

The Effect of Helicobacter Pylori on Liver Function Using the New Urea Breath Test Technique

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Abstract:- *Helicobacter pylori* (*H. pylori*) is a dangerous bacteria that is known to colonize the abdominal mucosa in humans. Infection with *H. pylori* is enormously conventional worldwide; the micro organism influences up to 90% of grownup populations in creating countries. Besides gastric problems, several latest investigations have confirmed the correlation between *H. pylori* infection and different illnesses like hematologic, ophthalmologic, dermatologic, neurologic, and hepatobiliary. According to some indicators, the liver is one of the organs may additionally also be affected with the aid of *H. pylori*; however, it is but unknown how exactly the contamination impacts the liver and the underlying mechanisms are unclear. The effects was once fantastically enormous liver features in serum of patient groups (P) in contrast to control group (C) ($p < 0.001$). Meanwhile, there had been no giant differences located between male and girl in all studies groups. And Urea Breath Test in Breath in all groups is presented. Patients' groups shows highly significant increase when compared to control group ($p < 0.001$).

Keywords:- Liver Function; *Helicobacter pylori*; GOT, GPT, TSB.

I. INTRODUCTION

Helicobacter pylori or (*H. pylori*) are gram-negative bacteria, bacillus, microaerophilic considerable [1]. This chronic bacterial infection is one of the most prevalent and ubiquitous in the globe, affecting over 50% of the population. [1-3]. In 1983, both Drs. Barry Marshall and Robin Warren isolate *Helicobacter pylori* for the first time [4]. Between 19 and 88% of patients have *H. pylori*, depending on a vary of elements such as geographical region, age of the patient, sanitation, and socioeconomic fame [1, 3, 5]. Patients are often infected with *H. pylori* in childhood and, if left untreated, persist at some point in life [1, 5, 6].

Both invasive and non-invasive *H. pylori* assessments are available [7]. and every technique has its benefits and disadvantages [2]. There are specificities and sensitivities, clinical settings, test costs, availability, and other factors that influence how desirable a test or set of tests is [2, 7, 8]. The gold popular check to affirm the presence of *H. pylori* is esophagogastroduodenoscopy (EGD) blended with a biopsy's histological analysis [9]. The general non-invasive techniques consist of serological testing, the *H. pylori* stool antigen test (SAT), and the urea breath check (UBT) [6].

UBT is beneficial for each the preliminary prognosis of *H. pylori* (testing and remedy strategy) and in the assessment of the condition after treatment [10]. Since urease is absent from mammalian cells, its presence suggests urease containing microbes. Urea is damaged down by way of *H. pylori* the use of urease to produce ammonia and carbon dioxide [11].

Urease is utilized with the aid of *H. pylori* to split urea into carbone dioxide and ammonia. In the neutralization of gastric acidity *H. pylori* is boost ammonia and assist it in migration [8]. Bacteria additional than *H. pylori* are commonly no longer located in the belly with little exceptions for example in sufferers with achlorhydria [12, 13]. UBT quickly hydrolyzes urea into ammonia and carbon dioxide (CO_2) by using the urease capacity that is widely present in *H. pylori*. [14]. After entering the bloodstream, the carbon dioxide is exhaled via the lungs. Carbon isotope (C) is employed as a substrate for urea, and exhaled carbon dioxide labeled with C can be used to test and diagnose *H. pylori*. For this purpose, two carbon isotopes are typically used: experiment ^{13}C (stable radioactive isotope) and experiment ^{14}C (radioactive isotope) (not naturally radioactive). All UBTs exhibit a similar appearance, with specificities reaching 93% and sensitivities reaching 95%. [10, 15, 16].

