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Detection of IgG and IgA Antibodies of *Helicobacter Pylori* Seroprevalence in Hepatitis C Virus Infection

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Abstract

Background: Globally, *Helicobacter pylori (H. pylori)* and hepatitis C virus (HCV) are the most common bacterial infection and viral infection respectively that are associated with wide range of complications include liver cirrhosis and liver failure.

Objective: This study was designed to detect sero prevalence of *H. pylori* IgG and IgA antibodies among patients with HCV.

Subjects and Methods: Two hundred HCV patients of both sexes participated in this study, they were enrolled in two equal groups where group (A) include HCV patients with liver cirrhosis, while group (B) include HCV patients without liver cirrhosis.

Results: The findings of this study revealed that the *H. pylori* infection prevalence was a higher in group (A) that included HCV patients with liver cirrhosis than group (B) that included HCV patients without liver cirrhosis, also the sero-prevalence test of IgG and IgA showed a high significant difference between both groups.

Conclusion: *Helicobacter pylori* infection antibodies are more prevalent among cirrhotic HCV patients than without cirrhosis.

Keywords: Chronic hepatitis C; Cirrhosis; Helicobacter pylori; Liver disease

Introduction

Helicobacter pylori (H. pylori) constitute the commonest infection among human being as it affects about 2/3 of population worldwide [1]. In addition, more than 185 million subjects are affected with chronic hepatitis C virus (HCV) infection worldwide [2] and cirrhosis is the late stage of progressive hepatic fibrosis among patients with hepatitis, where there are many risk factors that lead to progression of hepatitis is important for prevention of its occurrence [3]. While hepatitis C virus infection (HCV) and hepatitis C virus infection (HBV) are of these factors, excess alcohol consumption, male gender and schistosomiasis [4].

Helicobacter pylorus is common to be associated with pathogenesis of hepatic disorders [5-7] as it play an essential role in progression of viral hepatitis lesion to liver cirrhosis [8] as *H. pylori* secrete some hepatic specific toxin that damage liver parenchyma and causes hepatocyte necrosis that induce liver cirrhosis and hepatic cancer [9-12].

In the other hand, patients with hepatic cirrhosis are at risk for some gastrointestinal disorders where *H. pylori* infection is a principal factor in gastric ulcer pathogenesis [13]. In addition, *H. pylori* antibodies were detected in HCV patients with hepatic cancer [14]. Moreover, about 30% of patients suffer from gastric ulcer and hepatic cirrhosis had *H. pylori* infection [15]. Therefore, this study was designed to detect seroprevalence of *H. pylori* antibodies among patients with HCV.

Subjects and Methods

Two hundred consecutive HCV-positive patients (124 male and 76 female, age range 32–50 years) attending outpatient clinic, King abdulaziz university teaching hospital were enrolled in the present study to determine anti-*H. Pylori* antibodies presence in their serum. All participants underwent biochemical, histological and clinical evaluation to detect hepatic cirrhosis. Exclusion criteria included other hepatic disorders as hepatitis B virus infection (HBV), diabetes, autoimmune and endocrine disorders. They were enrolled in two study groups: Group (A) one hundred HCV-positive cirrhotic patients (64 male and 36 female, age range 33–48 years) and Group (B) one hundred HCV-positive non-cirrhotic patients (60 male and 40 female, age range 32–49 years). The Scientific Ethical Research Committee of Faculty of Applied Medical Sciences approved this study.

All participants singed the consent form before sharing in the present study.

Detection of serum anti-*H. pylori* (IgG and IgA) antibodies was done with a commercial enzyme-linked immunosorbent assay (ELISA) (Helori-test® Eurospital, Trieste, Italy) . However, ELISA (Axsym System, Abbott Diagnostics) and an RIBA (RIBA II, LIA, HCV3, Sorin, Saluggia, Italy) were used to diagnose HCV infection and confirmed using polymerase chain reaction (PCR) to confirmed circulating HCV-RNA detection.

Results

Table 1: Baseline and laboratory data of all participants.

Demographic and Laboratory Data	Median (Range)
Age mean age±SD (range)	40.32 ± 6.45 (32-50)
Gender (male/female)	124/76
ALT mean ± SD (range)	58.42 ± 13.51 (30-87)
AST mean ± SD (range)	47.31 ± 11.35 (25-65)
Total bilirubin mean ± SD (range)	0.83 ± 0.17 (0.52-1.1)
Platelets	167,460 (12,000-225,000)
Albumin	4.7 (4.0-5.2)
HCV RNA titre	340,000 (50,700-7430,000)

Two hundred HCV patients were selected on referral to Gastroenterology and Hepatology Department, King Abdulaziz University Teaching Hospital, all these patients were anti HCV positive by ELISA. Patient characteristics (Table 1). The findings of this study revealed that the *H. pylori* infection prevalence was higher in group (A) that included HCV patients with liver cirrhosis than group (B) that included HCV patients without liver cirrhosis, also the sero-prevalence test of IgG and IgA showed a high significant difference between both groups (Table 2 & 3).

