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RESEARCH ARTICLE
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An exploratory study to know the effects of Helicobacter pylori on (Hepatitis C Virus) patients in Iraq

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ABSTRACT

This study aims to investigate the effects of Helicobacter pylori on hepatitis C virus (HCV) patients in Iraq. The demographic data and information were gathered from various hospitals to study 35 cases, under two groups, H. pylori+ (20 patients) and H. pylori− (15 patients). The statistical relationships were analysed using the IBM SOFT SPSS 25 data analysis programme, to know the effect of H. pylori on HCV patients in Iraq and were represented by mean SD. The mean SD to the age of the patients participating in this study was 44 + 8.2 and a higher BMI was observed in H. pylori+ patients (30.33 ± 2.9). The patients were distributed based on the severity of fibrosis into two groups: no cirrhosis for 10 patients (P% = 28.5%) and HCV for 25 patients (P%= 71.42%). The mean + SD for the no-fibrosis group was 4.4 + 1.2 and the HCV group was 12.9 + 5.5. The study found a statistically significant relationship between H. pylori and HCV patients at a level of 0.0023 with R corrections of 0.083.

Keywords: *Helicobacter; pylori; Hepatitis C virus; BMI; microaerophilic*

INTRODUCTION

Helicobacter pylori is a flagellated helical bacterium about 3.5 μm long and 0.5 μm wide that moves freely along the stomach wall.1,2 H. pylori infection occurs when the gram-negative, microaerophilic bacteria infects the stomach and duodenum.3–5 It is the causative agent for chronic gastritis with an acute period lasting about 10 days.

A literature study on a small group of patients with the same comorbidities revealed the link between the bacteria and gastric cancer development.6 Studies revealed that H. pylori infection plays a role in the development of more severe outcomes in patients with hepatitis C virus (HCV). Ponzito et al. (2000) reported the presence of antibodies to H. pylori in 77% of cirrhotic patients infected with HCV and in 59% of healthy subjects examined. Similar results were obtained by R. Pellicano et al. (2000), S. J. Konturek et al. (2003) and P. Stalke et al. (2005). This demonstrated the prevalence of more severe outcomes such as cirrhosis among H. pylori infected viral hepatitis patients, compared to control groups.7–10 V. Lonngren et al. (2009) studied the presence of antibodies to EHC antigens among people infected with HCV and found that 18% of patients examined had antibodies to H. pullorum and in 8% of patients.11,12

This study aims to determine the relationship between H. pylori and HCV in patients from different hospitals in Iraq between 2019 and 2020. A higher prevalence of H. pylori was found in HCV patients compared to the control group (patients without HCV) (64.5% vs. 53.2%). The results with a significant difference ($P = 0.003$) prove the existence of a relationship and the prevalence of negative symptoms in patients.13

ETHICAL APPROVALS

The study obtained permission from the hospitals to gather the data on patients and use the hospitals' technology and capacity for diagnostic support in determining H. pylori infection and hepatitis of different etiologies.

MATERIALS AND METHOD

An exploratory survey was conducted in Iraq to investigate the effects of H. pylori on HCV patients. The patient information was collected from different hospitals and a total of 35 cases were included. These cases were divided into two groups, namely H. pylori+ (patient group; 20 patients) and H. pylori− (control group; 15 patients).

Patient-specific primary and demographic data were gathered, including age, weight, height, body mass index and biochemical characteristics. The patients were also categorised based on age groups ranging from 40 to 55 years old for H. pylori+ and 35–50 years for H. pylori−. Patients were distributed based on the severity of cirrhosis into two groups: no cirrhosis for 10 patients and HCV for 25 patients.

The incubation period for hepatitis C ranged from 2 weeks to 6 months. Approximately 80% of the infected individuals do not experience any symptoms after the initial infection. Jaundice, abdominal pain, weariness, lack of appetite, nausea, vomiting and black urine may be present in severe cases.

Once hepatitis C infection has been identified, the severity of liver damage (cirrhosis) should be assessed through liver biopsy. The level of liver damage is then utilised to inform treatment and management choices.

Statistical analysis

In this study, statistical relationships were examined to determine the kind of relationship between H. pylori and HCV in patients and is represented by mean SD. The results were analysed using the IBM SOFT SPSS 25 data analysis tool. The P-value was calculated using the Chi-square test, to understand the results of the research and determine the fate of the hypothesis scale (5%).

