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Research Article

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The Relation between Helicobacter Pylori Infection, Serum Ammonia and Hepatic Encephalopathy in Yemeni Cirrhotic Patients

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Abstract

Introduction: *Helicobacter pylori (H. pylori)* infection in the group of patients with liver cirrhosis may affect the exacerbation of inflammatory injuries in the stomach, which could directly, and indirectly, lead to loss of liver function.

Aim of the study: Assessment of possible relation among *H. pylori* infection, serum ammonia, and hepatic encephalopathy in the middle of patients with liver cirrhosis.

Patients and methods: This study was conducted in the Hepatobiliary-Gastroenterology Specialized Research Center in Sana'a City Yemen. This is a prospective study and patients from April 2008 to April 2014 were collected. All patients with established *Chronic Liver Disease* (CLD) with hepatic encephalopathy.

Result:A total of 78 patients with liver cirrhosis included in the present study, there were selected, (n=42; 54%) were males and (n=36; 46%) were females. Clinical and demographic characteristics of the study patients were shown H.E grade had the most significant relationship with *H. pylori* state (p=0.000). There was a significant relation between stage A, B, C, and *H. pylori* state (p=0.09).

Conclusion: This study comes to more reliable and confident that is a highly significant association among *H. pylori* infection, Hepatic Encephalopathy, stage A, B, C, and serum ammonia in cirrhotic patients.

Keywords: Helicobacter pylori infection; Hepatic encephalopathy; Serum ammonia; cirrhotic patients

Abbreviations

CP: Child-Pugh; CLD: Chronic Liver Disease; HBV: Hepatitis B Virus; HCV: Hepatitis C Virus; PBC: Primary Biliary Cirrhosis; H.E: Hepatic Encephalopathy; H. pylori: Helicobacter Pylori; LC: Liver Cirrhosis

Introduction

Helicobacter pylori bacteria are rich in the urease enzyme and are recognized to produce ammonia from urea that is fast absorbed from the gastric lumen into circulation. Infection with these bacteria has been shown to be related to elevated blood ammonia levels and repeated attacks of obvious hepatic encephalopathy [1]. The most common biochemical irregularity in patients with chronic *Hepatic Encephalopathy*(HE) is hyperammonemia [2,3]. Elevated blood ammonia levels have similarly been implicated in the causality of HE [4]. Infection with these bacteria is one of the most common in the world. In highly developed countries, 50% of the population is infected, whereas in the developing countries the percentage reaches as much as 90% [5]. *H. pylori* infection in the assembly of patients with liver cirrhosis may impact the exacerbation of inflammatory injuries in the stomach, which could directly, and indirectly, lead to deficiency of liver function. This is mainly dangerous in patients with progressive liver disease [6].

In this regard, two theories have been postulated regarding the relationship between *H. pylori* infection and *HE* which showed that ammonia has still an essential role in the pathogenesis of hepatic encephalopathy [7]. *HE* in its numerous grades, according to the West Haven Criteria, is assessed to be present in 30%-45% of individuals with *Liver Cirrhosis* (LC), while approximately 60%-80% of these individuals show cognitive dysfunction in specified testing. All clinical appearances of HE is considered by the nonappearance of any physical cerebral changes and are possibly changeable by adequate therapeutic management [8,9]. Only a minority of gastroenterologists are prepared to test for HE. In a survey conducted in the USA, 38% of the members of the American Association for the Study of Liver Diseases had never tested for it, although the majority (84%) is aware of the medical problem and the need to test for it [10]. There are only a few data published so far from small prospective clinical studies on the role of *H. pylori* in subclinical HE, and their findings are inconsistent [11,12]. Elevated levels of ammonia could deteriorate hepatic encephalopathy. As a result, it can be suggested that *Helicobacter Pylori* infection may possibly contribute to the progress of hepatic encephalopathy [13]. A number of studies showed that ammonia levels both in gastric juice and blood were significantly developed in cirrhotic patients with *H. pylori* infection than those without [1,14]. The aim of our study was Assessment of possible relation among *H. pylori* infection, serum ammonia, and hepatic encephalopathy in the middle of patients with liver cirrhosis, and also an assessment of clinical and laboratory characteristics of cirrhotic patients (etiology, age, sex and stage of LC in relation to *H. pylori* infection.

