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Association of IL-17 with hepatitis A virus Childhood infection patients less than 10 years old

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Abstract

Infectious hepatitis infection is the main cause of severe liver failure through the childhood period. People infected with Hepatitis A virus represents the primary source of this virus. After a person ingests a contaminated food or drink, the live virus invades the body, although it might/will take 2–6 weeks after exposure for symptoms to manifest. infectious hepatitis prompts an severe infection that consequences in the removal of the disease via the host, throughout the infection is the consequence of specific immune reactions under the control of cytokines. The humoral immune system reaction to infectious hepatitis is characterized by the specific production of IgM immunoglobulin against the VP1 viral protein and IgG immunoglobulin against the VP1 and VP3 viral proteins .In this study established the serum T-helper (Th)17-related cytokine during distinct hepatitis A virus (HAV)-induced clinical course in children .The clinical spectrum of disease is variable.

Keywords: Hepatitis A Virus; interleukin 17,childhood.

INTRODUCTION

Hepatitis A virus (HAV) is an earliest human pathogen and the major cause of acute liver failure in pediatric patients. The virus is greatly transferable in both youngsters and adults and more predominant in developing countries than developed countries (1). Every year , worldwide, infectious hepatitis (HAV) contaminates nearly 1.5 million by polluted food or drink; this kind of transmission has indicated unsanitary conditions(2,3).
Recently, The related studies reported variations in cytokine profiles identified as a potential immune-modulator in HAV infection(4). The acute infection of HAV , it self - limiting illness , most cases recover spontaneously without remaining harm or sequel. In spite of that, during infection, the spectrum of clinical symptoms is wide, extending from clement to intermediary infection to severe liver failure (5). The undisclosed r reasons of the changeability in the medical course originated by infectious hepatitis have not been clearly recognized . In spite of the fact that, infectious hepatitis is a no- cytopathic effect, the impairment to the liver causing from the infection most likely would not stem directly from Viral Replication; somewhat , it is created by the virus-specific, acquired immune response to afflicted Hepatocytes (6,7). Evidences are reviewed indicating that liver destruction produced by infectious hepatitis has been related with the response of host (CD8) T lymphocytes engaged against virus-infected hepatocytes and with the construction of a T helper type 1 cytokine profile(8).

Cytokines are basic components of suitable immune responses and production a important role during viral infections (9,10).
(IL) -17 is a cytokine prototype of T-helper (Th)17 cells and is linked with the stimulation of inflammation (11).

Th17 cells produce a pro-inflammatory reaction and need converting growth factor (TGF)-β, IL-1β, IL-6 , IL-21 and IL-23 to recognize and discharge cytokines, for example IL-17A, IL-17F, IL-21, IL-22 and (TNF)-α tumor necrosis factor alpha (12).

MATERIALS AND METHODS

Patients group

A total of (48) blood samples were collected from hepatitis A pediatric patients (less than 10 years old) were admitted to Babylon hospital for Gynecology and children between 2015-2016. A permission was provided from children's parents, then, blood samples were collected from patients by vein puncture and serum samples were tested for total serum bilirubin determination by used Bilirubin Special Kit, its quantitative TSB in human (cobasTM c 111 chemistry analyzer) then serum was exam for the existence of anti- HAV IgM by using HAV IgM rabid test – cassette CTK Biotech, Inc. USA. in order to detect severe infectious hepatitis .
A Human IL-17 (Interleukin 17) ELISA Kit Elabscience Biotechnology Co., Ltd. Was used for evaluation of the cytokine concentration in the serum samples of patients with infectious hepatitis infection.

Control group:

A total of (48) serum samples were collected from patients with symptoms other causes than of hepatitis. All were tested by (TSB, anti- IgM and IL-17) as in patients group.

RESULTS

This study was carried out on (1-10) years old children with hepatitis A infection, who are admitted to Babylon hospital for Gynecology and children. A total(48) pediatric patients were tested. 25 patients (52 %) were female and 23 patients (47%) were male (Table 1). The inactivity with this virus increased in March (21%) followed by September(14.6%), whereas the lowest percent of the infectivity recorded in January, May, and November (Table 2). Serum bilirubin showed elevated concentration (6.320± 2.852) in hepatitis A infection group than (0.392 ± 0.197) in control group with a significant difference (0.001) between them (Table 3).

The study result was shown anti-HAV IgM antibodies significantly differ (0.001) between hepatitis A patients and control group (table 4). The titer of Th17-related cytokines were examined in samples from infectious hepatitis infected patients and control donors as in (Table 5).

Table 1: Sex distribution of infectious hepatitis infection

Sex distribution		No.	%	p
Positive patient with HAV infection	Male	23	47.9	o.7
	Female	25	52.1	
	Total	48	100	

Table 2: Distribution of infectious hepatitis infection during year

Positive Patients		No.	%	P
Positive Patients /Month	January	2	4.2	0.02
	February	6	12.5	
	March	10	21	
	April	2	4.2	
	May	1	2.1	
	June	4	8.3	
	July	2	4.2	
	August	6	12.5	
	September	7	14.6	
	October	3	6	
	November	0	0	
	December	5	10.4	
	Total	48	100	

Table 3: Mean concentratin of TSB in Hepatitis A patients and controls.

Test	Hepatitis A infection group		Control group		P Value
TSB > 1.4 mg/dl	Mean	SD	Mean	SD	0.001
	6.320	± 2.852	0.392	± 0.197	
		48		48	

Table 4: anti-HAV IgM antibody rabid test in Hepatitis A patients and control group.

Test	Hepatitis A infection group	Control group	P value
Anti- IgM HAV			0.001
Positive	47	0	
Negative	1	48	
total	48	48	

Table 5:Compersion of IL-17 mean level and stander deviation between cases and controls

Group	N	mean	Std. Deviation	P
Control	20	0.6700	0.44260	0.0001
Cases	16	49.0826	28.03705	

DISCUSSION

Infectious hepatitis the main reason of severe liver failure in children patients and HAV is bind with increased shedding of the virus particles through the 3 -6 week incubation period. Anti-HAV antibody detecting initial in the course of our infectious hepatitis infection is mainly IgM (13).

For of a our patients the level of total serum bilirubin were shown elevated had a protracted clinical course (14).

The developing of virus may describe the extreme incident of infectious hepatitis contagion in areas with little principles of sanitation , which successively help the spread of the virus (15).

The cellular immunity to infectious hepatitis is strong and very active in removing the infection . Once, the initial signal of hepatocellular damage appears, Counteracting immunoglobulin to the virus(anti-HAV) usually seem in the serum simultaneous. Infectious hepatitis infection seems to product in a typical protected unlike from that produced from other hepatotropic viruses, counting transfusion hepatitis due to the regulator of specific cytokine like interleukin 17 and this lead to be determined to liver damage during the acute stage. Th17 cells may be major players through hepatitis A infection by secreting IL-22 (16). Cytokine control through infectious hepatitis contamination influences in viral clearance in 108 days (17).

CONCLUSIONS:

We can conclude the high occurrence of infectious hepatitis virus recorded in age less than 10 years.

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