

Evaluation of Hepatitis B infection in AL-Muthanna Province

Saad Awad Muzaa*¹, Noor Sami Aboud², Yasir Adil Jabbar Alabdali³

^{1,2,3} Department of Biology, College of Science, Al Muthanna University, Al Muthanna, Iraq
awadsaad860@gmail.com

Abstract

HBV (Hepatitis B Virus) is a DNA virus that was discovered in the 1960s. HBV infection can be detected via diagnostic tests, which can also be used to track the evolution of the condition. HBV DNA, HBsAg (s-antigen), and HBeAg (e-antigen) are three viral components discovered in serum samples, as well as non-viral components such as antibodies to the respective antigens, including HBsAb, and host serum transaminase (TSB, ALT, AST and ALP) levels. This study aims to compare concentration of (TSB, ALT, AST and ALP) between HBV patients and control. This study was carried out from January 2022 to April 2022. 40 patients infected with HBV and 40 controls were selected from different regions of AL-Muthanna Province in this study. The results of (TSB, ALT, AST and ALP) of infected patients with HBV and control people in different regions of Al Muthanna province showed that of (TSB, ALT, AST and ALP) concentrations were higher in Hepatitis B Virus patients than control group. The (TSB, ALT, AST and ALP) concentrations were higher in males infected with HBV than females. The rate of incidence of HBV in AL-Samawa city was higher than other regions.

Key word: Hepatitis B Virus, TSB, ALT, AST and ALP

Introduction

A DNA virus called HBV (Hepatitis B Virus) was first identified in the 1960s. The Orthohepadnavirus genus of the Hepadnaviridae family, as well as the Retroviridae family's Spumaretrovirinae subfamily, represents the only other animal virus with a DNA genome known to replicate by reverse transcription of a viral RNA intermediate, according to the ICTV classification (1). A blood-borne virus called hepatitis B is up to 200 times more infectious than HIV (2). Acute hepatitis infections have an incubation period of 1 month (4–6 weeks) to as long as 6 months after transmission as the virus progresses throughout the liver. About 65 percent of acute infections are clinically silent before treatment. Clinically discernible symptoms in the remaining cases include decreased appetite, nausea and vomiting, tiredness, and stomach pain, as well as jaundice in the more serious cases. The most typical manifestations of pro-inflammatory cytokine elevations, such as INF and TNF, are these symptoms (3). Diagnostic tests are available to identify HBV infection, and they can also be used to monitor the condition's development. Three viral components HBV DNA, HBsAg (s-antigen), and HBeAg (e-antigen) as well as non-viral elements antibodies to the corresponding antigens, including HBcAb, and host serum transaminase (ALT) levels have all been found in serum samples. Typically, HBsAg, HBsAb, and HBcAb are the three main markers that are looked for in HBsAg, HBsAb, and HBcAb detection. Core antibodies can be subtyped to distinguish between acute (IgM class) and chronic (IgG class). The most common secondary markers are HBeAg and HBeAb. However, other methods may be used, such as ALT levels and molecular testing (quantitative PCR of HBV DNA). According to a recent data-collecting study, depending on the stage (acute vs. chronic) and phase of

persistent infection, these markers' titres can change and even nearly disappear (2). Treatments So far, seven drugs have been licenced to treat chronic hepatitis B. the presence of a virus, which are alteration in biochemical indications of liver disease, which could be problematic to physicians when making a diagnosis of liver or other organ-related disorders (4).

The initial vaccine experiments, which demonstrated efficient protection, found immune protection brought on by immunisation with short HBsAg, regardless of subtype (5). On the other hand, the majority of anti-HBsAg antibodies created during the initial immune response was specific type. First-generation d-specific IgM antibodies were produced after immunising humans (6). with SHBsAg of serotype. When the reaction was broadened, somatic hypermutation and epitope maturation resulted in the inclusion of the a-determinant. Consequently, HBV serotypes could be applied to offer quicker defence. The first indication of vaccine escape mutants was identified in a study group that received genotype A vaccination inoculation in an area where genotype D predominates circulatory strain (7). Some of the metabolic pathway's end products and enzymes that are particularly susceptible to the occurrence of abnormalities may be regarded as biochemical indicators of liver dysfunction. Serum bilirubin, alanine amino transferase, aspartate amino transferase, and aspartate amino transferase are a few examples of biochemical indicators. Individuals may experience a single or conjugated SGPT (serum glutamic-pyruvic transaminase) and SGOT (serum glutaic-oxaloacetic transaminase) enzymes leak into the bloodstream when liver cells are damaged (8). The cells from the liver, heart, skeletal muscles, and red blood cells contain the highest concentrations of ALT and AST, which may be noted on lab slips with either their new and old names or just their newer ones. Patients with liver illnesses characterised by

