a serious challenge. The finding of the present study indicate that health care workers and blood donors may act as important reservoirs of hepatitis B virus . Approaches to controlling hepatitis B would be to decrease the carriage rate by vaccination of peoples at risk[21,22,23] , adequate sterilization, and by screening blood donors for all marker of hepatitis B virus infection using more sensitive and specific techniques, in order to minimize post-transfusion hepatitis[24,25].

REFERENCES

1. MacCallum, F.O. (1947).Homologous serum hepatitis. Lancet; 2:691.
2. Alter HJ, Blumberg BS (1966). "Further studies on a "new" human isoprecipitin system (Australia antigen)". Blood ; 27 : 297–309.
3. Dane DS, Cameron CH, Briggs M (1970). "Virus-like particles in serum of patients with Australia-antigen-associated hepatitis". Lancet 1 (7649): 695–8.
4. Galibert F, Mandart E, Fitoussi F, Tiollais P, Charnay P (1979). "Nucleotide sequence of the hepatitis B virus genome (subtype ayw) cloned in E. coli". Nature ; 281 : 646–50.
5. Nothdurft HD, Dietrich M, Zuckerman JN, etal. (2002). A new accelerated vaccination schedule for rapid protection against hepatitis A and B. Vaccine ;20:1157-62.
6. Lee WM.(1997).Hepatitis B virus infection.New Engl J Med 337:1733-1745.
7. Redd JT, Baumbach J, Kohn W, et al. (2007). "Patient-to-patient transmission of hepatitis B virus associated with oral surgery". J Infect Dis 195 : 1311–4..
8. Alter MJ.(2003). "Epidemiology and prevention of hepatitis B". Semin. Liver Dis. 23: 39–46.
9. Chen CH, Yang PM, Huang GT,etal.(2007). Estimation of seroprevalence of hepatitis B virus and hepatitis C virus in Taiwan from a large -scale survey of free hepatitis screening participants. J Formos Med Assoc ;106:148-155.
10. Omar AR .(1984). Proceedings of the Second National Symposium on Viral Hepatitis I. Baghdad, Iraq.
11. Grawford JM.(2005).The liver and biliary tract. In Kumar V, Abbas AK, Fausto N.(eds) Robbinis and Cotran Pathologic basis of disease(7th ed.pp 877-936). Philadelphia, Elsevier Saunders.
12. Porth CM.(2007). Essential of Pathophysiology (2nd ed., pp.638-643).Philadelphia: Lippincott William & Wilkins.
13. Shamliyan TA, MacDonald R, Shaukat A, et al.(2009). Antiviral therapy for adults with chronic hepatitis B: a systematic review for a National Institutes of Health Consensus Development Conference. Ann Intern Med;150:111-124.

14. El-Hazmi MM.(2004).Prevalence of HBV, HCV, HIV-1,2 and HTLV-I/II infection among blood donors in teaching hospital in central region of Saudi Arabia. Saudi Med J ;25:26-33.
15. Kahattak MF, Salamat N, Bhatti FA,etal.(2002). Seroprevalence of hepatitis B ,C and HIV in blood donors in northern Pakistan. J Pak Med Assoc ;52:398-402.
16. Gibas A, Blewett DR, Schoenfold DA,etal.(1992). Prevalence and incidence viral hepatitis in health worker in the prehepatitis B vaccination era. Am J Epidemiol ;136: 603-610.
17. Lok ASF, McMahon BJ.(2004)American Association for the Study of Liver Diseases, Chronic hepatitis B: update of recommendations. AASLD practice guideline. Hepatology;39:1-5.
18. Marsano LS.(2003). Hepatitis .Primary Care ;30:81-107.
19. Viola LA, Barrison JG, Coleman JC, etal.(1982). Natural history of liver diseases in chronic hepatitis B surface antigen carriers. Survey of 100 patients from Great Britain. Lancet ;ii:1156-1159.
20. Beasley RP, Hwang LY, Lon Cs, etal.(1981). Hepatocellular carcinoma and hepatitis B virus. A prospective study of 22,707 men in Taiwan. Lancet;ii:1127-1132.
21. Szumuness W, Steven CE, Zang EA, etal.(1981). A controlled clinical trial of the efficacy of hepatitis B vaccine (Hepatax B). Final report. Hepatology ;1:377-385.
22. Lemon SM, Thomas DL.(1997).Vaccine to prevent viral hepatitis. New Engl J Med ; 336:196-203.
23. Advisory Committee on Immunization practice.(1999).Notice to reader update: Recommendations to prevent hepatitis B transmission. United State. MMWR Morbidity & Mortality, weekly report 48(2) ;33-34.
24. Gerlich WH, Luer W, Thomssen R, etal.(1980). Diagnosis of acute an inapparent hepatiris B virus infection by measurement of IgM antibody to hepatitis B core antigen. J Infect Dis ;142:95-101.
25. Soldan K, Davison K, Dow B.(2005). Estimates of the frequency of HBV, HCV, and HIV infectious donations entering the blood supply in the United Kingdom, 1996 to 2003. Euro Surveill ;10:17.