IMMUNOLOGICAL AND BIOCHEMICAL PARAMETERS OF CHRONIC HEPATITIS B INFECTION AMONG PATIENTS IN DUHOK CITY – KURDISTAN REGION / IRAQ

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ABSTRACT

Background: Hepatitis B virus infection (HBV) is regarded as serious public health problem and approximately two billion people are infected with the virus. The presence of HBsAg with the clinical and biochemical features of hepatitis B infection reflects onset of the infection. It is sometimes very difficult to distinguish between inactive HBV carrier and patients with active HBeAg negative chronic hepatitis B. The current study aimed to measure the immunological and biochemical markers other than HBsAg in patients with chronic hepatitis B virus (HBV) attended the infectious diseases unit at Azadi Teaching Hospital in Duhok.

Subjects and Methods: Eighty nine chronic hepatitis B patients were included in this study from June 2015 through December 2015. Serum samples were collected from each patient and measured for HBsAg, anti HBs Ab, HBeAg, anti HBe Ab, and total Anti HB core Ab by using a rapid test (PLASMATEC One Step Multi –HBV Test Device). The HBsAg level was further tested by ELISA (HBsAg ELISA Test Kit, PLASMATEC Laboratory Products). The HBeAg & HBeAb were confirmed by immune-chemilumencense assay. Levels of Alanine Aminotransferase (ALT), Aspartate Transaminase (AST), and Alkaline Phosphatase (ALP) in each serum sample were also measured.

Results: Out of the 89 HBs Ag positive patients 51(57.3%) were males and 38 (42.7%) were females by using ELISA test, 21(23.6%) were positive for HBeAg, and 76 (85.4%) were positive for anti-HBe antibodies by using Chemiluminescent Immunoassay (CLIA) techniques. There were 68 cases of HBe Ag negative and anti HBe Ab positive, 13 patients were HBe Ag positive and anti HBeAb negative and only 8 cases were both HBe Ag and anti HBeAb positive. Among the male patients, 10(47.61%) and 44(57.9%) were positive for HBeAg and anti-HBe antibodies respectively, whereas for the females, 11(52.40%) and 32(42.10%) patients were positive for HBeAg and anti-HBeAb respectively. Regarding the ALT, 11(12.35%) were upper than normal range in which 7 (7.9%) were elevated (40-80 IU/mL) and 4 (4.5%) were twice upper than normal limit (>80). For AST, 12 (13.48%) patients were upper than normal range in which 11(12.35%) were elevated and only 1 (1.12%) was twice upper than normal range. In ALP, only 7(7.9%) of the patients were upper than reference range.

Conclusion: From the current study, it has been concluded that HBeAg and anti Hbe Ab are dependable indicators for the active stage of HBV replication in carrier patients when both of these variables are linked with elevated levels of serum ALT. Furthermore, most increased levels of HBsAg quantitatively were found among the early and young adults who were in the age range of (24- 44) years.

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Keywords: HBsAg, anti HBs Ab, HBeAg, anti HBeAb, Total HB core Ab, ALT, AST, ALP

Infection with Hepatitis B virus (HBV) is regarded as a global threat to public

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health that causes significant morbidity and mortality in the liver. Approximately two billion people are infected with HBV worldwide[1]. It is estimated that 44% of cases of cirrhotic diseases and 47% of hepatocellular carcinoma (HCC) are localised in Africa[2]. Recently, it has been considered that Iraq has the intermediate endemicity of Hepatitis B infections reflected by 3% sero-prevalence of hepatitis surface antigen (HBsAg) in normal population[3].

The infection of HBV consequences leads to a broad spectrum of liver disease starting from chronic hepatitis, cirrhosis, and HCC. Treatment of HB infections with antiviral agents is based on the precise and accurate interpretation of pathophysiologic parameters[4],[5&6].

Conforming to World Health Organization report, the distribution of HBV infection in the South Asian region estimates to be 2/8(4%). In 2001, the WHO grouped Bangladesh in the moderate to high risk group of countries for HBV infection[7].

The existence of HBsAg with the clinical and biochemical features of acute hepatitis reflects the onset of acute infection in patients from low endemic areas but not in patients from high or intermediate endemic regions in which acute and chronic infection may be misdiagnosed if the patient's carrier status is unknown[8]. To monitor vaccine-induced immunity and to distinguish acute, self-limited infections from chronic HBV infections, usually serological tests are used[9].