Patients and Methods

This study was conducted in the Hepatobiliary-Gastroenterology Specialized Research Center in Sana'a City, Yemen. This is a prospective study and patients from April 2008 to April 2014 were collected. The total number of patients included in this study was 78 patients divided into age groups. They included 42 males and 36 females. According to the etiology of liver cirrhosis, the patients were categorized into the following groups: Group 1: HBV, Group 2: HCV, Group 3: schistosomiasis. Group 4: Autoimmune, Group 5: Idiopathic. Serum ammonia level was expressed as normal (Less than 60 micromol/L) were 10 patients — Mild elevation (60-100 micromol/L) were 38 patients — marked elevation (more than 100 micromol/L) were

30 patients. According to the stage of liver cirrhosis A, B, C, all patients with established *Chronic Liver Disease* (CLD) with HE. CLD was diagnosed on the basis of the clinical features like finger clubbing, palmar erythema, spider naevi, splenomegaly, hepatomegaly or shrunken liver, or the persistent elevation for more than 6 months of the liver enzymes. Plus a positive abdominal ultrasound for irregular liver margins, coarse liver appearance, and a dilated portal vein > 12mmor). To assess the severity of the liver disease, the patients were scored according to the Child-Pugh classification. This mark is established on the grade of encephalopathy, the existence of ascites, prothrombin time, and the serum levels of bilirubin, and albumin. Accordingly, the patients had either compensated liver disease (Class A, 5-6 points), moderate liver disease (Class B, 7-9 points), or severe liver disease (Class C, 10-15 points).

Diagnosis of viral etiology based on *Hepatitis B surface antigen*/ *Hepatitis C Virus* (HBsAg/HCV Ab PCR while Autoimmune depends on sever sero immune markers — ve viral marker hypergamaglobulinemia response to immunosuppressive therapy Schistosomiasis a liver disease depend on the sch.Ag, rectal snip. Whereas idiopathic CLD based on —ve viral, autoimmune markers, -ve evidence for schistosomiasis normal metabolic or even a liver biopsy. Assessment of HE was clinically as well as psychometric test diagnosis of *H. pylori* infection was made by stool Ag, *Universal Backup Tool* (UBT), Endoscopy with clo test or biopsyhistopathology.

Ammonia measurement

Fasting venous blood samples were obtained from each patient to measure ammonia concentration (micromole/L), according to the manufacturer's instructions.

Inclusion criteria

All Yemeni patients with liver cirrhosis complaining of manifestations suggestive of hepatic encephalopathy whatever the grade. These patients are suffering from liver cirrhosis of different stages and due to different etiologies.

Exclusion criteria

Patients with liver cirrhosis with HCC, advanced renal and cardiac diseases

Statistical Analysis

Data were stored and analyzed by the statistical program, *Statistical Packagefor the Social Sciences* (SPSS) Version 16. All the quantitative variables corresponding age, blood ammonia levels, and so out were analyzed for mean+standard duration. Frequencies and proportions were calculated for the quantitative variables like gender, *Child-Pugh grade*(CP grade). Hyperammonemia was stratified amongst disease severity grade (CP grade) to see effect modification. Statistical significance was established at p value<0.05 was taken as a criterion standard.

Results

A total of 78 patients with LC included in the present study, there were selected, (n=42; 54%) were males and (n=36; 46%) were females. Clinical and demographic characteristics of the study patients were shown *H. pylori* state (Figure 1) had the most significant relationship with H.E grade, stage A, B, C, and serum ammonia (p=0.000, p=0.009, p=0.041 respectively) (Table 1).

		H.pylori				p values
		Negative –		Positive +		
		Count	%	Count	%	
H.E	Grade 1	2	11%	32	53%	0.000*
	Grade 2	15	83%	18	30%	significant
	Grade 3	1	6%	10	17%	
Stage	A	6	33%	4	7%	0.009*
	B	7	39%	25	42%	significant
	C	5	28%	31	52%	
Serum Ammonia	Normal	5	28%	5	8%	0.041*
	Mild	8	44%	22	37%	significant
	Marked	5	28%	33	55%	

Table 1: Show the relation between H.E, stage, s. ammonia with *H. pylori* state.

