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Web Site: https://jmed.utq.edu **Email:** utjmed@utq.edu.iq

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Serological Study of Human Parvovirus (B19) Antigen Detected among Patients with Viral Hepatitis

Ahmed Waleed Dheyab Alnassar Department of Microbiology, Medical Microbiology, College of Medicine, University of Thi-Qar
E. Mail: ahmed-w@utq.edu.iq
Mohammed Jasim Mohammed Shallal Department of Microbiology, Medical Microbiology, Medical Virology, College of Medicine, University of Thi-Qar
E. Mail: mohammed-j@utq.edu.iq

Abstract

Background: The B19 Human Parvovirus is an icosahedral virus with a single strand and no envelope. A total of 5596 bases make up its DNA, which places it in the parvovirus family. The deoxyribonucleic acid (DNA) B19 virus mutation has been linked to liver dysfunction in transplant recipients and has been associated with an increased risk of developing liver disease. As a result, scientists suspect that B19V has a negative role in liver damage, especially in instances of acute hepatitis and acute liver failure of unclear origin, as well as in those with hepatitis B or C co-infection, all of which have a worse prognosis.

Methods:

From August 2022 to the end of February 2023. This study included the collection of serum samples for the detection of Human Parvovirus antigen in 60 patients with viral hepatitis. Also included was a control group consisting of 30 individuals of different ages who did not have viral hepatitis. All these serum samples are detected for Parvovirus antigen by the enzyme-linked immunosorbent assay method.

Conclusions:

According to the results of this study, it is first concluded that the presence of human Parvovirus in patients with viral hepatitis was not influenced by most of the demographic factors studied. It is found that there are no significant differences in the detection rate of human Parvovirus among patients in terms of sex, age, residence, COVID-19 infection, or vaccination against COVID-19. The study did find a 10% detection rate of Human Parvovirus protein antigen among patients infected with hepatitis B and C viruses. Overall, the study suggests that viral hepatitis may play a role in the prevalence of Human Parvovirus B19, but further research is needed to fully understand the direct relationship between the two viruses that affect their prevalence in humans.

Keywords: HPV: Human Parvovirus, viral hepatitis

Introduction

An infection with B19 may lead to a variety of liver problems, including elevated transaminases, acute hepatitis, fulminant liver failure, and chronic hepatitis. Mihaly et al. (2012), found that 4.1% of patients infected with Parvovirus B-19 went on to develop hepatitis. Numerous studies have linked B19 infection with liver damage, most often manifesting as mild acute hepatitis (1,2).

A receptor on the cell membrane that seems to be specific for B19 has been identified in mature liver cells, revealing a mechanism for B19 entrance into hepatocytes (3,4). Long-term persistence of B19 DNA in the blood, bone marrow, and synovial fluid has been reported in asymptomatic, immunocompetent individuals. Active B19 infection was found in hepatitis patients from Brazil who tested negative for hepatitis C and hepatitis B, but there was no correlation between the infection and liver damage. B19 is often retained in immunocompromised persons (5). Chronic B19 infection in renal transplant patients was associated with increased cytolytic liver damage, as reported by Lee et al. These results suggest that B19 may in certain cases be the deciding factor in chronic liver injury (6). The presence of B19V DNA in the liver tissue of children with fulminant liver failure and aplastic anemia has led researchers to hypothesise that B19V infection is the source of their symptoms. (7). While B19V was the main causal agent in certain groups of children with acute viral hepatitis, it was shown to be co-infected with other hepatotropic viruses in the vast majority of cases. Although B19V has been linked to elevated liver transaminases, acute and chronic hepatitis, and even fulminant liver failure in very rare situations, hepatitis it causes remains largely unrecognized (8). In adults, B19V infection is non-permissive and may lead to life-threatening consequences including liver failure or bone marrow failure even if B19V receptors are present on fetal hepatocytes (9). However, the mechanism by which Human Parvovirus B19 causes hepatic injury remains unknown, despite the fact that the non-structural-1 (NS1) and VP1 unique region (VP1u) proteins of B19V have been proven to generate direct hepatic damage in mice (8,10).

Aim: Investigate the correlation between the presence of Parvovirus (B19) and socio-demographic characteristics (such as sex, age, region of residence, etc.) in patients with viral hepatitis.