The other two important markers of hepatitis B infection are Serum HBV DNA and hepatitis B e antigen (HBeAg) that is reflective for active viral replication and the risk of infection transmission 4. Estimation of serum HBV DNA level is one of great importance for liver disease activity assessment and differentiating various aetiologies of hepatitis activity in HBV carriers that play as hepatocellular carcinoma development also it helps in decision making for antiviral therapy[10]. Data from the clinical and experimental studies suggest that serum HBeAg may play as an immunoregulatory role in natural infection[11] and is considered as immuolotolerant agent.

Some HBeAg negative chronic hepatitis B patients may follow sero-conversion from HBeAg to anti-HBe antibodies or may develop after years or decades of the inactive carrier state[12]. Furthermore, it represents a later immune reactive phase in the natural history of infection and characterized by periodic reactivation with a pattern of fluctuating levels of HBV DNA and ALT/AST and active hepatitis[12]. It is sometimes very difficult to distinguish true inactive HBV carrier and patients with active HBeAg negative chronic hepatitis B in whom phases of spontaneous reduction of infection may occur[13].

However, it is a crucial consideration particularly during defects and shortages of laboratory techniques that measure the HBV DNA viral load which affects the approach of initiating and justification of the antiviral therapy. In consequences, the alternative serological and biochemical markers may be helpful in determining the explicative activity of the HBV.

The present study was aiming at evaluating the serological and biochemical markers during the course of HBV chronic infection and making the correlation between them and with the status of the disease that might help in finding some

alternative parameters that interpret the status of the HBV infection in the areas with defects of advanced lab facilities.

PATIENTS AND METHODS

This study was performed in Duhok Medical Research Centre (DMRC) at the Medical College - Duhok University, between June 2015 and December 2015. The research has been approved by the ethical committee of the health directorate of Duhok governorate and the consent has been provided by all participating patients. Eighty nine (38 females and 51 males) consecutive serum samples of patients with HBV infection were taken from central laboratory in Azadi teaching hospital in Duhok city. Patients' data have been recorded including age, sex, residency and the date of sample collection.

All the cases were confirmed having HBV infection by rapid test for immunological markers; HBs Ag, anti HBsAb, HBeAg, anti HBeAb, and total HB coreAb using (PLASMATEC One Step Multi –HBV Test Device). HBsAg was measured quantitatively (HBsAg ELISA Test Kit, PLASMATEC Laboratory Products).

Biochemical Tests

The serum sample of each patient has been subjected to biochemical to estimate the levels of alanine aminotransferase (ALT) at references range from (5-40 IU/L), aspartate aminotransferase (AST) at reference range (up to 40 IU/L). Alkaline phosphatase (ALP) is measured within reference range of (up to 100 IU/L). ALP was measured using BIOLABO colorimetric Kit for quantitative determination. Both ALT and AST were estimated using [Boeki Prestige 24i - Biolis 24i kit].

Immunological Tests

For total HB core Ab and Anti HBs measuring was estimated by the rapid test (PLASMATEC Laboratory Products) using (ONE STEP Multi-HBV TEST DEVICE). HBsAg was measured quantitatively by (HBsAg ELISA Test Kit, PLASMATEC Laboratory Products), HBeAg and anti HBeAb titre was estimated in biochemistry section in Heave Paediatric Teaching Hospital in Duhok city by using Chemiluminescent Immunoassay (CLIA) techniques (DiaSorin LIAISON® HBeAg and LIAISON® Anti-HBe Kit).

STATISTICAL ANALYSES

Results were analysed by SPSS version (p value < than 0.05 was considered significant).

RESULTS

The total number of patients included in the study was 89. The mean age of the patients was (32.10± 13.398) years. Fifty one cases (57.3%) were males and thirty eight (42.7%) were females. Baseline characteristics of the study parameters are demonstrated in (Table 1).

Table1: Characteristics of the Parameters Study

Parameters		Mean ± SD	No[%]
Age [yrs.]		32.10 ± 13.398	
Gender	Male		51 [57.3]
	Female		38 [42.7]
HBsAg IU/mL		16238.14± 2488.675	
HBeAg U/ml		97762.54± 291656.512	=Mean± SD
Anti HBeAb IU/ml		75445.06930±2 12324.584914	
Anti HBs IU/mL		2.00 ± 0.00	

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Total Anti HBc Ab U/ml	1.00 ±0.00
ALT IU/L	29.20± 41.259
AST IU/L	28.2262± 18.88560
ALP IU/L	51.001± 42.034

Moreover, 38 (50.0%) of positive anti HBeAb were among age group in young adults.