Relation *H. pylori* with H.E grade 1, found patients (n=2; 11%) were negative, and (n=32; 53%) were positive. While in grade 2, found patients (n=15; 83%) were negative, and (n=18; 30%) were positive. Compared with grade 3, found patients (n=1; 6%) were negative, and (n=10; 17%) were positive (Table 1). Additionally, the present study shown H. E in grade 1 in autoimmune patients (n=3; 21%), HBV patients (n=2; 14%), HCV patients (n=12; 67%), idiopathic patients (n=8; 50%) and Schistosoma patients (n=9; 56%). On the other hand, H.E in grade 2 in autoimmune patients (n=6; 43%), HBV patients (n=11; 79%), HCV patients (n=3; 17%), idiopathic patients (n=6; 38%) and Schistosoma patients (n=7; 44%). This study found significant relationship between H.E and etiology of liver cirrhosis (p=0.005). Whereas shown non-significant relation among *H. pylori* infection with etiology of liver disease (p=0.250).Moreover,in the stage (A, B, C)

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there was non-significant correlation between Child-Pugh class and etiology in liver cirrhosis (p=0.920). Similarly, there was non-significant association between serum ammonia and etiology in liver cirrhosis (Table 2).

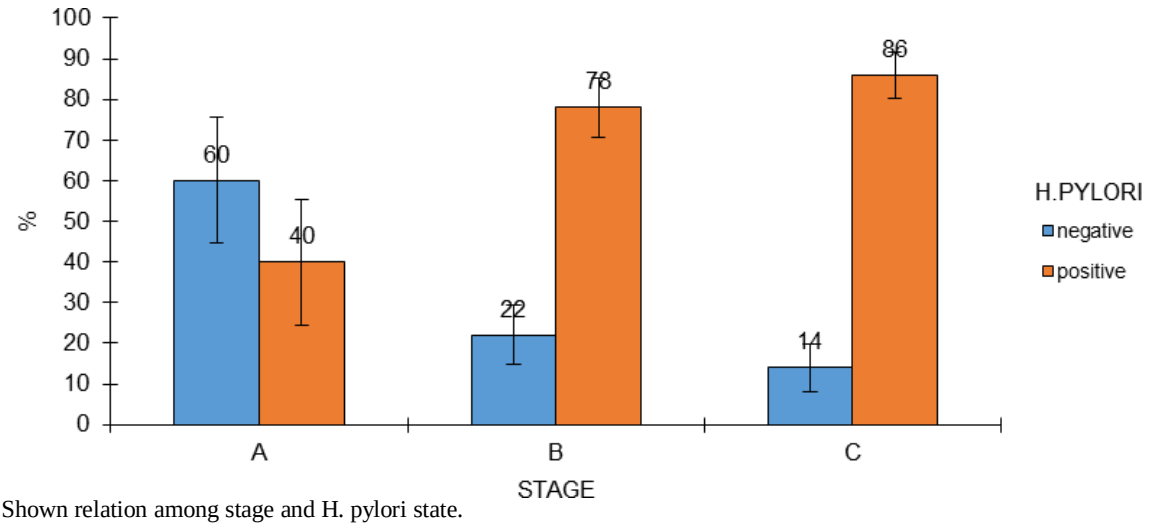

Figure 1: Shown relation among stage and H. pylori state.

In Figure 1: We found percentage relation *H. pylori* (positive +) with stage C were higher than stage B and stage C. whereas, percentage *H. pylori* (negative -) with stage C were lesser than stage A and stage B.

		ETIOLOGY										P values
		Autoimmune		HBV		HCV		Ideopathic		Schistosomal		
		Count	%	Count	%	Count	%	Count	%	Count	%	
H.E	GRADE 1	3	21%	2	14%	12	67%	8	50%	9	56%	0.005
	GRADE 2	6	43%	11	79%	3	17%	6	38%	7	44%	Significant
	GRADE 3	5	36%	1	7%	3	17%	2	13%	0	0%	
H. pylori	Negative –	4	29%	5	36%	1	6%	3	19%	5	31%	0.25
	Positive+	10	71%	9	64%	17	94%	13	81%	11	69%	Non-significant
Serum Ammonia	Normal	0	0%	3	21%	4	22%	1	6%	2	13%	0.373
	Mild	7	50%	5	36%	5	28%	9	56%	4	25%	Non-significant
	Marked	7	50%	6	43%	9	50%	6	38%	10	63%	
Stage	A	2	14%	2	14%	2	11%	3	19%	1	6%	0.92
	B	7	50%	6	43%	8	44%	4	25%	7	44%	Non-significant
	C	5	36%	6	43%	8	44%	9	56%	8	50%	

Table 2: The relation between H. pylori, H.E, stage, serum ammonia with etiology.