hepatocellular damage exhibit a significant rise in transaminases in their LFTs (9). Unlike AST, which can be raised in conditions affecting other organs, ALT is largely found in the liver, with minor levels also present in the kidneys, heart, and skeletal muscle. As a result, it is a more precise sign of hepatic inflammation of body parts like the heart or muscles. (10). Aspartate transaminase, which is elevated following a myocardial infarction, acute pancreatitis, acute hemolytic anaemia, severe burns, acute renal disease, musculoskeletal disorders, and trauma, catalyses the breakdown of aspartate in the body. Oxaloacetate and glutamate are produced from aspartate and -ketoglutarate. (11). When there are risk factors for HBV infection and no other explanation for elevated enzyme levels in a patient, ALT is employed more often than AST because it is more specific for liver injury. Patients with risk factors for HBV infection and no known HBV infection are highly related with elevated aminotransferase levels.

ALP is found in the mucosal epithelia of the small intestine, the proximal convoluted tubule of the kidney, bone, liver, and the placenta, where it conducts lipid transfer and calcification. Serum ALP activity is mostly derived from the liver, with bone contributing 50% of the total; normal serum ALP levels range from 41 to 133 U/L. ALP levels in acute viral hepatitis are generally normal or slightly elevated. Hepatitis A presenting cholestasis is linked to elevated ALP with persistent itching; tumours produce ALP into plasma, and there are tumor-specific isoenzymes (12). An increase in ALP can be caused by hepatic and bone metastases, as well as other disorders such as infiltrative liver diseases, abscesses, granulomatous amyloidosis and liver disease cirrhosis, hepatitis, and congestive heart failure all have mildly elevated ALP values (13). Low levels of

ALP are present in hypothyroidism, pernicious anaemia, zinc deficiency, and congenital hypophosphatasia. The prevalence of ALP in peripheral artery disease has been found to be high, despite other common cardiovascular risk factors (14).

Materials and Methods

Patient samples

A total of 40 patients with HBV and 40 controls were selected from AL-Muthanna Province, and participated in this study from January 2022 to April 2022.

Blood samples collection

A sample of 5 ml of fresh blood was taken from the subject and placed in a gel tube. The blood was then allowed to clot at room temperature before being centrifuged at 2000 rpm for 10 minutes. The serum was then collected in a sterile tube and tested for the presence of HBsAg using an ELISA assay and (TSB, ALT, AST and ALP) tests.

Results

1-Seroprevalence of HBV infection

The results of serological examination by Enzyme Linked Immunosorbent Assay (ELISA) for detection of antigens of HBV in Al-Muthanna province showed that 40 positive Hepatitis B Virus and 40 health people.

2-Distribution of the infected patients with HBV infection according to sex

Table (1) explained distribution of HBV infection according to sex of the infected patients found (57.5%) males and (42.5%) females.

Table(1).Distribution of the infected patients with HBV infection according to the gender									
AL -Khather		AL-Warkaa		AL-Rumathya		AL-Samawa		No.ofinf.with %	Gender
%	Inf.No.	%	Inf.No.	%	Inf.No.	%	Inf.No.		
12.5	5	12.5	5	15.0	6	17.5	7	23(57.5%)	Male
10.0	4	7.5	3	10.0	4	15.0	6	17(42.5%)	Female
22.5	9	20.0	8	25.0	10	32.5	13	40	Total

Cal. χ^2 20.192 Tab. χ^2 29.488 P value: 0.995

3- Seroprevalence of HBV Infection in relation with Age

The results of seroprevalence of HBV infection by using ELISA in relation to the different age groups 20-30 years , 31-40 years ,41-50 years and 51->60 years old were 20% ,35% ,35% and 10% respectively, the highest rate of the seropositivity was in age groups 31-40 years and 41-50 years 35% and the lowest rate of seropositivity was in age groups 51->60 years old 10%. There was significant differences at ($P < 0.05$) (Table 2).

Table (2): Seroprevalence of HBV infection in different Age of patient.		
Age (years)	No. infection	% of infection
20-30	8	20%
31-40	14	35%
41-50	14	35%
51->60	4	10%
Total	40	100

4-Seroprevalence of hepatitis B virus infection in relation to the area of residence

The results of seroprevalence of HBV infection by using ELISA in relation with residence of different geographical study area, that the prevalence rate of infection in AL - Samawa , AL -Rumathya , AL -Khather and AL -Warkaa were 40%, 25%, 20% and 15% respectively. The highest percent of seropositively was in AL -Samawa 40% and the lowest was in AL -Warkaa 15%. There was significant differences at ($P < 0.05$) (Table 3).