Study population and methods:

From August 2022 to the end of February 2023. This study included the collection of serum samples for the detection of human Parvovirus antigen in 60 patients with viral hepatitis. Also included was a control group consisting of 30 individuals of different ages who did not have viral hepatitis. All these serum samples are detected for Parvovirus antigen by the enzyme-linked immunosorbent assay method. All the patients who had dialysis centers were diagnosed in Al-Hussein Teaching Hospital, and the main blood bank was in Al-Muthana Governorate.

Inclusion criteria:

The inclusion criteria for patients have been categorized. The patients in this research have been categorized in accordance with the information that was sought from them. All of the patients in the sample had their information gathered, which includes their illness type, diagnosis, and family medical history as stated in the questionnaire. Age, sex, location, and profession are all examples of demographic data. Patients with prior COVID-19 infection either got the vaccination or did not get the vaccine.

Exclusion criteria

The included criteria in this study include a specific age group of patients and exclude patients aged 70 or older. Also, all patients suffering from autoimmune diseases or chronic diseases are excluded. Three ml of these blood samples are centrifuged at 4000 RPM for five minutes and then separated into two parts: 0.2 ml for virus detection was determined by Enzyme-Linked Immuno-Sorbent Assay antigens according to the manufacturer's instructions (BT LAB Bioassay Technology Laboratory, China).

Statistical Analysis

Microsoft Office Excel 2010 and SPSS version 23 were used for data collection, summarization, analysis, and presentation. Quantitative (numerical) variables were first tested for normality with the Kolmogorov-Smirnov chi-squared statistic test, and then normally distributed numerical variables were expressed as mean (an index of central tendency) and standard deviation. (an index of dispersion). Specifically, the current study used the statistical analyses: The Chi-square test was replaced by Fischer's exact test after it was shown to be invalid. A *P*-value of 0.05 or below was regarded to indicate statistical significance.

Ethical consideration:

Participation in the study was subjected to an informed consent an informed consent was obtained from all participants; patients and control group). All participants (patients) had viral hepatitis. This protocol was approved by the Board of the Research Ethics Committee of the Health Office in Al-Muthana Governorate, according to the ethical number (No. 866 on 1/8/2022).And all samples taken from those patients are within the direct supervision of specialist physicians in hospitals.

Results

According to the findings of the current study, 10% of viral hepatitis patients were infected with human Parvovirus B19. The findings also revealed a non-significant difference in the distribution of viral infection by illness type ($p > 0.05$), as can be seen in Table 1.

Table (1): The frequency distribution of results of HPV antigen ELISA detection according to study groups.

Elisa	Control Group *N* = 30	Viral Hepatitis Group *N*= 60
Positive	**0 (0.0 %)**	**6 (10.0 %)**
Negative	**30 (100.0 %)**	**54 (90.0 %)**
***P*-Value**	**Reference**	**0.173 F Ns**

The present study found that males were more likely to be infected with human Parvovirus B19 than females were across all demographics. Table 2 showed that the findings also found no statistically significant variation in the sex distribution of viral infections both within and across the groups.

Table (2): Antigen detection of human Parvovirus B19 according to disease types and sex.

Group	Sex	Positive-HPV *N* (%)	Negative-HPV *N* (%)	Total *N*	*P*-Value
Viral Hepatitis Group	**Male**	**4 (13.3 %)**	**26 (86.7 %)**	**30**	**0.671 F NS**
	Female	**2 (6.7 %)**	**28 (93.3 %)**	**30**	

F: Fischer exact; ***n***: number of cases; **NS**: not significant

The results showed no significant association with residence in all cases of viral hepatitis, as shown in Table 3.

Table (3): **Antigen detection of human Parvovirus B19 according to disease types and residence**.

Group	Residence	Positive-HPV *N* (%)	Negative-HPV *N* (%)	Total *N*	*P*-Value
Viral Hepatitis Group	**Urban**	**3 (13.0 %)**	**20 (87.0 %)**	**23**	**0.666 F**
	Rural	**3 (8.1 %)**	**34 (91.9 %)**	**37**	

F: Fischer exact; ***n***: number of cases; **NS**: not significant

The results showed no significant association with COVID-19 infection in all cases of viral hepatitis, as shown in table 4.

Table (4): Antigen detection of human Parvovirus B19 according to COVID-19 infection.

Disease Type	Covid-19 Infection	Positive-HPV *N* (%)	Negative-HPV *N* (%)	Total *N*	*P*-Value
Viral Hepatitis Group	Positive	0 (0.0 %)	4 (100.0 %)	4	1.000 F NS
	Negative	6 (10.7 %)	50 (89.8 %)	56	

F: Fischer exact; ***n***: number of cases; **NS**: not significant

The results showed no significant association with COVID-19 vaccination in all cases of viral hepatitis, as shown in table 5.