Regarding the frequency of chronic HBV infections among age groups, it has been found that the highest frequency was within the age group (25-44) years (47%) followed by age group (15-24) years (27%) as in table 2.

Other immunological parameters like HBeAg and anti HBe Ab were analysed and correlated with age and with the biochemical variables.

Table 2: Frequency of Chronic HBV Patients among Age Groups.

Age group	Frequency	[%]
1-14	5	5.6
15-24	24	27.0
25-44	42	47.2
45-64	15	16.9
65-More	3	3.4
Total	89	100.0

There were 51 (57.30%) male and 38 (42.70%) female among the patients (table 3). The frequencies and percentages of positive cases of HBeAg and anti HBeAb in male were 10 (47.61%) and 44 (57.9%) respectively, while in female there were 11 (52.4%) and 32 (42.10%) positive cases for HBeAg and anti HBeAb respectively. In HBsAg results it showed not significant relation with gender as all cases were positive and results were constant but most cases were among male than female.

Table 3: Gender of Patients Regarding Hbs Ag

	Frequency	[%]
Female	38	42.7
Male	51	57.3
Total	89	100.0

Serological parameters:

All parameters of serological analysis HBsAg, HBeAg and anti HBeAb are shown in table (4&5).

Table 4: Hbeag & Anti Hbeab Frequencies and Percentages

		Frequency [%]
HBeAg	Positive	21/89 (23.6%)
	Negative	68/89 (76.4%)
Anti HBeAb	Positive	76/89 (85.4%)
	Negative	13/89 (14.6%)

Table 5: Correlations of Age Category with all Immunological Parameters: Hbsag, Hbeag and Anti Hbeab

Age category	HBs Ag	HBe Ag	Anti HBe Ab	Frequency, % Total
1-14	5 [5.6%]	3 [14.3%]	2 [40.0%]	5 [5.6%]
15-24	24 [27.0%]	7 [33.3%]	19 [79.2%]	24 [27.0%]
25-44	42 [47.2%]	10 [47.6%]	38 [50.0%]	42 [47.2%]
45-64	15 [16.9%]	1 [4.8%]	14 [18.4%]	15 [16.9%]
> 65	3 [3.4%]	0 [0.0%]	3 [3.9%]	3 [3.4%]

One of the main aim of this study to analyze the serological parameters as data of both HBeAg and HBeAb are the most crucial categories to investigate the patient's progression of the disease in HBV infection. The parameters were

grouped into three categories, when HBeAg positive and anti HBeAb negative, HBeAg negative with anti HbeAb positive, and both the parameters of HbeAg and anti HBeAb are positive. This has been revealed in correlation with age variable in table 6.

Table 6: Age Distribution in Relation to Hbe Ag and Hbeab Status

Age	HbeAg –ve/ HBeAb+ve	HBeAg+ve/ HBeAb-ve	HBeAg+ve HBeAb+ve
1-14	2/68 [2.94%]	3/13 [23.07%]	0
15-24	17/68 [25%]	6/13 [4.15%]	3/8 [37.5%]
25-44	32/68 [47.05%]	3/13 [23.07%]	5/8 [62.5%
45-64	14/68 [20.60%]	1/13 [7.69%]	0
≥ 65	3/68 [4.41%]	0	0
Total	**68**	**13**	**8**

Rapid test for HBs Ag and Anti-HBs Ab.:
All the patients were HBs Ag positive and anti HBs Ab negative. Among HBeAg positive 10/21 (47.61%) were male and 11/21 (52.4%) were female. In anti HBe Ab positive, 44/76 (57.9%) were male while 32/76 (42.1%) were female.

Total Anti HBcAb:
Among the 89 studied cases, all patients were positive 89 (100%) in anti HBc Ab marker as indicating the chronic infection of Hepatitis B. There were only 13 (14.6%) patients positive for HBeAg and negative for anti HbeAb and the other 68 (76.4%) patients were HbeAg negative and anti HBeAb and 8 (8.9%) of the patients were positive for both HBe Ag and anti HBe Ab.

Biochemical markers:The measured levels of the biochemical markers in the sera of the chronic hepatitis B patients are shown (table 7 & 8).

