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The inflammatory reaction Of Hepatitis B Virus Patients.

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Abstract

study aim to estimate immune molecules CD54 & CD74 and TNF-B and IL-8 in patients infected with HBV , A total of (80) seropositive patients for HBV were screened for this study to know level of TNF-B & IL-8 in serum of patient and show expression of CD54 and CD74. Results showed that serum samples were analyzed for IL-8 & TNF-B by ELISA , showed highly significant increases ($p<0.05$) in serum level of HBV patients as compared with healthy control groups , acute HBV revealed high as well as , increases in serum level of TNF-B significantly($p<0.05$) ,while chronic liver disease patients express high increase in serum level of IL-8 significantly($p<0.05$).Activated markers study revealed high expression of CD74 & CD45-R in HBV patients as compared with healthy normal groups

Keywords: *HBV, CD54, CD74*

I. Introduction

Hepatitis B virus (HBV) was first exposed by Blumberg et al in 1965 (1), and the connection between HBV and acute hepatitis after blood transfusion was stated by (2). At that time, most trainings were based on immunological and serological means. Molecular-based studies progressed rapidly after the HBV element was exposed and the HBV genome cloned (3). HBV infection is chief global matter, and is a specific fear in Asia and Africa. While HBV itself is not right cytotoxic, the immune response to HBV infection causes liver harm and finally leads to liver cirrhosis and hepatocellular carcinoma (HCC). More than 350 million persons worldwide are believed to be chronically infected with HBV and 1-2 million people die each year from HBV-related cirrhosis and HCC (4). HBV is the main foundation of non-digestive hepatitis. The record shared of HBV layers are taken by changed method of blood , In people having numerous blood transfusion ,like hemophilia or thalassemia patients, are mostly at great danger of having HBV [6,5]. Both sexual &Prenatal spread are quite rare. Anyway, the pathway of contamination is private in almost 50% of people having HBV.

ended the outside of infested cells to get red of apoptosis The rate of vital CD8+ and CD4+ T answers in patient blood grieves from acute disease appearances connected with rescue[9,10].. The controller of acute disease is related with a falling HBV variation, reproducing a “surrounding” of HBV change with a positive unaffected reaction, while chronicity is linked with quasi-species allowance [11].

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Cytokines function as the molecules of defense reaction that result in numerous physiological roles and adjust the defensive, provocative and repairing patient reactions [12,13].

II. Materials &Methods

1- Patients

The study enrolled 80 HBV patient , acknowledged at the public health laboratory and with yellowish color or signs and symptoms sensitive of critical and chronic HBV patients and showed seropositive for anti HBV antibody.

Serum cytokine

Sizes of cytokines in the serum were done by ELISA test (R&D Systems). Absorbance was restrained in copies with a micro plate reader (Beckman Coulter). The last concentration was expressed in pg/ml.

Statistical analysis:

Statistical analysis was showed by using Chi-square ($\chi 2$) test to adjust the statistical vagaries amid assorted sets by spending a application statistical stand for social science (SPSS 19). The opportunity of ($P \leq 0.05$) was restrained to be statistically important.

III. Results & Discussion

1-Medical Remarks

Medical marks in HBV patients were comprised vomiting, fever, loss of appetite , while other patients never exposed any of these signs and shows asymptomatic carrier as shown in table (1).

Table (1) Clinical signs for HBV patient .

NO.	Clinical signs	Number	Percentage%
1	Acute sings	10	12.5%
2	Chronic Liver disease	5	2.5%
3	(asymptomatic signs)	60	75%

Our results of this study showed that 15(15%) cases showed signs of vomiting ,fever ,loss of appetite and abdominal pains ,while 80 (80%) cases showed asymptomatic and 5(5%) develop into chronic liver disease separately. According to F- exam the variation in medical marks were substantial ($p<0.05$) . Symptoms of acute phase of HbV disease remains clinically quiet for most patients, and only (15% - 20%) of people succeeding medical grades [14]. Symptoms of acute HBV infection are nonspecific and include fatigue, poor appetite,

nausea, vomiting, abdominal pain, low-grade fever, jaundice, and dark urine. Clinical signs include liver tenderness, hepatomegaly, and splenomegaly.(15,16). While the majority of these (80–90%) have cirrhosis at the time of diagnosis of HCC, it may occasionally follow without the presence of cirrhosis; this is principally real for HCC due to HBV(17)

2. IL-8 in hepatitis patients

Serum of all patients with HBV and those with acute or asymptomatic disease action contain higher level of IL-6 than healthy control group . IL-6 concentration was particularly increased in patients with chronic liver disease and asymptomatic patients , correspondingly than acute HBV disease

T-test ,showed that there was an increased arithmetical substantial differences among asymptomatic , chronic liver disease and acute infection group ($p<0.05$) as table (2)

Table(2) The level of IL-8 in patients and controls

Group	NO.	Serum level of IL-6		
		Mean ±SE	Minimum	Maximum
Asymptomatic	60	814.82±61.84	180.00	2200.00
Acute HBV	12.5	518.20±71.94	113.00	988.00
Chronic liver disease	2.5	1324.60±211.50	1319.00	1110.00
Control	10	55.50±1.49	72.00	75.00
Total	90	713.21±59.65	71.00	2100.00

In vivo, the chemotactic rank can be produced by the joining of IL-6 to proteins of basement membrane . This position helps in success cells in the bearing of the location of inflammation besides preserves them when they are reached(18). Furthermore to draught, IL-6 supports to excite the initiative of neutrophils and monocytes [,19,20]. Neutrophils offer the principal-route of security in distinction to offensive different pathogens as virus. [24,25].

