RELATIONSHIP BETWEEN SERO-POSITIVITY OF H. PYLORI AND HEPATIC ENCEPHALOPATHY

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Abstract:
Objective: There is very less knowledge on the H. Pylori (Helicobacter Pylori) involvement in the pathology of various diseases of liver and biliary tract in human beings. This research work aimed to evaluate the possible relationship between the sero-positivity of helicobacter Pylori and hepatic encephalopathy.
Methodology: This transverse research work carried out on three groups, cirrhotic with HE, cirrhotic without HE and group of healthy controls. The examination of all the patients carried out serologically to determine IgG class antibodies to helicobacter pylori depending upon the ELISA method.
Results: There was presence of sero-positivity of H. pylori in 88.0% cirrhotic patients with HE, 86.0% cirrhotic with no HE and 66.0% of controls.
Conclusion: In accordance with the findings of this research work, the rate of positivity of H. pylori in the cirrhotic patients present with HE or without HE was much high as compared to their healthy controls. But the rate of positivity of H. pylori was not much different in cirrhotic patients present with HE or without HE.
Key Words: Positivity, antibodies, hepatic encephalopathy, ELISA, transverse, serologically, biliary tract, infection, pathology.

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INTRODUCTION:

In current decade, there are more attention towards the probable relationship of infection of H. pylori with the diseases of gastro-intestinal tract but also with many other diseases of gastro-intestinal tract like colorectal cancers, liver diseases and biliary diseases [1]. HE is very common complication influencing the patients present with diseases of liver [2]. There is much fragmented knowledge on the contribution of H. pylori in pathology of diseases of biliary tract and liver. There is association of helicobacter pylori in the development of HE. This is probably because of the enhanced creation of ammonia by the action of bacterial urease on the urea in gastric lumen.

There is not fully clarification of the role of helicobacter pylori as a reason of hyper-ammonaemia in the patients suffering from liver cirrhosis. The very first research work showing the H. pylori as a factor of risk for HE was published in the year of 1993 [3]. There is demonstration of the conflicting opinions by the clinical observations. Some specialists have emphasized on the beneficial impact of the eradication treatment on course of HE, many authors were not in favor of this opinion [1]. The rationale of this research work was to interrogate the relationship between positivity of H. pylori and HE.

METHODOLOGY:

This research work carried out in Gastroenterology Department of Mayo Hospital, Lahore from March 2018 to December 2019. There were three groups in this research work as patients of cirrhosis with HE, patients of cirrhosis without HE and healthy controls with any clinical complication. Total 50 patients with HE & liver cirrhosis, fifty patients of without HE and with liver cirrhosis and fifty healthy controls were the recruited members of this research work. All the patients present with past history of H. pylori eradication treatment, using acid suppressive medicines, proton pump inhibitor, patients with past history of surgery of gastro-intestinal tract or suffering from disease of peptic ulcer were not the participants of this research work.

All the patients of Group-1 and Group-2 had to undergo upper endoscopy and assessment was negative for PUD and rapid urea test were also carried out for helicobacter pylori. Healthy controls were the willing persons from OPD of the department and they were present without liver cirrhosis. In the patients of cirrhosis, the confirmation of cirrhosis carried out by liver biopsy. The determination of the availability of the encephalopathy carried out by the clinical evaluations including psychological status and complaints of the disturbance in the patterns of sleep.

Clinical assessment of all the patients carried out including temperature, anemia, fever, spider naevi, jaundice, edema, ascites, splenomegaly, hemorrhage in gastro-intestinal tract and developed response to the lactulose. We excluded the patients in coma because of some other reasons. The evaluation of all the patients carried out in serological survey to determine IgG class anti-helicobacter pylori antibodies on the basis of ELISA method. Rapid urease test was in use for the confirmation of sero-positive findings. Ethical committee of the institute gave the permission to conduct this research work. We took written consent from all the patients after explaining them the purpose of this research work. SPSS V. 20 was in use for the statistical analysis of the collected data. Fisher exact test was in use for the assessment of the association between HE and infection of H. pylori. After that the comparison of the groups of healthy controls and cases carried out with each other by the frequency of the infection of H. pylori. We presented all the results of all three groups in percentages.
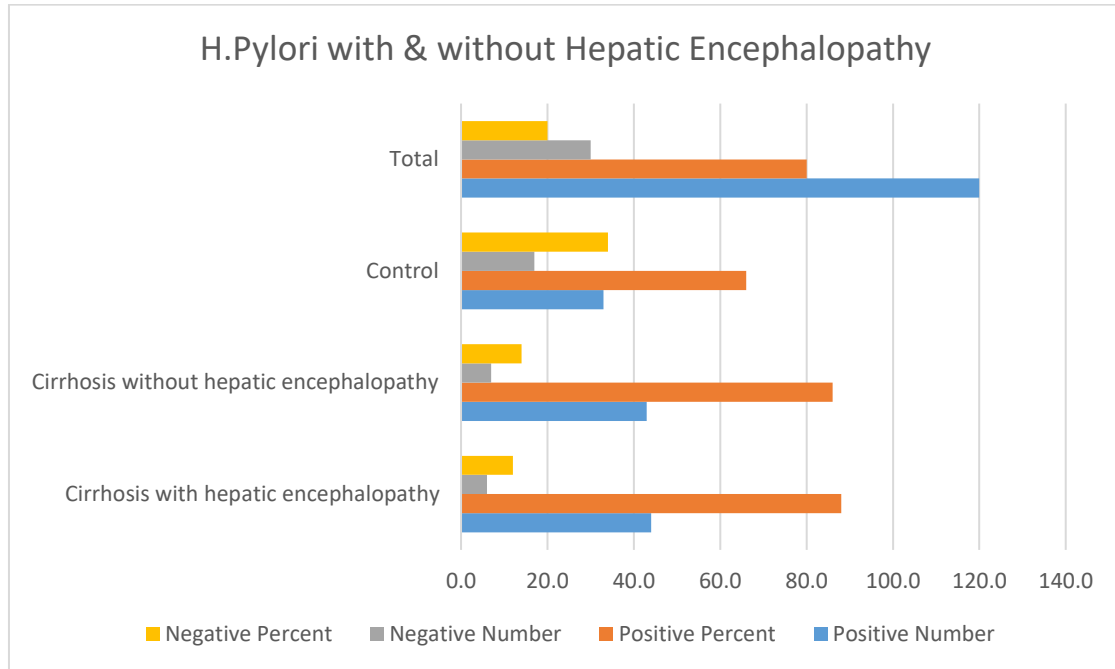
RESULTS:

There were total 81.80% males and 78.10% females positive for anti-helicobacter pylori (P= 0.560). The average age of the cirrhotic patients present with HE was 48.920 ± 16.950 years and among cirrhotic patients with no HE was 48.860 ± 18.450 years along with average age of 45.960 ± 11.330 for healthy controls (P = 0.560). The average age among sero-positive & sero-negative participants was 47.810 ± 16.20 years and 48.330 ± 14.490, correspondingly (P = 0.440). The antibody of H. pylori was positive in forty four out of fifty (88.0%) patients suffering from liver disease with HE, in forty three out of fifty (86.0%) patients suffering from liver disease with no HE and in thirty three out of fifty (66.0%) controls (P = 0.010). Most frequent etiology of the cirrhosis was HBV infection (43.0%) with sero-positivity of H. pylori as 88.370%. Among fifty patients of cirrhosis present with HE, there were total forty four patients positive for anti-helicobacter pylori and among fifty patients of cirrhosis with no HE, forty three patients were present as positive for anti-helicobacter pylori (P= 0.7660) [OR= 1.190 (95.0% CI: 0.330-4.430)].

The OR (Odds Ratio) for the availability of the anti-helicobacter pylori IgG among cirrhosis patients with no HE as compared to their controls was 3.160. (95.0% CI: 1.070-9.620) (P= 0.0190) (Table-1). OR for the availability of the anti-helicobacter pylori IgG in patients of cirrhosis with HE as compared to their healthy controls was 3.780. (95.0% CI: 1.220-12.160) (P = 0.010). The rate of sero-positivity of H. pylori was greater in high grades of HE.

Table-I: Association between H. pylori infection in cirrhotic with and without hepatic encephalopathy

Group	Positive		Negative	
	Number	Percent	Number	Percent
Cirrhosis with hepatic encephalopathy	44.0	88.00	6.0	12.00
Cirrhosis without hepatic encephalopathy	43.0	86.00	7.0	14.00
Control	33.0	66.00	17.0	34.00
Total	120.0	80.00	30.0	20.00

DISCUSSION:

Suto also reported the authentic pathogenic role of infection of helicobacter pylori in HE [4]. In this current research work, sero-positivity of H. pylori in higher grades of HE was greater, which is correlated with the results of research work conducted by Gubbins. In his research work, he discovered the sero-positivity of H. pylori in HE- G-1 (77.630%), G-2 (78.130%), G-3 (100%) and G-4 (75%) [5]. Shavakhi compared the sero-prevalence of anti-helicobacter pylori antibodies in the patients of liver cirrhosis with their controls in Iran. According to the results of that research work, IgG antibody to helicobacter pylori was available in 73.0% patients of cirrhosis and 52.0% in the group of healthy controls (P < 0.0030). He also discovered that relative rate of occurrence of IgG antibody to helicobacter pylori was much high in the patients of cirrhosis in as compared to their controls [6].

Wang stated in his research work that infection of H. pylori was a significant factor for inducing the high concentration of blood ammonia and HE in the patients suffering from cirrhosis [7]. There are some previous research work which did not examine the relationship between sero-positivity of helicobacter pylori and HE. Nam also evaluated his specimens

[8]. The concentration of NH3 and pH were calculated in gastric juice achieved by application of endoscopy. The diagnosis of the infection of helicobacter pylori carried out with the utilization of the rapid urease test. The rate of prevalence of H. pylori in the patients suffering from liver cirrhosis was same to their healthy controls and he found no association levels of gastric and blood NH3 [8]. Chakraborty assessed the association among infection of H. pylori gastric juice concentration of ammonia and levels of arterial ammonia in the patients suffering from liver cirrhosis, overt HE, sub-clinical HE and patients without HE and discovered no difference [9].

In his research, Zullo failed to determine an association between levels of ammonia in plasma, helicobacter pylori and scores of psychometric testing among patients of cirrhosis with mild or latent HE [10]. Research works have not concluded important impact of infection of helicobacter pylori on the levels of fasting ammonia or other parameters normally used for evaluation of HE [11]. There is development of overt disease in some patients while some patients so not have it, this is because of the combination of differences of bacterial stain, various factors of environment and host susceptibility to

disease [12]. Cag status strain of H. pylori is pertinent to the risk of amount of clinical outcomes. The protein of Cag-A is much immunogenic [13].

CONCLUSION:

Understanding of the infection of H. pylori role in the patients of cirrhosis and its related complications, for instance HE permits further research works. Until the achievement of further data, it would be worthwhile to follow the similar strategies for the eradication of the H. pylori in cirrhotic as well as non-cirrhotic patients. The findings of this research work suggested that there should be assessment of the genomic structure of the H. pylori as well as the impact of strains of this organism on HE.

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