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Seroprevalence of Hepatitis B markers in Iraqi patients with chronic Hepatitis B virus

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Abstract: *To determine the seroprevalence of hepatitis B markers in chronic hepatitis B patients, 75 patients with chronic hepatitis B virus of ages (8-70) years have been investigated and compared with 50 apparently healthy individuals. All the studied groups were carried out to measure (HBsAg), (HBsAb), (HBeAg), (HBeAb), and (Total HBcAb) by Enzyme linked immunosorbent assay (ELISA) technique. The percentage distribution of HBsAg was (86.67%) and HBsAb was (1.33%) in sera of CHB patients and there were a highly significant differences (P<0.01) when compared between studied groups, while, the percentage distribution of HBeAg was (22.67%) in sera of CHB patients and the significant represent the difference in distribution of HBeAg as infection but not as HBsAg distribution. Whereas the percentage distribution of HBeAb was (50.67%) in sera of CHB patients and there were no significant differences (P>0.05) when compared between studied group. The statistical analysis also demonstrated that the percentage distribution of total HBc Ab was (73.33%) in sera of CHB patients and there were a highly significant differences (P<0.01) when compared between studied group. These results indicated that HBsAg was the predominant markers in patients with chronic hepatitis B virus*.

Keyword: *Chronic hepatitis B virus, HBsAg, HBsAb, HBeAg, HBeAb, and Total HBcAb*

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I. Introduction

Hepatitis B is an infectious inflammatory illness of the liver caused by the hepatitis B virus (HBV) that affects hominoidea, including humans, originally known a serum hepatitis. The disease has caused epidemics in parts of Asia and Africa, and it is endemic in China (1)

Hepatitis B virus, abbreviated HBV, is a species of the genus *Orthheapdna- virus*, which is likewise a part of the *Hepadnaviridae* family of viruses (2), 42 nm DNA virus and has a partially double-stranded DNA genome and contains a core antigen (HBcAg) surrounded by a shell containing surface antigen (HBsAg) .The virus is divided into four major serotypes (adr, adw, ayr, ayw) based on antigenic epitopes presented on its envelope proteins, and into eight genotypes (A-H) according to overall nucleotide sequence variation of the genome. The genotypes have a distinct geographical distribution and are used in tracing the evolution and transmission of the virus (3)., Chronic hepatitis B infection is a major cause of chronic liver disease world wide, each phase of hepatitis B infection stimulates distinct viral kinetics and host immune responses resulting in liver damage and hepatic fibrosis (4). The first phase of chronic hepatitis Immune Tolerant Phase: is more frequent and prolonged in perinatally-infected patients via perinatal transmission from HBeAg-positive mothers (5) or those infected in the early years of life. It is characterized by HBeAg positivity, high levels of HBV replication, normal or low levels of aminotransferases, and mild or no liver involvement. Immune Reactive Phase, This phase may occur after several years of immune tolerance and is more frequently observed in patients infected during adulthood. It is characterized by HBeAg positivity, a lower level of HBV replication, increased or fluctuating levels of aminotransferases, (6) . Inactive HBV Carrier State**:** This phase may follow seroconversion from HBeAg positive to anti-HBe antibody positive. It is characterized by very low or undetectable serum HBV DNA levels and normal aminotransferases (7). HBeAg-Negative Chronic Hepatitis B: This phase may follow HBeAg seroconversion during the immune reactive phase and represents a later stage in the natural history of chronic hepatitis B. It is characterized by periodic reactivation with a pattern of fluctuating levels of HBV DNA and aminotransferases and active hepatitis (8). Hepatitis B Surface Antigen (HBsAg) Negative Phase: Low levels of HBV replication may still occur in the liver after loss of HBsAg, HBsAg loss is associated with improvement in outcome with a lowered risk of cirrhosis, decompensation and hepatocellular carcinoma, immunosuppression can lead to reactivation in these patients (9,10).

The aim of present study was to determine the prevalence of hepatitis B virus markers in Iraqi patients with chronic hepatitis B virus.

II. Materials And Methods

Patient's samples:
The study was selected of 75 patients positively to chronic hepatitis B virus that attended to hepatic and gastrointestinal tract hospital from different capitals in Iraq during the period from first of November 2013 until February 2014.The ages of the total patients were ranged from (8-70) years.

Control samples:
Fifty samples of healthy individuals; 23 female and 27 male were studied as a control groups of same ages and sex. All samples were marked by the number of sample, name of patient and day of sample collection.

Blood samples collection:
Blood samples (5ml) were collected by disposable syringe into gel tubes and stand at room temperature until the coagulant was form. Then the samples were centrifuged at 3000 rpm for 5 minutes. Serum samples were dispended separated on a seven Ependroff tubes. All samples were marked by the name, day and numbering and stored at (-20˚C) until carried out to examination.

Serological markers:
All the study group were carried out to measurement of hepatitis B markers (HBsAg), (HBsAb), (HBeAg), (HBeAb), and(Total HBcAb) with the aid of commercially available Enzyme linked immunosorbent assay (ELISA)kit foresigh, USA according to the leaflets of kits (11).

Statistical analysis:
The statistical analysis used included Student t-test and Pearson chi-square test (χ^2). The Statistical Package for Social Science V.13 (SPSS) was used. A p-value <0.05 was considered statistically significant (12).

