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A COMPARATIVE STUDY ON THE RELATION BETWEEN HEPATIC ENCEPHALOPATHY AND HELICOBACTER PYLORI SEROPOSITIVITY AT NISHTER HOSPITAL MULTAN

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Abstract:

Objective: *There is a shortage of availablity of awareness and knowledge on the incidence of (H. Pylori) Helicobacter pylori and its contribution to biliary tract disease incidence and liver pathology in human. We aimed at the assessment of the possible correlation between hepatic encephalopathy and seropositivity of the H. Pylori.*

Methodology: *This research was held on cirrhotic patients having HE (Hepatic Encephalopathy), cirrhotic patients who do not have HE incidence and control group that included healthy patients. The serological assessment was carried out in all the patients in order to determine the classification of IgG antibodies to Helicobacter Pylori on the basis of the ELISA method.*

Results: *Our outcomes reported that the presence of seropositivity of H. pylori was shown in eighty-eight percent of the patients with the incidence of HE; whereas, among patients, without HE and healthy controls it was respectively 86% and 66%.*

Conclusion: *In the light of this research outcomes the element of seropositivity of H. Pylori seropositivity rate in cirrhotic patients having HE or without the incidence of HE was reported high in the healthy control group participants; whereas, the seropositivity of the H. Pylori was non-significant in the cirrhotic patients with the incidence of HE and also without HE incidence.*

Keywords: *Hyperammonemia, Ammonia, Peptic Ulcer Disease, Bismuth, Palmar Erythema.*

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INTRODUCTION:

There is a great attention diverted to the development of any possible association between H. Pylori infection just not with the various extra gastrointestinal disease but also with the upper gastrointestinal tract diseases such as liver diseases, biliary diseases, chronic cardiovascular diseases and colorectal cancers [1]. The incidence of HE is severely serious and repeated complication in the liver disease patients [2].

There is a scarcity of the availability of awareness and knowledge on the incidence of (H. Pylori) Helicobacter pylori and its contribution to biliary tract disease incidence and liver pathology in human. There are implications of the H. Pylori infection for the encephalopathy development in the patients; which is possible because of the enhanced ammonia production by the bacterial urease action on urea reported in the gastric lumen. There is no clarification or validated information or proof about the involvement of the H. Pylori as a reason for the development of hyperammonemia in liver cirrhosis patients. Back in 1993, there are evidence about the H. Pylori implicating potent factor of HE in the patients [3].

Conflicting reports, opinions and demonstrations are also available in the outcomes of clinical assessments. Various advantages are also forwarded by numerous authors about their influence on the eradicative management on HE management; therefore, no other evidence is available which is supported or validated by other authors [1]. We aimed at the assessment of the possible correlation between hepatic encephalopathy and seropositivity of the H. Pylori.

METHODOLOGY:

This research was held on 50 cirrhotic patients having HE (Hepatic Encephalopathy) (50), cirrhotic patients who do not have HE incidence (50) and control group (50) that included healthy patients. The serological assessment was carried out in all the patients in order to determine the classification of IgG antibodies to Helicobacter Pylori on the basis of the ELISA method. This research was carried out at the Gastroenterology Department of Nishter Hospital Multan from 2016 to 2017.

We did not exclude any patient with the previous history of H. Pylori, eradicative management of H. Pylori, acid-suppressive drugs intake, proton pump inhibitors, H^2 receptor blocker, bismuth compounds intake and antibiotics intake in the last six months timeframe. We also avoided patients with vagotomy history or upper gastrointestinal tract operated cases, upper endoscopy (PUD, Peptic Ulcer Disease) and various other causes of the coma. Age and gender were matched in all the three groups for onward comparison and analysis.

Every research participant underwent an assessment of PUD and upper endoscopy which was reported negative. Patients also underwent an assessment of the rapid urea to identify the infection of H. Pylori. No exclusion criteria were implemented in the control group as all the patients in this group were controls and volunteered themselves for the research.