Table 7: Correlations of ALT Levels, with Hbsag (By ELISA) in Chronic Hepatitis B Patients

Frequency of patients with normal levels of ALT*	78 (87.6%)	**Mean of HBsAg titre (16238.14 IU/mL) P= 0.584**
Frequency of patients with elevated ALT**	7 (7.9%)	
Frequency of patients with twice upper than normal range of ALT***	4 [4.5%]	
Total	89 (100%)	

[In correlation of HBsAg titre with the frequency of ALT levels of patients, p value was 0.584 which was not significant {p = or <0.05]

* ALT/AST Normal = < 40 IU/mL according to central Lab Reference .Range,

** Range between of 40-80 IU/mL,

*** Twice upper than 80 and more

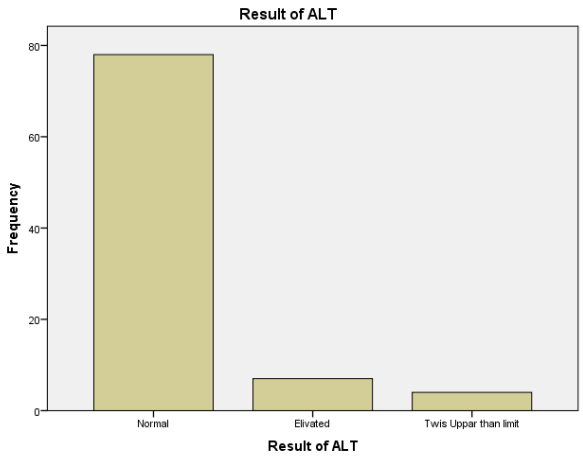

Diagram 1: Distribution of ALT Marker as Normal, Elevated and Twice Upper than Normal Range

Table 8: ALP Serum Levels in Chronic Hepatitis B Patients

	ALP
Patients with normal levels of the measured biochemical markers*	82 (92.1%)
Upper than normal Range**	7 (7.9%)

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Total	89 (100%)

* ALP Normal < 100 IU/mL according to central Lab Reference Range.

**ALP >more than 100.

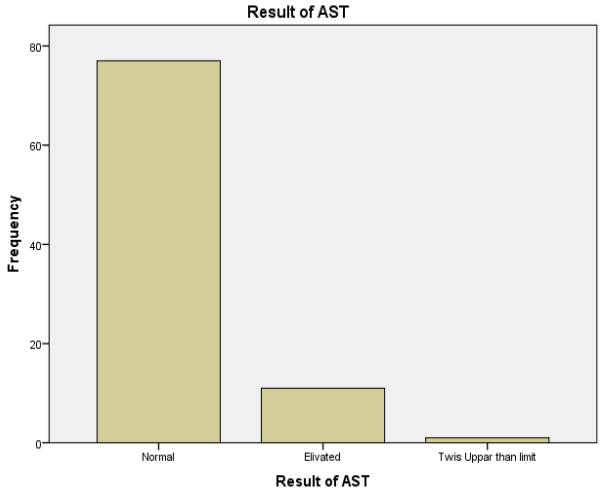

Diagram 2: Distribution of AST Marker as Normal, Elevated and Twice Upper than Normal Range

Correlations of biochemical parameters with each of HBsAg, HBeAg and anti HBeAb showed the most percentage among ALT, and AST with HBeAg negative and anti HBeAb positive. The cases that showed highly increased in ALP among group that are both HBeAg and anti HBeAb are positive were in low incidents and those are most likely are entering the stage of liver damage in case of further diagnosis applied such as biopsy and X rays as shown in table 9.

Table 9: Frequency of all Biochemical Marker Levels Upper than the Normal Limits in Regard to Hbe Ag/Anti Hbeab Levels

	Total	ALT> 40 IU/mL		AST>40 IU/mL		ALP >100 IU/mL
		Elevated	Twice[ULN]	Elevated	Twice[ULN]	
HBeAg –ve Anti-HBeAb+ve	68	5 [7.35%]	2 [2.94%]	7 [10.29%]	0	4 [5.88%]
HBeAg +ve Anti HBeAb-ve	13	2 [15.38%]	0	3 [23.0%]	0	3 [23.07%]
HBeAg +ve Anti HBeAb+ve	8	1 [12.5%]	1 [12.5%]	1 [12.5%}	1 12.5%]	0
Total	89	11 [12.35%]		12 [13.48%]		7 [7.86%]