Table (3): Seroprevalence of HBV infection according of different regions.		
City	No. Infection	%
AL-Samawa	16	40%
AL-Rumathya	10	25%
AL-Warkaa	6	15%
AL-Khather	8	20%
Total	40	100

* Significant differences at ($P < 0.05$).

5-Results of liver function test parameter detection in HBV patients according to sex

The results of liver function test parameter in seropositive patients as compared with control group. Male patients showed that the concentrations of Bilirubin (3.13 ± 0.86), AST (61.08 ± 6.79), ALT (70.88 ± 8.74) and Alklinephosphat (176.32 ± 12.47) respectively as compare with Bilirubin (0.67 ± 0.16), AST (16.00 ± 1.55), ALT (15.67 ± 1.75) and

Alklinephosphat (109.92 ± 10.42) of male control. Female patients showed that the concentrations of Bilirubin (2.96 ± 0.80), AST (61.33 ± 7.17), ALT (70.88 ± 8.74) and Alklinephosphat (174.07 ± 10.45) respectively as compare with Bilirubin (0.67 ± 0.16), AST (15.53 ± 1.82), ALT (15.40 ± 1.20) and Alklinephosphat (109.47 ± 9.69) of female control. The highest concentrations liver function parameter was in patients as compared with control (Table 4).

		TSB	ALT	AST	ALP
Male	Patients	3.13 ± 0.86	70.88 ± 8.74	61.08 ± 6.79	176.32 ± 12.47
	Control	0.67 ± 0.16	15.67 ± 1.75	16.00 ± 1.55	109.92 ± 10.42
P. value		0.001	0.003	0.0003	0.0001
Female	Patients	2.96 ± 0.80	66.87 ± 9.12	61.33 ± 7.17	174.07 ± 10.45
	Control	0.63 ± 0.14	15.53 ± 1.82	15.40 ± 1.20	109.47 ± 9.69
P. value		0.01	0.007	0.0003	0.0001

6- Results of liver function test in HBV patients according to age

The results of liver function test in seropositive patients as compared with control group showed that the concentrations of TSB (3.25), AST(61.5),

ALT(73) and ALP(179.5) of HBV patients were higher than control group in all age groups. The highest ALT concentration was in age group 51->60 and the lowest concentrate was in age group 41-50 and there was a significant difference between age group and control at ($P < 0.05$) (Table 5)

Age		TSB mg/dl	ALT U/L	AST U/L	ALP U/L
20-30	Patients	3.25 ± 0.902	73 ± 8.413	61.5 ± 7.55	179.5 ± 13.91
	Control	0.66 ± 0.15	15.73 ± 1.751	16.27 ± 1.438	110.1 ± 9.906
P. value		0.0001	0.0002	0.0001	0.0001
31-40	Patients	3.2 ± 0.874	70 ± 10.73	62 ± 7.173	178 ± 10.98
	Control	0.69 ± 0.162	15.58 ± 1.881	16.17 ± 1.586	109.3 ± 10.86
P. value		0.05	0.0001	0.0001	0.0001
41-50	Patients	2.75 ± 0.931	68.5 ± 7.148	61.5 ± 6.524	174.5 ± 11.72
	Control	0.60 ± 0.141	15.17 ± 2.483	16.17 ± 1.722	112 ± 9.055
P. value		0.001	0.0001	0.00001	0.0001
51->60	Patients	3.629 ± 0.559	75.57 ± 4.86	76.71 ± 4.03	181.6 ± 11.73
	Control	0.586 ± 0.168	16 ± 1.528	15.86 ± 1.773	108.6 ± 12.38
P. value		0.001	0.00023	0.0001	0.00001
Age P. value		0.0001	0.0001	0.0001	0.0001

Discussion

The patients with hepatitis B were distributed by sex, as indicated in table (1). The rate of infection with HBV was in male 23(57.5%) more than female 17 (42.5%). This observation is consistent with research of Xi,et.al.(15), the sex disparity of HBV-related liver diseases has been observed for a considerable and may be related to sex hormone effects rather than gender-specific behaviours or environmental influences. Sex hormones may potentially play a role in addition to the direct effects on the HBV life cycle. The advancement of related liver disorders and the immunological response to HBV infection, even it is still unknown how certain mechanisms work. While Al-Rubaye et.al.(16) revealed that no difference between males and females in HBV infection in Basra City. According to this study, there were no significant

changes in hepatitis B virus infection among age categories (Table 2). Infection rates were found to be particularly high in pateint ages groups of (31-40) and (41-50). This finding is in same line with several studies from various nations, including those by Jang et.al. (17) who found that the median age for HBV infections is 48 years old; Mese et.al. (18) who found that the mean age for HBV infection was 44.5 years old; and Hadi et.al. (19) who found that the majority of HBV infections occur in the elderly in the Baqubah City/ Iraq.