Liver biopsy confirmed the incidence of the cirrhosis in the cirrhotic patients or on the other hand clinical features also helped in the diagnosis of cirrhosis such as oesophagal varices, ascites and an abnormality in the PT (Prothrombin Time). We determined the encephalopathy presence through clinical features which included various mental status parameters such as orientation, worry, mood and alertness. We also considered all the complications such as disturbance of the sleep pattern, naps during day-time and insomnia with reversal of day and night. We also performed various other clinical assessments such as jaundice, fever, anaemia, oedema, spider naive, abdominal collateral veins, palmar erythema, hepatomegaly, ascites, gastrointestinal haemorrhage, splenomegaly and better reaction to lactulose. We did not include any patient in a coma because of various reasons such as diabetes, cerebrovascular accident and uremic. An assessment of serological survey confirmed the classification of IgG about anti-H. pylori antibodies on the basis of the ELISA method; whereas, urea test confirmed the seropositivity with specificity and sensitivity above 98%.

We secured ethical approval and patient's consent before the commencement of the research and every protocol of the research was communicated with the research participants. Statistical analysis was carried out on SPSS software.

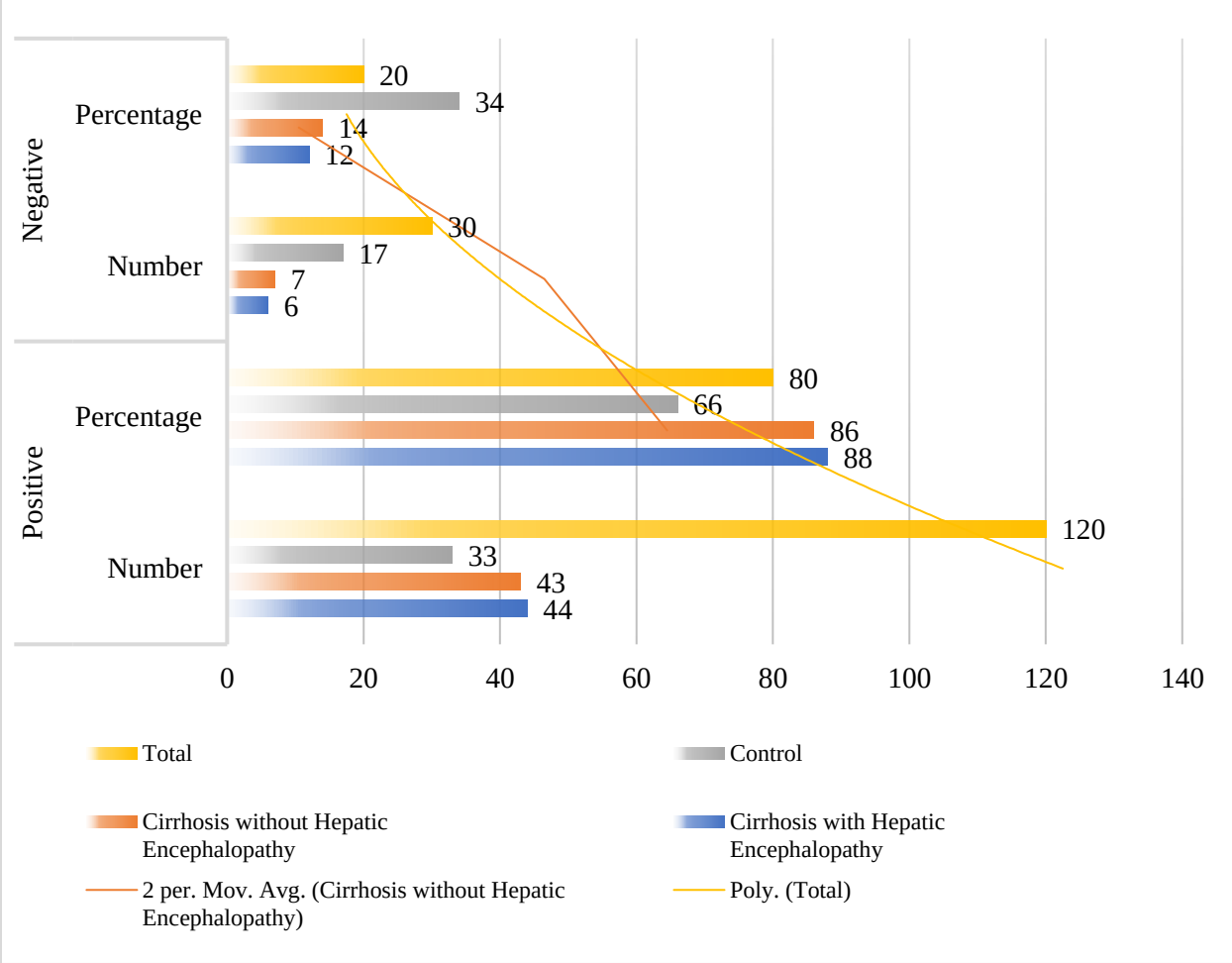
RESULTS:

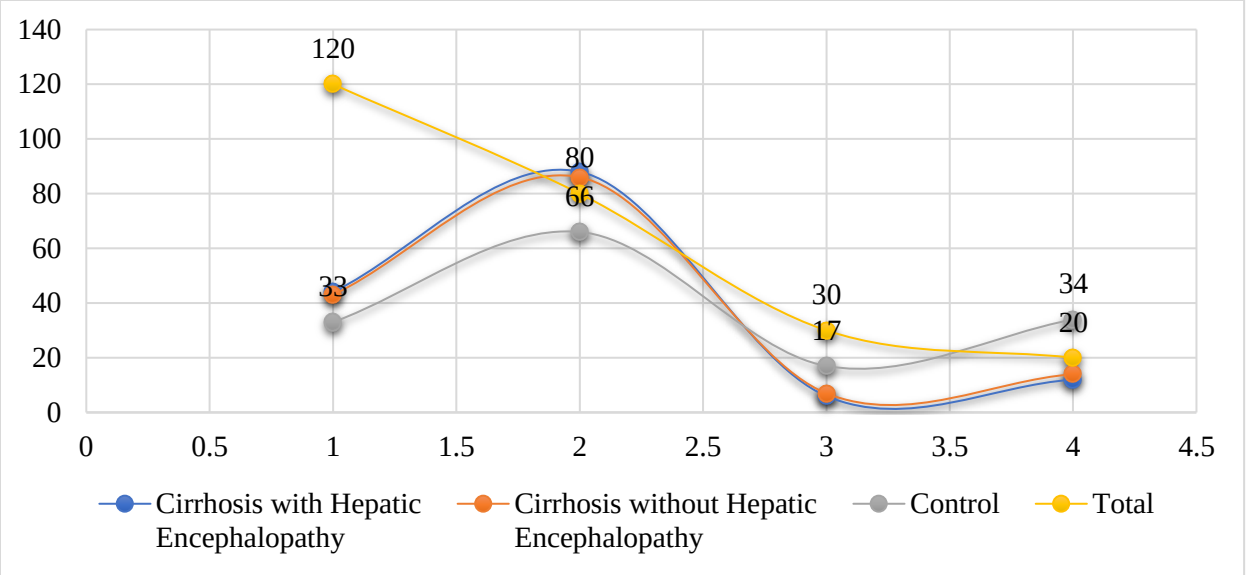
Our outcomes reported that the presence of seropositivity of H. pylori was shown in eighty-eight percent of the patients with the incidence of HE; whereas, among patients, without HE and healthy controls it was respectively 86% and 66%. We had a combination of both genders in the research population with females as 78.1% and males as 81.8%. All these patients were anti-H. pylori positive with a significant P-value of 0.56. Mean and SD values of age in cirrhotic with HE and without HE and control group was respectively (48.92 ± 16.95), (48.86 ± 18.45) and (45.96 ± 11.33) with a significant P-value of 0.56.

Mean and SD values of age among seronegative and seropositive patients was respectively (48.33 ± 14.49) and (47.81 ± 16.20) with a significant P-value of 0.44). Detailed outcomes analysis has been carried out in the given tabular and graphical data.