DISCUSSION

In the current study, the biological and immunological profile has been evaluated and correlated with the prognosis of HBV infection in Duhok city that may help in finding some alternative parameters to interpret the status of the HBV infection and link them with the risk factors in the areas with defects of advanced lab facilities. The population age in this study ranged from [5-75 years] category with mean of about (32.1) years. According to the correlation between age and the presence of each of HBs Ag and HBe Ag and anti HBeAb parameters, this study revealed that approximately near to half of the cases (47.2%) were in the range (25-44) years group while the lowest one was 3/89 (3.4%) among >65 years age group. This finding is in consistent with those obtained by another study that has been done in Bangladesh[14], they showed that the highest frequency was found to be within

the (21 - 40) years of age which is close to the age range that showed in this study. Moreover, it has been shown that slightly above half of cases in this study were among male patients and this might be due to the more the frequency of exposure of males to hepatitis B virus than females, however, there was limited number of chronic carrier cases thus this finding needs further studies.

In the current study results show no significant correlations among age groups with regard to HBsAg yet the highest titer (47.2%) was found among the age group (25-44) years. This has been also proved by a study which is done in Bangladesh (Asadur R.& et al.2013)14 which revealed that maximum of patients were among the age group between (21-40) which it is similar to our findings. Furthermore, all of the patients (100%) recruited in this study were negative for anti HBsAb and positive for total HBcAb which is an indicator for the chronicity of the infection[15].

In HBeAg and anti HBeAb markers there were only 13 patients (14.60%) found to be HBeAg positive but with only 2(15.38%) of elevated ALT that was most likely to be within the 'active stage' of infection but none of the cases were twice upper the normal range of ALT. On the other hand, in the current study the high rate of patients who were positive for anti-HBeAb was found among the age group between (25-44) years while the lowest rate was within (1-14) years. This is suggesting that most likely the chronicity of infection was established among the patients during neonatal period of their life or could be transmitted vertically with HBV through perinatal time, this is also supported by other studies when they stated that the a chance of developing chronic infection among neonates infected with HBV through perinatal transmission[14].

In the present study, when patients categorised based on the ALT levels into [normal, elevated and twice upper the normal] and compared with the HBsAg titre, it was found that HBsAg did not correlate significantly with the level of ALT among the whole studied patients [p 0.584]. This means that HBsAg titre on its own could not be relied on for the evaluation of the liver function and enzymes level.

Also, it has been found that none of the liver biochemical markers (ALT and AST) were significantly associated with the immunological markers (HBsAg, anti HBeAb and anti HBeAb), but only (7.35%) of the HBe Ag negative and anti HBeAb positive had elevated ALT and only two patients with HBeAg positive and anti HBeAb negative had elevated ALT. It seems that sero-conversion of HBeAg to anti HBeAb in the patient's serum is linked with the decrease or normalization of the level of serum ALT. This has been supported by another study which revealed that elevated ALT of chronic hepatitis B were correlated significantly with detectable HBsAg, HBeAg and HBeAb levels[14], this means that raised ALT level is reflecting more vigorous response to the replicating HBV and therefore more hepatocyte damage[4].

A study by (Hussain AB, and et al 2004) revealed that out of 50 patients, 48 positive samples of HBeAg HBV DNA PCR hepatitis B virus carriers had elevated serum ALT level. They concluded that detectable HBeAg should be considered as

a surrogate marker for HBV DNA in hepatitis[16]. Also this has been also proved in another study which showed that most children who are HBeAg positive have elevated ALT levels and sero-conversion to anti-HBeAb is common near or shortly after the onset of puberty[13]. Moreover, in developed countries, most probably HBV infection is usually acquired during adulthood through sexual transmission and injecting drugs[9, 17].

Regarding ALP results, there were few cases of upper than normal range among groups with both HBe Ag and anti HBe Ab positives. This might indicate the occurrence of liver damage however, further studies in larger scale are needed and other investigations are considered to be done.

It has been suggested that those who are HBeAg positive are better tolerating the antiviral therapy[18], and consequently, the sero-conversion from HBeAg-positive status to an anti-HBe Ab positive status has been used to indicate antiviral response and results in a reduced morbidity and mortality[9, 19].