It is commonly known that *Helicobacter pylori* infection contributes significantly to extragastric damage and has the potential to cause metabolic and cardiovascular disorders.. Furthermore, documentation suggests that *H. pylori* is also associated with liver illness, playing a significant role in the emergence of insulin resistance, non-alcoholic fatty acidosis, cirrhosis, non-alcoholic steatohepatitis, and liver disease. [17]. Additionally, with *H. pylori* infection, we can expect a reduction in hepatitis along with autoimmune manifestations involving the bile duct and liver. [18]. Le Roux-Goglin et al. previously showed in rat hepatocyte subcellular models that the promotion of podosome formation and collagen growth in hepatocytes is one possible pathway that could lead to liver fibrosis and possibly cancer in vivo. [19]. According to a biometa-analysis, the prevalence is higher in emerging nations like Asia and Latin America than it is in industrialized nations like the United States and Europe. [19].

Different types of biochemical, secretory and structural features are generated by the liver, so no biochemical test can detect global characteristics of the liver. All laboratories generally use a combination of tests to detect and correlate significant liver disease, and these evaluations are often

called “liver function tests,” while they cost little to evaluate liver characteristics per second. Although this term has received much criticism, the phrase “tests of liver properties” is firmly entrenched in the clinical lexicon. It may be a mistake that “tests for liver damage” is a more correct term. Furthermore, physical examination and clinical history play a vital role in determining function. Liver biopsy, signs, radiological imaging and the role of a particular disorder cannot be underestimated [20, 21].

➤ *Chemicals*

All chemical compounds used in this find out about have been of exceedingly purified grads.

➤ *Subjects*

The study was conducted on 50 individuals divided into two groups. Group 1 consists of 30 patients with Helicobacter Pylori (H. Pylori) (30 ± 13.3 years) . with Group 2 consists of 20 healthy persons as a control group with age (31± 9.79 years) . The patients’ samples were collected from Baghdad Digestive Teaching Hospital / Medical City.

➤ *Blood Samples*

The venous blood samples about five ml from patients and manage organizations used to be drawn and accrued in the plane tube then left for 5 – 10 minutes at room temperature. After that, the blood samples had been

centrifuged at 4000 rpm for 5 – 10 minutes and accrued the sera. The got sera were aliquot and saved at -20 ° C until use.

➤ *Determination of Liver Function Test :*

Aspartate Aminotransferase (AST, or SGOT) ,Alanine Aminotransferase (ALT, or SGPT) and Bilirubin Test was determined by colorimetric method for the quantitative in vitro diagnostic measurements on Wavelength 340 nm[22] .

➤ *Determination of Urea Breath Test :*

Urease in the stomach can be detected with the urea breath test (UBT). Urea is converted to ammonia and carbon dioxide by the urease that H. pylori produces in the abdomen after consumption of carbon-labeled urea (13C or 14C). The carbon isotope can then be detected right away after being inhaled as tagged carbon dioxide. The recent use of antibiotics, bismuth, and acid-suppressing medications has decreased the sensitivity of the breath test to urea by inhibiting H. pylori's capacity to metabolize urea. UBT has a sensitivity of about 90% and a specificity of about 96%. [23].

II. RESULTS

The mean ± SD ages of the groups (P & C) who enrolled in this study were 29.25± 9.69 and 30.06± 7.02. As shown in Table 1, there were no significant differences in Age in all studied groups (p> 0.05).

Table 1: The Age, Gender of P and C Groups.

| Groups | n | Age (years) | Gender | | p-value |
|--------|----|-------------|--------------|---------------|---------|
| | | | Male No. (%) | Female No.(%) | P&C |
| C | 20 | 29.25± 9.69 | 10 (50%) | 10 (50%) | > 0.05* |
| P | 30 | 30.06± 7.02 | 15 (50%) | 15 (50%) | > 0.05* |

The results of Liver functions(GOT,GPT &T.S.B) shown in Table 2. highly significant Liver functions in serum of P groups compared to C group (p<0.001), Meanwhile, no significant differences observed between male and female in all studies groups.

Table 2: Mean Values± SD of Parameters in the Patients and Control Groups

| Parameters | Groups | | p-value |
|-------------|--------------|-------------|---------|
| | P n = 30 | C n = 20 | P&C |
| GOT U/I | 105.46±18.00 | 16.75±4.529 | 0.000↑* |
| GPT U/I | 110.96±15.12 | 18.4±4.956 | 0.000↑* |
| T.S.B mg/dl | 3.27±0.761 | 0.65±0.654 | 0.000↑* |

*The difference is significant at p<0.05 and highly significant at p<0.001.