Despite the fact, there were non-significant among etiology (autoimmune, Hepatitis B virus (HBV), Hepatitis C Virus (HCV), idiopathic, Schistosoma), serum ammonia (Figure 2), H.E, H. pylori, and stage (Child-Pugh class),with age (Table 3). On the other hand, there was non-significant difference between H. pylori, etiology (autoimmune, HBV, HCV, idiopathic, Schistosoma), serum ammonia, H.E, and stage (Child-Pugh class), with sex (Table 4). The present study found non-significant in relation among patients H.E, and serum ammonia (p=0.435). This study shown relation serum ammonia level with H.E grade which shown percentage in grade 1 expressed as normal (Less than 60 micromol/L) were (n=3; 30%) patients — Mild elevation (60-100 micromol/L) were (n=15; 50%) patients — marked elevation (more than 100 micromol/L) were (n=16; 42%) patients. In the other hand, percentage in grade 2 expressed as normal (Less than 60 micromol/L) were (n = 4; 40%) patients — Mild elevation (60-100 micromol/L) were (n=13; 43%) patients — marked elevation (more than 100 micromol/L) were (n=16; 42%) patients. Moreover, percentage in grade 3 expressed as normal (Less than 60 micromol/L) were (n=3; 30%) patients — Mild elevation (60-100 micromol/L) were (n=2; 7%) patients — marked elevation (more than 100 micromol/L) were (n=6; 16%) patients (Table 5).

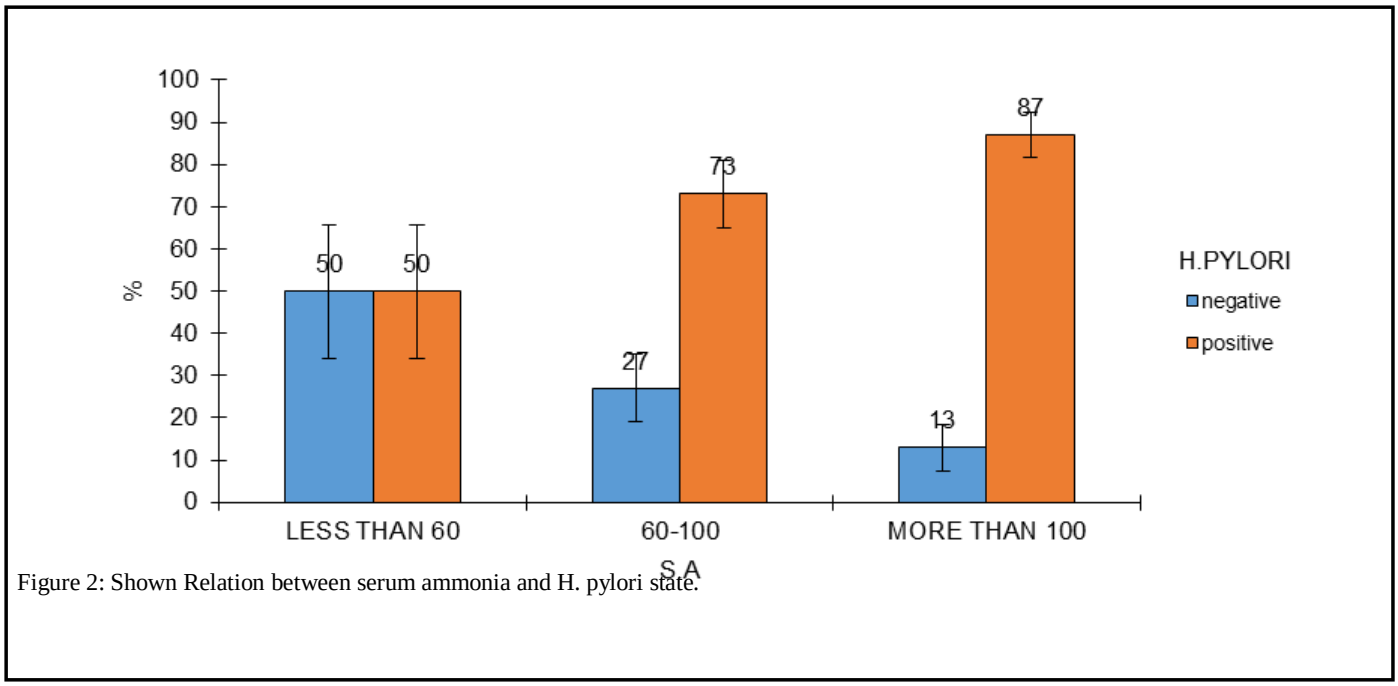

Figure 2: Shown Relation between serum ammonia and H. pylori state.