The results of the current study demonstrate that there are many infected individuals living in AL-Samawa city (urban area), as indicated in (Table 3), and these individuals appear to be similar to those who took part in prior studies such as (21)

In contrast, participants in other research who resided in rural rather than urban regions had higher

prevalence of HBV antibodies. This is in the same line with research conducted in Pakistan in 2005 by (22). Tables (4 and 5) showed that average values of enzymes liver TSB,ALT,AST and ALP in patients infected with viral hepatitis B type in both males and females in different age groups. This result is agreed with results of (22,23).The elevated level of hepatic enzymes in the blood serum is a clear indication of liver cell injury, as the study of (24) showed that the elevation of the AST, ALT and ALP is associated with the destruction or injury of hepatocytes. These enzymes are present in low concentrations in the blood serum in the normal state and when the liver is infected, the autoimmune reactions stimulated by the surface protein of the virus work to destroy the liver tissues through the process of programmed cell death, so that the hepatocyte membranes become more permeable and these enzymes leak into the blood circulation through the blood vessels and then its concentration rises in the blood serum, showed that the amounts of hepatic enzymes leaked into the blood serum change according to the patient's conditions and the amount of damage caused by the virus to the liver cell (25).

Conclusions

1. The rates of males infected with hepatitis B virus were more than in females.
2. The highest age-specific frequency in patients positive for HBV was in the age groups of 31-40 years and in age group 41-50 years.
3. The prevalence of HBV according to residence highest rate was 40% AL-Samawa city and the percent of infection in AL-Rumathya was 25%, AL-Khather 20% and lowest Al-warkaa was 15 %.
4. The concentration of (TSB, ALT, AST and ALP) in males infected with hepatitis B virus was higher than females.
5. The concentration of (TSB, ALT, AST and ALP) in HBV patients was higher than healthy people.

Reference

- 1-Norder, H., Couroucé, A. M., Coursaget, P., Echevarria, J. M., Lee, S. D., Mushahwar, I. K., & Magnus, L. O. (2004). Genetic diversity of hepatitis B virus strains derived worldwide: genotypes, subgenotypes, and HBsAg subtypes. *Intervirology*, 47(6), 289-309.
- 2-Bowyer, S.M. & Sim, J.G.M., 2000. Relationships within and between genotypes of hepatitis B virus at points across the genome: footprints of recombination in certain isolates. *Journal of General Virology*, 81(2), pp. 379
- 3-Seeger, M. W., & Sellnow, T. L. (2007). Post-crisis communication and renewal: Expanding the parameters of post-crisis discourse. *Public relations review*, 33(2), 130-134.
- 4-Shivraj, H., & Khobragade, C. N. N. (2009). Determination of nutritive value and mineral elements of some important medicinal plants from western part of India.
- 5-Schaefer, S., & Warren, J. (2005). Mean value coordinates for closed triangular meshes. In *ACM Siggraph 2005 Papers* (pp. 561-566).
- 6- Legler, G., Vasilov, R., Claesson, L., Peterson, P., & Ploegh, H. (1983). Effects of the glucosidase inhibitors nojirimycin and deoxynojirimycin on the biosynthesis of membrane and secretory glycoproteins. *The EMBO Journal*, 2(6), 823-832.
- 7-Carman, W. F., Karayiannis, P., Waters, J., Thomas, H. C., Zanetti, A. R., Manzillo, G., & Zuckerman, A. T. (1990). Vaccine-induced escape mutant of hepatitis B virus. *The lancet*, 336(8711), 325-329.
- 8-Hayashi, F., Means, T. K., & Luster, A. D. (2003). Toll-like receptors stimulate human neutrophil function. *Blood*, 102(7), 2660-2669.
- 9-Gaze, R., Carvalho, D. M. D., Santoro-Lopes, G., & Tura, L. F. R. (2013). From hepatic diseases and jaundice to viral hepatitis: the configuration of a kaleidoscope. *Revista de saude publica*, 47, 117-122
- 10- Hirotsu, Y., Moriyama, M., & Omata, M. (2019). Molecular hepatocellular carcinoma in chronic mechanisms driving progression of liver cirrhosis towards hepatitis B and C infections: a review. *International journal of molecular sciences*, 20(6), 1358
- 11- Berg, T., & Asselah, T. (2021). Perspectives on stopping nucleos(t)ide analogues therapy in patients with chronic hepatitis B. *Antiviral Research*, 185, 104992.
- 12-Saod, W. M., Zaidan, T. A., & Alfaluji, A. W. A. R. (2019). Hepatitis B and Renal Function of Patients with Chronic Hepatitis B in Fallujah District, Iraq. *Biochemical and Cellular Archives*, 19(Suppl. 1), 1999-2004
- 13-Pocino, K., Napodano, C., Gragnani, L., Ciasca, G., Colantuono, S., Marri, S., & Basile, U. (2021). Solving the mystery of HBV-related mixed cryoglobulinemia: potential biomarkers of disease progression. *Rheumatology*, 60(9), 4418-4427
- 14-Mak, L. Y., Huang, Q., Wong, D. K. H., Stamm, L., Cheung, K. S., Ko, K. L., & Yuen, M. F. (2021). Residual HBV DNA and pgRNA viraemia is associated with hepatocellular carcinoma in chronic hepatitis B patients on antiviral therapy. *Journal of Gastroenterology*, 56(5), 479-488
- 15-Xi, Z., Huang, R., Li, Z., He, N., Wang, T., Su, E., & Deng, Y. (2015). Selection of HBsAg-specific DNA aptamers based on carboxylated magnetic nanoparticles and their application in the rapid and simple detection of hepatitis B virus infection. *ACS Applied Materials & Interfaces*, 7(21), 11215-11223
- 16-Al-Rubaye, A., Tariq, Z., & Alrubaiy, L. (2016). Prevalence of hepatitis B seromarkers and hepatitis C antibodies in blood donors in Basra, Iraq. *BMJ open gastroenterology*, 3(1), e000067
- 17-Jang, J. W., Kwon, J. H., You, C. R., Kim, J. D., Woo, H. Y., Bae, S. H., & Chung, K. W. (2011). Risk of HBV reactivation according to viral status and treatment intensity in patients with hepatocellular carcinoma. *Antiviral therapy*, 16(7), 969-977.
- 18-Mese, S., Nergiz, S., Tekes, S., & Gul, K. (2014). Seroprevalence of serum HBsAg positivity and