↑ Significant increase, ↓ Significant decrease, ↔ Non-significant

Urea Breath Test in Breath in all groups is presented in Figure (1). A highly significant increase is found in patients groups compared to control group (p<0.001).

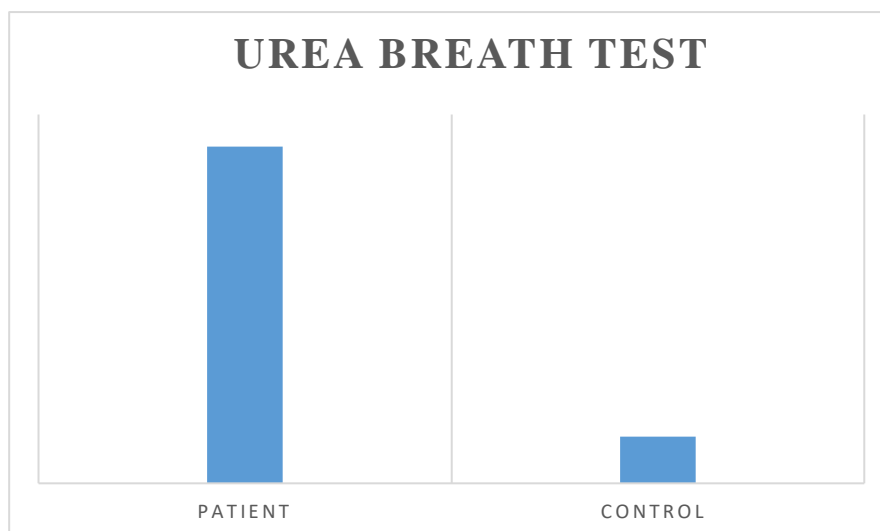


Fig 1: Mean values \pm SD of Urea Breath Test in Patients and Control Groups.

III. DISCUSSION

Helicobacter species appear to be implicated in the pathogenesis of liver cancer and persistent liver sickness, as evidenced by the recovery of these bacteria from liver samples belonging to a range of species.[24, 25]. Nevertheless, it is unknown if *H. pylori* directly causes liver diseases. Once upon a time, a human gallbladder patient's excised gallbladder mucosa contained an *H. pylori*-like microbe. [26]. The authors of multiple investigations have reported identifying *H. pylori* in liver samples from individuals with essential sclerosing cholangitis and, more significantly, biliary cirrhosis using molecular biology techniques. [27, 28].

A correlation between fatty liver and *H. pylori*-positive individuals was previously shown in a study involving 174 patients using the helpful resource of Dogan et al. Additionally, an increase in the liver and spleen using ultrasonography was also reported. [29]. Some investigators have also reported that patients with cirrhosis had a significantly higher incidence of anti-*H. pylori* antibodies compared to the control group. [30]. It was unclear from these data if *H. pylori* DNA arose from the retrograde transfer of DNA from the duodenum to the liver or if these findings genuinely reflect liver colonization, even though *Helicobacter* spp. DNA was detected in liver samples of individuals with basic liver carcinoma. Liver samples' capacity to identify *H. pylori* on culture media was proof positive for a true bacterial invasion. [28, 31].

Furthermore, Akbas et al. Patients with *H. pylori* infection exhibited much higher AST and ALT levels than those without infection. Other researchers confirmed that there was a significant difference in AST and ALT between companies that had *H. pylori* contamination and those that did not. This perspective is consistent with their findings.[32].

In 2014, a study examined the connection between elevated liver transaminases enzymes and *H. pylori* infection. 107 patients with dyspepsia, an *H. pylori* infection, and unexplained high liver enzymes were included in the study. The *H. pylori* infection was successfully treated in 93 out of 107 patients, and the serum ALT and AST levels were returned to normal, with a p-value of 0.001 and a difference of roughly 46.6% and 45.7%, respectively. This was in line with our final finding, which connected higher liver transaminases to *H. pylori* infection.[33].

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