3-level of TNF

Current study showed that all patients with H BV cover higher level of TNF-α than healthy control group , T NF-α concentration was improved particularly with acute H CV patients ,asymptomatic patients(81.43± 5.00) and liver cirrhosis patients correspondingly .Analysis of variance among acute H CV , asymptomatic, liver cirrhosis , and control people ($p<0.001$) .T- test exhibited that there was great statistical significant alteration among acute HBV and asymptomatic, liver chronic disease group ($p<0.001$)table(3)

Table(3) The level of TNF-B in patients and controls

Group	NO.	Serum level of TNF-B		
		Mean ±SE	Minimum	Maximum
Asymptomatic	60	72.13±5.00	27.21	211.46
Acute HBV	12.5	583.64±17.43	503.00	688.00
Chronic liver disease	2.5	17.61±0.82	18.87	18.44
Control	10	11.51±1.01	9.18	17.48

In acute agreeing infections, the response of the innate and adaptive

Immune system to *HBV* is capable and well-timed.(26) Viral clearance involves the generation of a stout adaptive T cell retort inducing both a cytolytic(27,28)

4- CD54 molecules in HBV patients

Results shown that there was highly significant differences in mean of CD 54 expression among HBV patients and healthy control groups ($p<0.005$) ,the cell surface CD45-R was over expressed in acute HBV compared to asymptomatic HBV patients, liver cirrhosis and healthy control groups respectively the high expression seen in acute HBV disease.

The hepatic vascular opinion is doubled known past vesseles that frequency into web definite tubes well-known as hepatic sinusoids[29] .These sinusoids are creased with pored endothelial cells (E Cs) & luminal Kupffer cells (K Cs), & track same organized via liver parenchyma passing conveying blood stream gorgeous with O2 as well as nutrition & Ag to body tissue [30].On reoccurrence run, "blood" provide to prime vessels then liver strains formerly retiring complete the spare hepatic poorer venacava [29,30) The result of acute infection is usually prepared in the major 6 months and ultimately be commission on the scope, time and specific of the adaptive immune reaction[31].Acute shaping infections are considered thru primary widening of "poly clonal C D 4+andCD8 + T –cell" occupants that unremitting over budget[32]. On other hand, elongated infections are associated with fleeting stuck comebacks that are feeble and objective a slim arrangement of MHC class I and II restricted epitopes [33]

5- Expression of CD74 in HBV positive patients

The results demonstrated in table (4) shows there was high statistically significant difference in mean of CD79 expression

among HBV patients and healthy control groups ($p<0.005$),and the higher percentage of expression was found in acute patients , chronic liver disease followed by asymptomatic patients and control groups respectively .

T –test results showed that there was high significant difference between acute HBV ,asymptomatic and liver chronic liver disease groups($p<0.05$).

To develop rid of hepatitis (H BV) is correlated with bubbly multi-vague C D 4+ and C D 8+ T cell responses ,while societies that movement chronic infection prospective to have fragile, slimly dedicated responses [34]. Revisions on chimpanzees have exposed that reduction of C D4+ or C D 8+ cells inhibits H B V budget[10]. In H B V infected people, C D8+ T C M cells occur in the verge are able of peculiar into E MC, that are enrolled to the liver. CD8+ effector cells in the liver were pledgee to have less useable adeptness, as substantiated by low IF N -yuntruth [35 The determination of liver pathogens is frequently attended thru frail "CD8+ T cell response " antigens subsequent[36,37]. We exasperated to conclude the pathogenic status of C D79 over comparing of its expression during infection , our results make it clear that robust up-regulation of both C D45&C D79 manage a tough mark that lymphocytes in peripheral blood of H CV persons within formal of immune dysregulation. [38,39].

Table(4.) The level of CD54 in patients and controls

Group	NO.	Serum level of CD45		
		Mean ±SE	Minimum	Maximum
asymptomatic	60	9.01±0.33	4.51	11.30
Acute HBV	12.5	13.17±0.22	12.13	14.70
Chronic liver disease	2.5	6.61±0.33	6.12	6.13
Control	10	4.22±0.21	0.90	5.16

Table(5) The Concentration of CD79 in patients and controls

Group	NO.	Serum level of CD74		
		Mean ±SE	Minimum	Maximum
asymptomatic	60	8.22±0.55	6.00	19.00
Acute HBV	12.5	40.26±5.11	18.21	41.70
Chronic liver disease	2.5	14.09±1.00	11.69	13.69

Control	10	7.21±0.20	6.0	9.0

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