RESULTS

Table 1 shows the distribution of patients with H. pylori+ according to age, N = 20. The ages ranged between 40 and 55 years. Eight patients were in the age range of 50–55 years (40%), seven patients were in the age range of 45–49 years (35%) and five patients were between 40 and 44 years (25%).

TABLE 1. Distribution of patients with H. pylori+ according to age, N = 20.

	f	P%	Chi-square
40–44	5	25	5.56
45–49	7	35	
50–55	8	40	

Table 2 shows the distribution of control group with H. pylori− according to age, N = 15. The patients ranged in age from 35 to 50 years. Six patients were in the age range of 40–44 years (40%), five patients were in the age range of 45–50 years (33.3%) and four patients were between 35 and 39 years (26.6%).

TABLE 2. Distribution of control group according to age, N = 15.

	Frequency	Percentage	Chi-square
35–39	4	26.6	2.76
40–44	6	40	
45–50	5	33.3	

Table 3 lists the distribution of patients of both groups based on sex.

TABLE 3. Distribution of patients according to sex, N = 35.

Sex	Patient, N = 20	Control, N = 15	P
Male	12 (60)	11 (73.3)	
Female	8 (40)	4 (26.6)	

Table 4 displays the diagnostic test results for both groups.

TABLE 4. Results related to the demographic and biochemical characteristics of the cases of this study.

Variable	Patient	Control	P
ALT (U/L)	50.3 ± 2.2	33.1 ± 3.2	0.01
BMI	30.33 ± 2.9	28.2 ± 3.1	0.83
AST (U/L)	49.6 ± 4.5	29.8 ± 4.9	<0.001
Albumin (g/L)	3.11 ± 0.034	4.56 ± 0.045	0.11
Platelets	177.1 ± 6.8	242.2 ± 8.9	0.002
Serum creatinine (mg/dL)	1.221 ± 0.91	0.99 ± 0.67	0.65
eGFR	98.2 ± 4.5	84.1 ± 8.2	<0.001
Hypertension (%)	12 (60)	8 (53.3)	0.03
Diabetes mellitus (%)	8 (40)	7 (46.6)	0.99

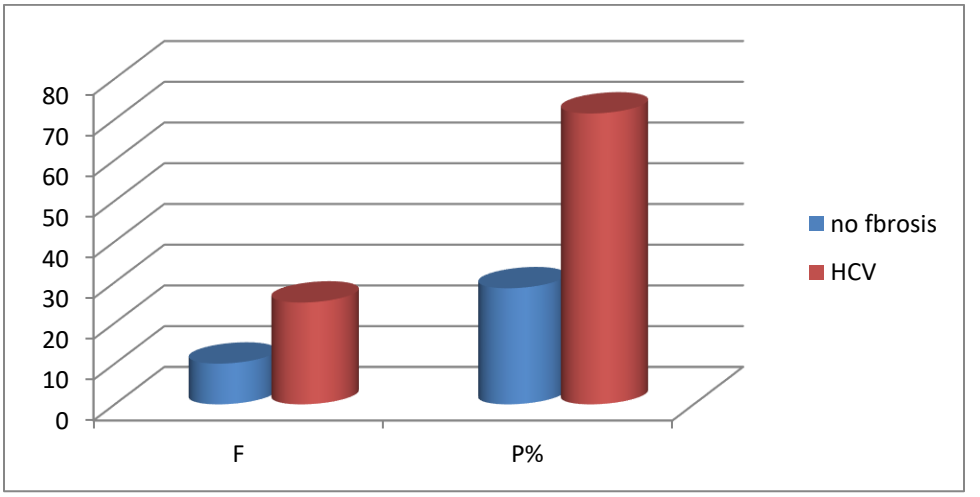

FIGURE 1. Distribution of cases according to HCV.