In the present study, as most cases of HBV were HBeAg negative and anti HBeAb positive, they have lost HBeAg and seroconverted to anti-HBeAb, accordingly the majority have gone into an 'inactive phase 'of chronic HBV infection[20]. However, some patients' after HBeAg seroconversion develop HBeAg-negative immune active disease, whereas others who have the inactive phase of HBV may experience a reversion to HBeAg positive immune active disease or develop HBeAg-negative immune active hepatitis[21]. Furthermore, during the natural course of HBV chronic infection, the loss of HBeAg appearance and the expression of antibodies directed against it, (anti-HBeAb) often represent the end of viral replication[20]. In this regard, HBeAg remain the most crucial immunological and biomarker as an indicator for the stage of the disease in a place where there are inadequate diagnosis facilities like molecular analysis[21].

In conclusion, this study determined the main objectives as the outcome of our findings indicates that most of the cases showed either in 'Immune tolerance phase' or 'active phase. Determinants such as HBsAg HBeAg, and anti HBeAb remain the most important markers for the diagnosis, of the infection specially if these markers are associated with age category to specify the period of transmission of the disease. However, it is recommended for further investigations such as molecular analysis like (PCR) to be done to determine the level of viral load (HBV DNA) in relation to other markers, to understand exactly the phase of infection in order to assess the treatment of the disease.

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CONFLICT OF INTERESTS

Nothing to declare.

REFERENCES:

1. Seto Wai-Ray, Ying- Rulo, Powlesky J-M and Yuen Man-Fung. Chronic Hepatitis B Virus infection. The Lancet.2018; Vol: 392, ISSUE 10161,P2313-2324.DOI:https://doi-org 110-1016/SO140-6736(18) 31865-8
   http://www.who.int/mediacentre/factsheets/fs204/en
2. Musa BM, Bussell S, Borodo MM, Samaila AA, Femi.OL. Prevalence of B virus infection in Nigeria, 2000-2013 A systemic review of Meta-analysis. Niger J. (Clin pract.2015; (2): 163-72).
3. Tarky AM, Akram W, Al-Naaimi AS, Omer AR. Epidemiology of viral hepatitis B and C in Iraq:a national survey 2005-2006. Zanco J. Med. Sci. 2013; 17:370-80.
4. Aspinall EJ, Hawkins GA, FraserA, Hutchinson S J, Goldberg D. Hepatitis B prevention, diagnosis, treatment and care: A review. Occupational Medicine. 2011; 61 (53):531-540.
5. Bowden S. Laboratory diagnosis of hepatitis B infection. In: Hepatitis B Virus. (Ed. Lai, C. L. and S. Locarnini). International Medical Press, London, UK. pp. 2002. 145-159.
6. Matthews G, Robotin M. B Positive - all you wanted to know about hepatitis B: A guide for primary care providers. Australasian Society for HIV Medicine (ASHM). 2008; pp: 31-39.
7. Islam MDU, Ahmed RU, Kibria SG, Hossain MT, Biswas, et al. Clinical Significance Of Serum HbeAg Among HbsAg Positive Patients, Faridpur Med. Coll. J. 2010; 5(2):50-52.
8. HanY, Tang Q, Zhu W, Zhang X, You L. Clinical, biochemical, immunological and virological profiles of and differential diagnosis between, patients with acute hepatitis B and chronic hepatitis B with acute flare. J. Gastroenterol. Hepatol. . 2008; (23): 1728-1733.
9. Krajden M, McNabb G, Patrick M. The laboratory diagnosis of hepatitis B virus. Can J Infect Dis Med Microbiol..2005; 16(2):65.
10. Sablon E. Shapiro F. Advances in Molecular Diagnosis of HBV Infection and Drug Resistance. Int. J. Med. Sci. 2005 (2): 8-16.
11. Milich D, Liang T. J. Exploring the biological basis of hepatitis B e antigen in hepatitis B virus infection.; Hepatology. 2003 (38): 1075-86.
12. Hadziyannis S J, Papatheodoridis G V. Hepatitis B e antigen negative chronic hepatitis B natural history and treatment. Semin line Dis. 2006, (26):150-141.
13. Brunetto M R, Oliveri F, Coco B, Leonardo G, Colombatto P Gorin J M, et al. Outcome of anti-HBe positive chronic hepatitis B in alpha-interferon treated and untreated patients. J Hepatol; 2002. (36):263-270.
14. Asadur R, Fhamid JA, Farjana R, Rashid N, Department of Immunology, Popular Diagnostic Centre Ltd., Dhaka-1205, Bangladesh. Journal of Bangladesh Academy of Sciences. 2013; (37), No. 1, 51-56.
15. Jake L, Hepatitis B: The Virus and Disease and Liver Diseases Branch, National Institute of Diabetes and

Digestive and Kidney Diseases (NIDDK), National Institutes of Health (NIH), Bethesda, MD. Hepatology. 2009 May; 49(5 Suppl): S13–S21. doi:10.1002/hep.22881.