Table 2: Number and percentage of *H. Pylori* IgG and IgA positivity and negativity in the cirrhotic and non-cirrhotic groups.

	Number (%)		T-value	P-value
	Cirrhotic group	Non-cirrhotic group		
IgG positive	100 (100%)	84(84%)	10.43	P<0.05
IgG negative	0 (0%)	16(16%)		
IgA positive	72(72%)	48(48%)	12.67	P<0.05
IgA negative	28(28%)	52(52%)		

Table 3: Comparison between H. Pylori IgG and IgA serological test in the cirrhotic and non-cirrhotic groups.

	Mean +SD		T-value	P-value
	Cirrhotic group	Non-cirrhotic group		
IgG	55.72± 11.65	41.87 ± 10.42	7.51	P<0.05
IgA	51.43 ± 10.86	24.22 ± 9.77	9.86	P<0.05

Discussion

Hepatitis C virus infection lead to liver cirrhosis in only 10% of patients [16]. However, *H. Pylori* presence in hepatic tissue could occur via a retrograde route from the duodenum or through the portal circulation. However, Rocha et al. [8] suggested that liver structural changes induce *H. Pylori* presence when liver cirrhosis occurs [8]. In this setting, a retrograde route, from the duodenum to the liver might be the underlying mechanism for *H. pylori* to colonize in liver tissue. It is worth noting that our results come in agreement Petrenkienë et al. [17] as all of our patients with *H. pylori* in liver were sero positive for anti-HP antibodies and patients negative for *H. pylori* DNA in liver tissue were also negative to anti-HP antibodies [17].

In our study, *H. pylori* prevalence was investigated using IgG and IgA as indicators for infection among patients with HCV either with cirrhosis or without cirrhosis this is consistent with many previous studies [13, 18-20]. Also, in our study there were higher prevalence of *H. pylori* IgG and *H. pylori* IgA in cirrhotic than non-cirrhotic patients, these data agreed with many studies [21,22]. Moreover, these findings agreed with Pellicano et al. [22] mentioned that 89% of HCV cirrhotic patients had *H. pylori* antibodies [23]. In addition, Spinzi et al. [24] found that 86.5% of HCV cirrhotic patients had *H. pylori* antibodies with high prevalence of gastroduodenal ulcer in cirrhotic patients [24]. Similarly, Ponzetto et al. [25] proved that, the prevalence of *H. pylori* antibodies was 79.5% among HCV cirrhosis patients [25]. While, Vorobjova et al. [26] stated that antibody levels of H. bilis/H. hepaticus were increased in sera of nighty patients with different chronic liver diseases [26]. However, Shavakhi et al. [27] reported that 73% of cirrhotic patients and 52% of control group had IgG antibody to *H. pylori* [27]. While, Nilsson et al. [28] stated that high level of H. hepaticus antibody in 8% of chronic liver diseases patients [28]. Also, Queiroz et al. [29] found an association between cirrhosis among HCV and *H. pylori* infection [29]. Moreover, this was in agreement with El-Masry et al. [30] who stated that 55.6% of Egyptian HCV patients had *H. pylori* as co-infection [30]. Moreover, Sathar and colleagues mentioned that reported that 44.3% of cirrhotic patients with portal hypertensive gastropathy had *H. pylori* antibodies [31]. Furthermore, Wang et al. [32] reported that HCV patients had higher prevalence of *H. pylori* than those without HCV [32].

The possible mechanism of liver dysfunction due to *H. pylori* may be due to release of hepatic-specific toxin produced by several Helicobacter spp. that may have an aetiological role in progression of HCV-related hepatitis [33]. In the other hand Innocenti and colleagues said that the possible role of *H. pylori* playing in developing or progression of CHV cirrhosis because of the systemic inflammation and subsequent hepatic damage triggered via autoimmune response induced by *H. pylori* which was evident by higher seroprevalence of anti-*H. pylori* antibodies in HCV cirrhotic patients [34].

Conclusion

Helicobacter pylori infection antibodies are more prevalent among cirrhotic HCV patients than without cirrhosis.

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