hepatitis delta virus infection among blood donors in Southeastern Turkey. *ClinTer*, 165(2), 95-8.

19-Hadi, L. M., Hussein, A. A., &Ja'afer, A. M. (2017). Seroprevalence of HDV Infection among HBsAg Positive Blood Donor in Baqubah City, Iraq. *Diyala Journal of Medicine*, 13(1), 74-83.

20-Chan, H. L. Y., Thompson, A., Martinot-Peignoux, M., Piratvisuth, T., Cornberg, M., Brunetto, M. R., ...& Marcellin, P. (2011). Hepatitis B surface antigen quantification: why and how to use it in 2011—a core group report. *Journal of hepatology*, 55(5), 1121-1131.

21-Fouad, R., Abdo, M., Eldeen, H. G., Sabry, D., Atef, M., Ahmed, R., &Zayed, N. (2016). Influence of delta virus infection on the virologic status in Egyptian patients with chronic hepatitis B virus genotype D. *Journal of medical virology*, 88(5), 837-842.

22-Zainal, I. G., Safa, A. A., &Obead, W. K. (2012). comparison of glyco proteins levels with some biochemical parameters in iraqi patients with chronic liver diseases. *Innovative Journal of Medical and Health Science*, 2 (5), 89 -93

23-Mohammed, A. A., Enan, K. A., Khair, O. M., Hussien, M. O., El Hussein, A. R. M., &Elkhidir, I. M. (2015). Prevalence of occult hepatitis B virus (HBV) infections in haemodialysis patients in Khartoum State, Sudan from 2012 to 2014. *Journal of Medical Laboratory and Diagnosis*, 6(4), 22-26.

24-Shahid, M., Khan, M. A., &Idrees, M. (2022). Frequency of Hepatitis D Viral Infection in Chronic Hepatitis B Patients in Pakistan.

25-Phillips, R. O., &Geretti, A. M. (2016). The gamma-glutamyltranspeptidase to platelet ratio (GPR) shows poor correlation with transient elastography measurements of liver fibrosis in HIV-positive patients with chronic hepatitis B in West Africa. Response to: 'The gamma-glutamyltranspeptidase to platelet ratio (GPR) predicts significant liver fibrosis and cirrhosis in patients with chronic HBV infection in West Africa' by Lemoine et al. *Gut*, 65(5), 882-884.