16. Hussain AB, Karamar KA, Anwar M, Kazmi SY, Tariq WU. Correlation of HBV DNA PCR and HBeAg in hepatitis carriers. J Coll Physicians Surg Pak. 2004; 14:18-20.
17. Stroffoliniand and et al. Hepatitis C virus infection and alanine transaminase levels in the general population: a survey in a southern Italian town. Journal of Hepatology. 2000; 33: 116-120.
18. Saikia et al. 2007. World Health Organization. Geographic patterns of hepatitis B prevalence. . 2010. The World Health Organizations pages on vaccines and immunizations. (http://www.who.int/vaccinessurveilance/graphics/htmls/hepbprev.html, accessed 20 January.
19. Croagh CM, Desmond PV, Bell SJ. Genotypes and viral variants in chronic hepatitis B: A review of epidemiology and clinical relevance. World J Hepatol .2015; 7: 289-303 [PMID: 25848459 DOI: 10.4254/wjh.v7.i3.289].
20. McMahon BJ. Chronic Hepatitis B: Update HEPATOLOGY, AASLD PRACTICE GUIDELINE UPDATE. 2009.
21. Patrizia C, Rita C, Giancarlo I, Bianca B. HBV mutations and infection management WJG|www.wjgnet.com 151 January 7, 2016;Volume 22|Issue 1| World J Gastroenterol. 2016 January 7; 22(1): 145-154 ISSN 1007-9327 (print) ISSN 2219-2840

ثوختة

ثێشەکی:

نفەکسیۆناڤایروسێیا هەپاتیت ب، پرسگرێکەک تەندورستیا گەلەمپەرییا گەلەمپەرییە، و نێزیکییەک میلیار کەس ب ڤیرووسێڤە تێتە کرن. هەبووناب تایبەتمەندیێن HBsAg کلینیکی و بیۆلۆژیکێن ئەنفەکسیۆنێ هەپاتی کو نیشان ددەدەستپێکا ئەنفەکسیۆنێB . جارنان ئەڤ پر زەهمەتە کو ژ جەرباندنا گەریدەیا نەیینییاو نەخوەشێن ب هەپاتیتێن کرۆنیکیێن چالاکڤەبێژن

ئارمانج:

ئارمانجا ڤێ لێکۆلینێ پیڤاندنا تایبەتمەندییێنو رێگەزێن HBeAg بیۆلۆژیکە ژ بلی Immunomodulatory د نەخوەشێن ب ڤایرووسا HBsAbهەپاتیتکرۆنیک کو بەشداری بەشا نەخوەشیێ Bبوو نەخوەشیێن ئنفەکسیۆنێ ل نەخوەشخانەیارِاگهاندنێیا ئازادی ل دهوک.

دەرئەنجام

ژ لێکۆلینەک نوها، هات ئەنجامدان کو HBeAg و HBeAb نیشانێن پێباوەر ئێن قۆناخا چالاک ئا دوبارەبوونائێHBV د نەخوەشێن خوەدان کاروانان دە نە دەما کو هەر یەک ژ ڤان گوهێرباران ب ئاستێنڤە گێدایییە. دگەل ڤێیەکێ، پرانیا ئاستێن زێدەبووناد ناڤ مەزنێن دەستپێکێ HBsA و جوانێن کو ژ کۆما تەمەنێ (٢٤-٤٤) سالیرەِراست بوون دە هات هەسباندن.