In Figure 2: We found percentage *H. pylori* (positive +) with serum ammonia more than 100 were 87%, less than 60 were 50% and 60 -100 were 73%. Compared, *H. pylori* (negative -) with serum ammonia more than 100 were 13%, less than 60 were 50% and 60-100 were 27%.

		AGE						p values
		20-60 years		fewer than 20 years		higher than 60 years		
		Count	%	Count	%	Count	%	
Etiology	Autoimmune	13	22%	1	50%	0	0%	0.178
	HBV	12	20%	0	0%	2	12%	Non-significant
	HCV	11	19%	0	0%	7	41%	
	Ideopathic	13	22%	0	0%	3	18%	
	Schistosomal	10	17%	1	50%	5	29%	
Serum Ammonia	Normal	6	10%	0	0%	4	24%	
	Mild	25	42%	0	0%	5	29%	0.334
	Marked	28	47%	2	100%	8	47%	Non-significant
H.E	Grade 1	27	46%	0	0%	7	41%	0.555

	Grade 2	24	41%	1	50%	8	47%	Non-significant
	Grade 3	8	14%	1	50%	2	12%	
H. pylori	Negative–	13	22%	0	0%	5	29%	0.6
								Non-significant
	Positive +	46	78%	2	100%	12	71%	
Stage	A	9	15%	0	0%	1	6%	
								0.839
	B	23	39%	1	50%	8	47%	Non-significant
	C	27	46%	1	50%	8	47%	

Table 3:Relation among H. pylori, H.E, stage, serum ammonia, etiology with age.

		SEX				**P-values**
		F		**M**		
		Count	**%**	**Count**	**%**	
H. pylori	Negative–	5	14%	13	31%	0.075
	Positive +	31	86%	29	69%	Non-significant
Etiology	Autoimmune	8	22%	6	14%	0.078
	Hbv	5	14%	9	21%	Non-significant
	Hcv	11	31%	7	17%	
	Ideopathic	9	25%	7	17%	
	Schistosomal	3	8%	13	31%	
Serum ammonia	Normal	3	8%	7	17%	0.268
	Mild	17	47%	13	31%	Non-significant
	Marked	16	44%	22	52%	
H.E	Grade 1	18	50%	16	38%	0.538
	Grade 2	13	36%	20	48%	Non-significant
	Grade 3	5	14%	6	14%	
Stage	A	5	14%	5	12%	0.763
	B	16	44%	16	38%	Non-significant
	C	15	42%	21	50%	

Table 4: shown relation among H. pylori, H.E, stage, serum ammonia and etiology with sex.

		Serum Ammonia						**p values**
		Normal		**Mild**		**Marked**		
		Count	Column%	Count	Column%	Count	Column %	
H.E	Grade 1	3	30%	15	50%	16	42%	0.435
	Grade 2	4	40%	13	43%	16	42%	Non-significant

	Grade 3	3	30%	2	7%	6	16%	

Table 5: Relation between H.E and serum ammonia.

Discussion

The pathogenesis of H.E is still poorly understood, but raised systemic ammonia concentration as a consequence of shunting of ammonia-rich portal blood away from the liver and impaired urea genesis have long been causally implicated in its development. *H. pylori* urease activity in the cirrhotic stomach has been proposed to represent a significant source of ammonia, contributing to the development of H.E. This study showed patients were H.E grade the most significant relationship with *H. pylori* state (p=0.000). Another study by DR. JAHANGIR LIAQUAT(15) et al. found the *H. pylori* was observed to be rising with the severity of hepatic encephalopathy [15]. Similarly, the study by El-seida [16], et al. exposed a highly significant association between *H. pylori* infection and MHE in cirrhotic patients [16].