Table: Association between with and without hepatic encephalopathy Helicobacter pylori infection

Group	Positive		Negative	
	Number	**Percentage**	**Number**	**Percentage**
Cirrhosis with Hepatic Encephalopathy	44	88	6	12
Cirrhosis without Hepatic Encephalopathy	43	86	7	14
Control	33	66	17	34
Total	**120**	**80**	**30**	**20**





OR values for the anti-H. pylori IgG presence in the in cirrhotic patients with HE in comparison to the healthy controls was reported as 3.78 with Confidence Interval of (95%) in the range of (1.22 – 12.16) with a significant P-value of 0.01. The seropositivity of the H. pylori was more in the HE patients.

DISCUSSION:

H. pylori infection contribution in the incidence of an upper gastrointestinal tract in HE is due to the factor of its capability of synthesizing of ammonia as this pathogen is a higher expressive of an active urease in peri-plasma and on the surface.

Suto reported the H. pylori infection pathogenic role in the incidence of HE; moreover, he and his colleagues also demonstrated an increase in peripheral and portal levels of ammonia in cirrhotic H. pylori-infected gerbils [4].

In the light of research outcomes, the seropositivity of the H. pylori in cirrhotic patients having HE or without HE was high in comparison to the healthy control group participants. We did not give any preference to the H. pylori genomic structure which is also evident in the previous research studies. The PUD absence was an important element of the inclusion criteria as PUD and H. Pylori association may have influenced the overall outcomes of the research study. Shirmali et al. reported about the seropositivity of H. pylori that it increased as the HE severity increases [2]. Their outcomes are not comparable with the outcomes of our research study. We reported higher HE grades in the seropositivity of H. pylori than the outcomes as reported by Gubbins and his colleagues. According to their research outcomes seropositivity of G – I, II, III and IV was respectively 77.63%, 78.13%, 100% and 75% [5]. The probable reason behind the incidence was the small research sample that was made a part of the research subgroups. Shavakhi also compared anti-H. pylori antibodies seroprevalence in the cirrhotic cases control group as reported in their outcomes the presence of IgG antibody to H. pylori in cirrhotic patients 73% and control group 52% with a significant P-value under 0.003. A relative IgG antibody frequency to H. pylori was also reported higher in the cirrhotic patients in comparison to the control group patients [6].

Sethar also studies the same hypothesis on a total of 76 portosystemic encephalopathy patients because of the liver disease; where he reported a higher frequency of the H. pylori antibodies in portosystemic encephalopathy patients [3]. According to Wang, the prevalence of H. pylori was different among the cirrhotic patients having HE which was about 74.4%; whereas, in the subclinical HE and without HE it was respectively 69.1% and 53.3%. According to Wang, the factor of H. pylori infection is very important for a higher concentration of blood ammonia and HE in the cirrhotic patients [7]. These research studies also included the PUD cases. Previously, numerous series have not investigated the H. Pylori infection association in the light of this subject [8]. The concentrations of NH^3 and pH were also calculated in gastric juice which was collected through endoscopy. Rapid Urea Test was employed to diagnose H. pylori infection in the patients. Same prevalence of H. pylori was reported in the controls and liver cirrhotic patients; there was

also no association between the levels of NH^3 and gastric levels [8].

Chakrabarti also reported no significant variation in the levels of ammonia and gastric juice in his sub-clinical assessment [9]. Zullo also failed to describe any association between levels of plasma ammonia, H. pylori and psychometric testing in the patients of cirrhosis with the mild or latent incidence of HE [10]. Various other studies failed to establish any solid evidence of the H. pylori infection on levels of the fasting ammonia or any other associated parameters that ate utilized for the HE assessment [11]. The fact about the overt disease development in the patients is not because of the bacterial strain combination, various contributing environmental factors and disease host susceptibility [12]. There is a relevancy of the H. pylori strain cag status with various clinical features and outcomes. The cag is referred to a highly immunogenic protein [13].

CONCLUSION:

In the light of this research outcomes the element of seropositivity of H. Pylori seropositivity rate in cirrhotic patients having HE or without the incidence of HE was reported high in the healthy control group participants; whereas, the seropositivity of the H. Pylori was non-significant in the cirrhotic patients with the incidence of HE and also without HE incidence.

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