Table (5): Antigen detection of human Parvovirus B19 according to COVID-19 vaccination.

Disease Type	Covid-19 Vaccine	Positive-HPV N (%)	Negative-HPV N (%)	Total N	P-Value
Viral Hepatitis Group	Positive	2(14.3 %)	12 (85.7 %)	14	0.617 F NS
	Negative	4 (8.7 %)	42 (91.3 %)	46	

F: Fischer exact; ***n***: number of cases; **NS**: not significant

The present research found that the number of patients infected with Human Parvovirus B19 was similar between those with Hepatitis B and those with Hepatitis C, whereas the number of patients infected with both Hepatitis viruses was not significantly different. Table 6 further shows that there was no statistically significant variation in the prevalence of Hepatitis infections across different viral subtypes.

Table (6): Rate of antigen detection of HPV according to ELISA with respect to type of viral hepatitis

Viral Hepatitis	Total	Positive Antigen		Negative Antigen		*P*
		N	%	*N*	%	
HBV	15	3	20.0	12	80.0	0.159 F NS
HCV	44	3	6.8	41	93.2	0.328 F NS
HBV-HCV	1	0	0.0	1	100.0	1.000 F NS

F: Fischer exact; ***n***: number of cases; **NS**: not significant

Table 7 displays the age-related HPV antigen detection rate using ELISA; the result found no statistically significant difference in mean age between cases with positive detection and those with negative detection in patients with viral hepatitis.

Table (7): Rate of antigen detection of HPV according to ELISA with respect to age.

Group	Age	Positive	Negative	*P-Value*
Viral Hepatitis Group	**Mean ±SD**	**28.00 ±8.05**	**28.76 ±13.55**	**0.894 I NS**
	Range	**20-36**	**3-68**	

I: Independent samples *t*-test; ***n***: number of cases; **NS**: not significant

Discussion

In this study, the proportion of patients with viral hepatitis who had positive antigens for Parvovirus B19 was 10%. In one previous study, by (11), anti-B19V IgM and IgG antibodies were found in the sera of 32% and 33% of HBV-infected patients, respectively; in HBV/HCV co-infected individuals, these numbers were 35% and 47%., these numbers were 35% and 47%.. Therefore, the rate in this study is far less than that of previous studies, and this discrepancy can be attributed to variations in detection methods, as stated previously.

Also in regard to the results of the current study observation, (12) and (13) found that there is a significant difference in the rate of infection with HPV according to sex based on the ELISA IgM and IgG detection methods in patients with viral hepatitis.

According to the available data taken from previous studies, there was no linkage or reference that linked the association among viral hepatitis (B, C, or combined) with HPV infection and residence. Therefore, it is not possible to compare this particular item of the current study with other previous studies. The lack of a significant association between residence and HPV infection in hepatitis in the recent study may be explained by the fact that the immune status in patients residing in rural and urban areas is essentially the same, so there is no higher susceptibility to infection can happen.

This point of the recent study can be considered novel because, to our best knowledge, no previous research has dealt with the association between HPV infection and COVID-19 in the general population or in a particular subset of them, including viral hepatitis.

In this study, it was found that there is no significant association between HPV infection rate and type of viral hepatitis (B, C, or combined). The results of the current study are similar to those of (12). The lack of a significant difference may be attributed to the fact that both viruses (HBV and HCV) cause the same magnitude of reduced immune response in both types of viral hepatitis.

Unfortunately, there is no previous data related to the age incidence of HPV with respect to viral hepatitis, but the results of the recent study indicated that the change in immunity will not increase susceptibility to infection with increasing age in those conditions.

Conclusions:

According to the results of this study, it is concluded firstly, the presence of human Parvovirus in patients with viral hepatitis was not influenced by most of the demographic factors studied. As it is found no significant differences in the detection rate of human Parvovirus among patients in terms of sex, age, residence, COVID-19 infection, or vaccination against COVID-19. 3- The study did find a 10% detection rate of human Parvovirus protein antigen among patients infected with hepatitis B and C viruses. Overall, the study suggests that viral hepatitis may play a role in the prevalence of human Parvovirus B19, but further research is needed to fully understand the direct relationship between the two viruses which affect their prevalence in humans.

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