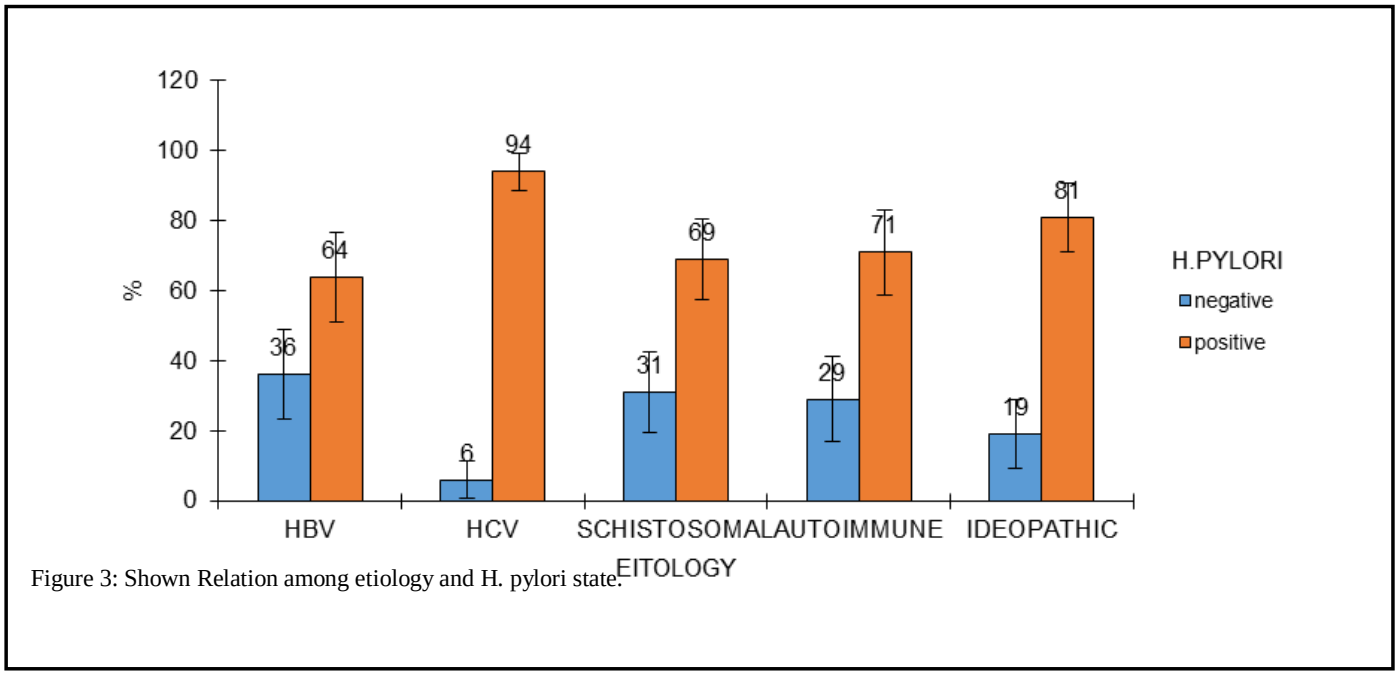

Figure 3: Shown Relation among etiology and H. pylori state.

However, the above study confirms with our study. In the present study, we found a significant association between stage (A, B, C) and *H. pylori* state. The Previous study by Sherweet M. Ibrahim [17] et al. found a non-significant difference in Child-Pugh class, hepatic encephalopathy grade and precipitating factors of HE between H pylori-positive and negative groups [17]. Whereas, our study found a significant relation between Child-Pugh class and *H. pylori* state (p=0.009). On the other hand, the previous study by Ibrahim [17] et al. opposed with our study. Additionally, the study by Masood [18] et al. found Patients with HE and *H. pylori* infection exposed a significant decrease in blood ammonia levels after anti-*H. pylori* treatment (p< 0.001) [18]. The current study found *H. pylori* and blood ammonia levels were significantly increased with the severity and mark of hepatic encephalopathy, which proposes that *H. pylori* infection may have a role in the pathogenesis of hepatic encephalopathy. Consequently, our study confirms with the above study by Masood et al. However, because of the inadequate number of published studies and procedural errors of the studies included in the past, more enormous randomized studies are necessary in order to supplementary confirm this association. But come to our study more reliable and confident. A Previous study by Pogorzelskaa [6] shown H. pyloriinfection was diagnosed in 69 (46.9%) patients, usually among those chronically infected with HBV or HCV. The incidence of *H. pylori* infection among patients with post-inflammatory liver cirrhosis was significantly higher (P=0.001), as compared with patients with alcoholic liver cirrhosis [6]. However, the previous study by Pogorzelskaa et al. opposite our study which found non-significant relation between *H. pylori* infection patients with etiology of liver cirrhosis. Another study by AvinashA[19] etal. Presence of MHE had a non-significant relationship with age, sex, Child-Pugh grade, and cause of cirrhosis [19]. But, different from our study which shown found a significant relationship between HE and causes of liver cirrhosis (p= 0.005).In our study, we found non-significant between stages, serum ammonia with the etiology of liver cirrhosis. On the other hand, the present study showed non-significant relation among H. pylori, H.E, stage, serum ammonia, etiology with age.Another study by Rekha [11] shown there was no statistically significant difference between the two groups (p > 0.05). The blood ammonia values are shown in Child-Pugh A, B, and C class of patients were non-significant different (p > 0.05) in the positive or negative of *H. pylori* infection [11]. However, the above study confirms with our study. The present study found non-significant relation among H. pylori, H.E, stage, serum ammonia and etiology with sex. Another study by Mohamed [20] shown non-significant groups regarding Age and Sex. This confirms with our study about non-significant for age and sex.The present study showed a non-significant relation between H.E and serum ammonia. It is concluded