III. Results And Discussion

The percentage distribution of HBsAg was (86.67%) and HBsAb was (1.33%) in sera of CHB patients and there were a highly significant differences (P<0.01) when compared between studied groups as shown in table (1). Meanwhile, the percentage distribution of HBeAg was (22.67%) in sera of CHB patients and the significant represent the difference in distribution of HBeAg as infection but not as HBsAg distribution. Whereas the percentage distribution of HBeAb was (50.67%) in sera of CHB patients and there were no significant differences (P>0.05) when compared between studied groups as shown in table (2). The statistical analysis also demonstrated that the percentage distribution of total HBc Ab was (73.33%) in sera of CHB patients and there were a highly significant differences (P<0.01) when compared between studied groups as shown in table (3).

Table (1): The percentage distribution of HBsAg and HBsAb in sera of CHB patients**.**

Test	HBs Ag		HBs Ab	
	No.	%	No.	%
Positive	65	86.67	1	1.33
Negative	10	13.33	74	98.67
Total	75	100 %	75	100 %
Chi-square value	---	13.781 **	---	15.833 **
** (P<0.01).				

Table (2): The percentage distribution of HBeAg and HBeAb in sera of CHB patients**.**

Test	HBeAg		HBeAb	
	No.	%	No.	%
Positive	17	22.67	38	50.67
Negative	58	77.33	37	49.33
Total	75	100 %	75	100 %
Chi-square value	---	11.962 **	---	0.351 NS
** (P<0.01).				

Table (3): The percentage distribution of HBcAb in sera of CHB patients**.**

Test	Total HBcAb	
	No.	%
Positive	55	73.33
Negative	20	26.67

Total	**75**	**100 %**
Chi-square value	**---**	**11.026 ****
**** (P<0.01).**		

The results of the current study was close to the findings of other studies done by Zafar*et al* (13) who identified the percentages for HBsAg, HBeAb and HBcAb were 42.5%,50.3% and 59%, respectively, while HBeAg 31% and HBsAb was 50.6% and Chen (14) who identified percentage of HBsAg was 53.6%. Our results disagreed with Al-HajiIsa(15)who reported the percentage of HBsAg 14%,HBeAg20% and 80% for HBeAb. Wang *et al* (16) reported prevalence of HBsAg and HBeAg was 6.1% and 4.8%, respectively, while HBeAb was 89.1% and 18.8% for total HBcAb which were incompatible with our results. In Indonesia, 4.6% of the population was positive for HBsAg in 1994 and of these, 21% were positive for HBeAg and 73% for anti-HBeAb 44% and 45% of Indonesian patients with cirrhosis and HCC, respectively, were HBsAg positive. HBeAg was found in 14.3% of patients with chronic hepatitis B in Turquia (17), 30%in Campinas in southeastern Brazil (18) and 37.3% in Rio GrandedoSul in southern Brazil (19).The differences in the results may be related to the size of sample and the technical methods that were used in diagnosis of infection. Previous studies have suggested that quantitative hepatitis B surface antigen (HBsAg) is also a surrogate marker that can be used to monitor patients with CHB who are being treated (20).

There are two forms of HBsAg-one over intact virion, which includes small, medium, and large proteins in envelops and is related to viral infectivity, and another that exists as subviral particles in serum and is produced in great excess. These are predominantly S protein and, to a lesser extent, M and L protein; these are not infectious but are strongly immunogenic, stimulating antibody production (21). Antibody to surface antigen (anti-HBs) this is a protective antibody that develops with the resolution of acute infection or following the successful vaccination against HBV. Very occasionally, anti-HBs and HBsAg can be found together, which has no known clinical significance (22).Hepatitis Be antigen (HBeAg) is an accessory protein from the precore region of the HBV genome, which is not necessary for viral infection or replication .It is, however, produced during active viral replication and may act as an immunogen or tolerogen, leading to persistent infection. In HBeAg-positive patients, the HBsAg levels were distributed in a triphasic-like decline pattern by two logs across age strata. Chronic hepatitis B virus (CHB) can be broadly divided into two major forms-namely, hepatitis B virus e antigen (HBeAg) positive and HBeAg negative (23). Antibody to e antigen (anti-HBe) while, is not a protective antibody, its appearance usually coincides with a significant immune change associated with lower HBVDNA replication (<105 copies/mL or 20,000 IU/ml).The loss of HBeAg and the development of anti-HBeis termed HBeAg seroconversion, and has been used as an end-point for treatment in HBeAg-positive people, as it has been shown that seroconversion is associated with a lower risk of disease progression (24).

Antibody to core antigen (anti-HBc), the HBV core antigen is not found as a discrete protein in the serum but it is produced in the hepatocyte cytosol during HBV replication, surrounding the viral genome and the associated polymerase. It is then packaged within an envelope before secretion from the hepatocyte(22). The antibody to HBV core (anti-HBc) is an antibody to a peptide of this core protein, which has been processed within an antigen presenting cell. Anti-HBcIgG remains positive for life following exposure to HBV, however, unlike anti-HBs, anti-HBc is not a protective antibody. Most serological assays do not directly measure anti-HBcIgG, but test for total anti-HBc antibody (25).

IV. Conclusion

These results indicated that HBsAg was the predominant markers in patients with chronic hepatitis B virus.

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