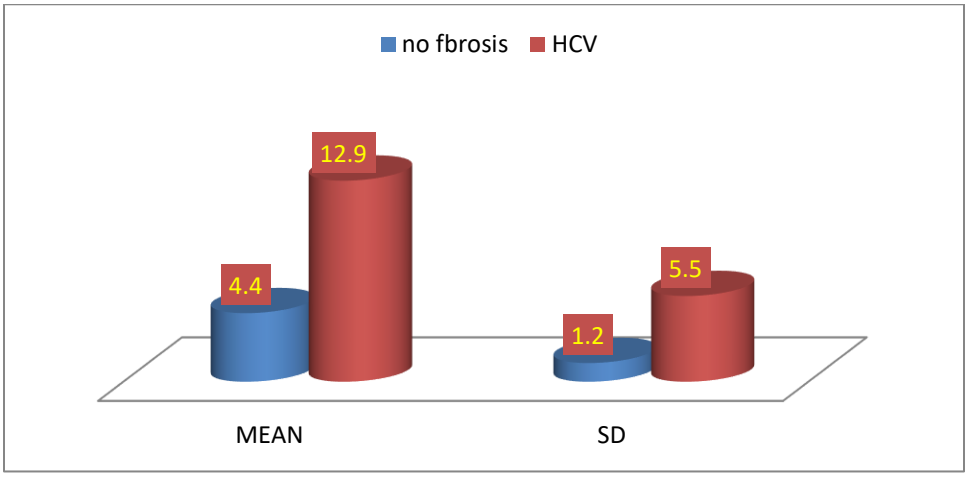

FIGURE 2. Mean ± SD of fibrosis score.

Table 5 displays the diagnostic test results for the patient group, infected with both H. pylori and HCV. Table 6 shows the correlation between H. pylori and HCV, while Table 7 lists the results of the logistic regression analysis of the risk factors of this study.

TABLE 5. Biochemical results for patients with H. pylori and HCV infection.

Variable	Patient	Control	P
ALT (U/L)	51.6 ± 1.9	31.6 ± 2.2	0.03
BMI	31.3 ± 1.9	29.1 ± 2.9	0.44
AST (U/L)	50.2 ± 3.4	30.1 ± 2.9	<0.001
Albumin (g/L)	3.22 ± 0.06	4.56 ± 0.07	0.44
Platelets	180.2 ± 6.8	246.6 ± 6.6	0.001
Serum creatinine (mg/dL)	1.25 ± 1.1	0.98 ± 0.88	0.81
eGFR	101 ± 3.8	80.9 ± 11.1	<0.001

TABLE 6. Pearson correlation analysis between H. pylori and HCV.

Variable	H. pylori	HCV
R correlation	1	0.083*
Sig.	0.023	–
Number	35	35

TABLE 7. Logistic regression analysis of risk factors on the patients of this study.

Variable	CS-95%	P
Age	1.5 (1.1–1.9)	0.001
Fibrosis severity	2.8 (1.3–3.7)	0.0009
ALT	1.7 (1.3–2.2)	0.001
AST	2.1 (1.4–2.7)	0.001

DISCUSSION

A total of 35 patients were studied. IBM soft SPSS and Microsoft Excel 2013 were used for the statistical analysis of parameters. The mean SD to the age of the patients participating in this study was 44 + 8.2 and a higher BMI was observed in H. pylori+ patients (30.33 ± 2.9).

Although for centuries bacteria were known to infect the human stomach, it was not considered in medicine until 1983, when H. pylori was rediscovered by Warren and Marshall.3 The population genetics of H. pylori mimics human genes. Patients likely acquired H. pylori very early in their history.

The results of this study indicate that there is a close and direct relationship between the frequency and prevalence of H. pylori in patients suffering from HCV.14,15 This finding aligns with previous research, such as the study by Ranaud Don (2000), who found a close correlation between H. pylori and HCV.

Most patients infected with HCV do not develop symptoms after infection and are in the early stages of the disease. The symptoms may show after a period ranging from 15 days to 6 months after the virus entered the body.16

This is because HCV frequently changes its structural features after entering the body, making it difficult for the body's immune system to recognise and eliminate it.

Routine blood tests are needed to detect elevations in liver enzymes ALT and AST, along with subsequent tests. The study revealed a valid association between the levels of liver enzyme (ALT, AST) and the severity of hepatitis; that is, the patient with elevated levels of liver enzymes are infected with HCV, compared with the control group.17,18 A liver needle biopsy is usually required to understand the severity of fibrosis in patients with HCV.

The association between H. pylori and HCV was found to be statistically significant for the 35 patients at a level of 0.0023 with R corrections of 0.083. This explains the negative impact on the development of the disease. Logistic analysis was conducted to analyse the risk to patients, which showed that there is a higher risk of fibrosis in patients with both infections [CS95% 2.8 (1.3–3.7) at P = 0.0009].

CONCLUSION

In summary, this study found a positive and direct correlation between H. pylori and HCV, which is statistically significant ($P < 0.05$), through independence test (Chi-square at 95%; $\alpha = 0.05$) and odds ratio. Logistic analysis also revealed that there is a higher risk of fibrosis in patients with both infections. However, this study did not provide information on the epidemiology and clinical manifestations of HCV in different patients, which is a limitation of this study.

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