that Helicobacter pylori do not contribute significantly to blood ammonia levels and the severity of hepatic encephalopathy which confirms with our study. In the present study shown Figure 3: relation *H. pylori* state with etiology which found percentage *H. pylori* (positive +) with HCV (94%) were higher than others etiology.

In Figure 3:Shown relation *H. pylori* state with etiology which found percentage *H. pylori* (positive +) with HCV (94%) were higher than other etiology. Compared, percentage (*H. pylori* negative -) with also HCV (6%) was lesser than others etiology but with HBV were higher than other etiology.

Compared, percentage (H. pylori-negative -) with also HCV (6%) was lesser than other etiology but with HBV was higher than other etiology. Moreover, Another study by Pogorzelskaa [6] et al shown Figure 4. Helicobacter pylori infection in different groups of patients. *Hepatitis B Virus(HBV)*; H*epatitis C Virus(HCV)*; *Primary Biliary Cirrhosis*(PBC) which found, hepatitis B virus higher than others [6].

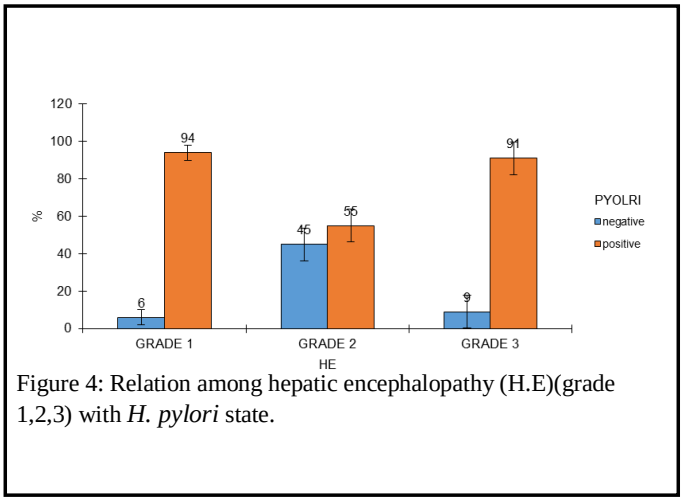

Figure 4: Relation among hepatic encephalopathy (H.E)(grade 1,2,3) with *H. pylori* state.

In Figure 4: This study found percentage relation *H. pylori* (positive +) with grade 1 were higher than grade 2 and grade 3. Whereas, percentage *H. pylori* (negative -) with grade 1 were lower than grade 2 and grade 3.

Conclusion

This study comes to more reliable and confident that is a highly significant association among *H. pylori* infection, hepatic encephalopathy, stage A B C and serum ammonia in cirrhotic patients. But found non-significant relation *H. pylori* infection with the etiology of liver disease. Whereas found a significant relationship between H.E and etiology of cirrhosis. Therefore, *H. pylori* infection is an effective curable risk factor for the clinical controlling of hepatic encephalopathy. There may be a role of anti-*H. pylori* therapy in patients of hepatic encephalopathy and should be studied additional.

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Authors' Contribution

Prof. Dr.Mansour A. Al-Amrani: Study concept and design, data acquisition, interpretation; manuscript drafting, study supervision, the idea of the manuscript, collection, writing and literature review.

Dr. Ali Al-Zaazaai: Revision of the manuscript for important intellectual content, analysis, and publishing.

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Conflict of Interest Statement

A statement is declared for no conflict of interest.

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