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HELICOBACTER PYLORI WORSENING FACTOR OF THE PATIENT'S CONDITION IN PATIENTS WITH LIVER ENCEPHALOPATHY

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Annotation
Hepatic encephalopathy is one of the neuropsychiatric reversible syndromes that manifests itself as impairment of brain function. H. pylori plays a vital role in providing a factor in the pathogenesis of peptic ulcer disease, and there is currently debate about its role in hepatic encephalopathy. Statistics from previous studies have shown that in the gastric secretions and at the same time in the blood serum, some association was found associated with high ammonia content. H. pylori is a gram-negative bacterium that mainly live and infect the acid-resistant mucous membrane of the human stomach. environment, and are the main cause of peptic ulcer and gastritis. They are rich in the enzyme urease, which increases the production of ammonia from the lumen of the stomach, passes into the systemic circulation and enters the brain, which is the main cause of hepatic encephalopathy. High blood ammonia levels are also found in patients with hepatic
encephalopathy, which may play a role in its pathogenesis

Key words: liver cirrhosis, H. pylori, seropositive infection, hepatic encephalopathy.

INTRODUCTION

Hepatic encephalopathy is one of the neuropsychiatric reversible syndromes that manifests itself as impairment of brain function. As soon as liver cells do not excrete metabolic products, especially ammonia, which then accumulates in the blood and causes clinical manifestations of hepatic encephalopathy. Hepatic encephalopathy is clinically represented by various disorders that affect the patient's attention and memory, cognitive abilities, functional abilities, personality changes and intellectual abilities [1]. Hepatic encephalopathy usually develops due to cirrhosis of the liver. Patients may experience certain signs and symptoms such as confusion, forgetfulness, sweet bad breath, and fatigue. Patients with hepatic encephalopathy can also go into

coma, reflecting its severity and poor prognosis. H. pylori is a gram-negative bacterium that mainly live and infect the acid-resistant mucous membrane of the human stomach and are the main cause of peptic ulcer disease and gastritis. They are rich in the enzyme urease, which increases the production of ammonia from the lumen of the stomach, pass into the systemic
blood circulation and enter the brain, which is the main cause of hepatic encephalopathy [2,10]. High blood ammonia levels are also found in patients with hepatic encephalopathy, which may play a role in etiopathogenesis [2]. Electroencephalographic (EEG) abnormalities in patients with hepatic encephalopathy that are observed in these patients are similar to those observed in uremia and acid - base disorders [3,5,9,10]. Hepatic encephalopathy has different types depending on the severity and their occurrence.
Hepatic encephalopathy is classified into three subtypes, subtypes A, B, and C [4,5]. Subtype A is associated with acute liver injury and is fatal [6]. This subtype leads to swelling of astrocyte cells in the brain, which are responsible for the detoxification of ammonia, and once these cells lose their ability to function normally, this may be the main factor contributing to the pathogenesis of hepatic encephalopathy [1,5]. Type B hepatic encephalopathy is quite rare in type B hepatic encephalopathy or hepatic bypass encephalopathy, in which an abnormal connection is formed and toxins enter the systemic circulation without detoxification [4,9]. Type C as one of the common encephalopathies associated with liver cirrhosis. Type C is further subdivided into episodic, persistent, and minimal encephalopathies [5,9,13].

Objective

To determine the prevalence of seropositive Helicobacter pylori infection in patients with liver cirrhosis with hepatic encephalopathy.

Materials and Methods of Research

The study was carried out in the 2nd Therapy department of the Clinic of the Samarkand Medical Institute No. 1, from September 2019 to June 2021, on 98 patients after the permission of the hospital's ethical committee. We collected questionnaire data on patients with cirrhosis of the liver with encephalopathy admitted to the therapeutic department. Patients were informed and their written consent was obtained (aged 21 years and older) with a full comprehensive clinical history and physical examination. 5 ml of blood was taken from each patient, which were obtained under aseptic conditions and sent to the Samarkand Medical Institute laboratory No. 1 to detect antibodies to H. pylori.

All patients (male and female) aged 21 years and older are included in the current study. The average age of the patients was 36.32 years ± 10.48 years, the minimum age was 21 years, and the maximum age was 65 years. Of these, 48 (48.97%) were women, 50 (51.03%) were men, and a positive result for H. pylori was found in 79 (80.61%) patients. The distribution of patients by the degree of encephalopathy shows that 18 (18.36%) patients have grade 1, 23 (23.46%) - grade 2, 40 (40.81%) - grade 3 and 17 (17.34%) have encephalopathy of 4 degrees. All patients were with liver cirrhosis, with clinical signs of grade 1 to 4 encephalopathy (West Haven criterion). All patients underwent a full check of a detailed clinical examination. All studies were carried out in the same laboratory. The complete material was documented in accordance with the design of this study. H. pylori seropositivity was stratified by age, sex, and encephalopathy grade to see effect modifiers. Percentage rates were calculated for all categorical variables such as gender, age, encephalopathy score, and serum positive H. pylori and mean (±). Standard deviation was calculated for continuous variables such as age. All data were presented in tables and graphs.

Research Results

H. pylori plays a vital role in providing a factor in the pathogenesis of peptic ulcer disease, and there is currently debate about its role in hepatic encephalopathy. Statistical data from previous studies showed that in gastric secretions and at the same time in blood serum, there was some association associated with high ammonia levels, and a significant increase in ammonia levels was also observed. The main aim of the study was to search for the effect of H. pylori in patients with hepatic encephalopathy. Numerous additional abdominal disorders are associated with Helicobacter pylori infection. The association of hepatic encephalopathy with H. pylori infection is possibly related to its virulence factors; of these, ammonia produced by this H. pylori pathogen in the stomach is one of the factors leading to its pathogenicity. When comparing patients with cirrhosis of the liver with hepatic encephalopathy, rather than without it, H. pylori, which breaks down urea into ammonia and increases the pH in the stomach thereby causing peptic ulcer disease, it can also lead to the accumulation of ammonia in the blood and may contribute to the development of hepatic encephalopathy. And also the level of ammonia in the blood improves in patients with hepatic encephalopathy after specific treatment with H. pylori [8]. In our study, the severity of hepatic encephalopathy showed a concomitant increase in H. pylori titer, which was taken into account. This factor may be responsible for the deterioration of the condition of patients with liver cirrhosis [9-14].

Conclusions

In patients with grade 3 and 4 encephalopathy caused by cirrhosis, antibodies to Helicobacter pylori showed a high titer, this may indicate that the presence of Helicobacter pylori may play a role or may enhance the pathogenesis of hepatic encephalopathy in patients with cirrhosis liver. A total of 98 patients were diagnosed with cirrhosis in hospitals based on clinical assessment, liver laboratory parameters, and ultrasound measurements. A study by our group showed that patients with liver cirrhosis and those who developed hepatic encephalopathy due to higher ammonia levels had an increased rate of H. pylori seropositivity. The highest H. pylori titer was demonstrated in patients with severe hepatic encephalopathy, clearly suggesting that H. pylori may play a very significant role in the development of hepatic encephalopathy. However, further research should be carried out at a higher level, involving a large number of patients for better understanding.

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