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الخلاصة

الخصائص المناعية والكيميائية- الأحيائية لمرضى لتهاب الكبد المزمن B في مدينة دهوك – إقليم كردستان / العراق

خلفية الدراسة و الهدف: تعتبرعدوى فيروس التهاب الكبدB مشكلةخطيرة في مجال الصحة العامة، كما أن حوالي مليار يشخص مصاب بالفيروس. وجود HBsAg مع السمات السريرية والكيميائية الحيوية للعدوى التهاب الكبد B اللذي يعكس بداية العدوى. في بعض الأحيان من الصعب جدا التمييز بين الناقل غيرنشط HBV والمرضى الذين يعانون من التهاب الكبد B المزمن النشط HBeAg. هدف الدراسة الحالية هو قياس الخصائص المناعية والوسائل الكيميائية الحيوية الى جانب Ag HBs في المرضى الذين يعانون من فيروس التهاب الكبد B المزمن اللذين حضروا وحدة الأمراض المعدية في مستشفى أزادي التعليمي في دهوك.

طريقةالبحث: تم ضم تسعة وثمانين من مرضى التهاب الكبدB المزمن في هذه الدراسة من يونيو 2015 لغاية ديسمبر 2015. وتم جمع عينات المصل من كل مريض وقياس مستضد الHBsAg، والاجسام المضادة لها HBs Ab، وقياس الHBeAg، والاجسام المضادة لهاanti HBeAb ، ومجموع Anti HB core Ab باستخدام الاختبار السريع(PLASMATEC) (Multi one step وتم اختبار مستوىHBsAg بواسطة ELISA (مجموعةاختبار HBsAg ELISAمن منتجات اختيارات PLASMATEC ELISA (HBsAg,Kit). تمتأكيد قياس كلا من HBeAb & HBeAg عن طريق الفحص المناعي الكيميائي (CLIA). أيضا تم قياس مستويات (ALT) Alanine Aminotransferase،(AST) Aspartate Transaminase، والفوسفاتيزالقلوي (ALP) في كل عينة مصل لكل مريض.

النتائج: منبين 89 من المرضى الإيجابيين ل HBs Ag كان 51 (57.3 ٪) من الذكور و 38 (42.7 ٪) من الإناث واللتي اجريت عن طريق اختبار ELISA ، و 21 (23.6 ٪) كانت إيجابية لHBeAg، و 76 (85.4 ٪) كانت إيجابية لمضادات الأجسام المضادة antiHBeAb– باستخدام تقنيات المناعية الكيميائية .(CLIA) كان هناك 68 حالة من HBe Ag سلبية ومضادة لـ antiHBeAbإيجابية، 13 مريضا كانوا HBe Ag موجبو مضاد ل .HBeAb سالبو 8 حالات فقط كانت HBe Ag و HBeAbإيجابية .

بين المرضى الذكور، كانت 10 (47.61 ٪) و 44 (57.9 ٪) إيجابية لل HbeAgو الأجسام المضادة ل antiHBeAb على التوالي، في حين أن الإناث، 11 (52.40 ٪) و 32 (42.10 ٪) كانت إيجابية لل HBeAg ومضادةHBeAb– على التوالي. بخصوص ALT ،كان 11 (12.35٪) أعلى من المعدل الطبيعي حيث ارتفع 7 (7.9٪) (40–80 IU/mL) و 4 (4.5٪) كانت أعلى مرتين من الحد الطبيعي . (80>). بالنسبة لـ AST ،كان 12 (13.48٪) من المرضى أعلى منالمعدل الطبيعي، حيث كان 11 (12.35٪) مرتفعة وكان فقط 1 (1.12٪) أعلى مرتين من المعدل الطبيعي. في ALP ، كان 7 (7.9٪) فقط من المرضى أعلى من النطاق المرجعي ومكافحةHbeAb هي مؤشرات يمكن الاعتماد عليها للمرحلة النشطة في المرض الناقل عندما يرتبط كل من هذه المتغيرات معا لمستويات المرتفعة من ALT في المصل. وعلاوة على ذلك، تم العثورعلى معظم مستويات الزيادة من HBsAgواللتي قيست مستوياتها كميا بين البالغين في وقت مبكروالشباب الذين كانوا في الفئة العمرية (24–44) سنة .

الاستنتاج: من الدراسةا لحالية، تم التوصل إلى أن HbeAgو HbeAb هما مؤشران يمكن الاعتماد عليهم اللمرحلة النشطة لتكرار HBVفي المرضى الذين يعانون من الناقلات عندما يرتبط كل من هذه المتغيرات بمستويات مرتفعة لل .ALTوعلاوة على ذلك، تم العثور على معظم مستويات الزيادة من ال HBsA واللتي قيست مستوياتها كميا بين البالغين في وقت مبكرو الشباب الذين كانوا فيا لفئة العمرية من (24–44) سنة.