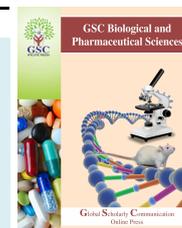


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## Association of *Helicobacter pylori* infection and vitamin B12 deficiency

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### Abstract

*H. pylori* infection is strongly related with chronic gastritis of the stomach, which causes impairment in gastric acid and secretion of pepsin, and linked to male absorption of food-vitamin B12. The most important virulence genes accompanying stomach and intestine disease are (cag A and vac A). Aim of the study: To evaluate the rate of gastric ulcer infection with *H. pylori* and its detect CagA gene in *H. pylori* isolated from gastritis. Materials and Methods: A total of 200 biopsy patients aged from (10- ≥ 60) years were collected from Baqubah teaching hospitals. They were suffering from gastric upset and attended to endoscopic unit of department of medicine. Questioners including, sex, age, smoking, presence of cancer, and biopsies of gastric tissue were collected from the corpus or the ant rum or corpus and ant rum of the patient's stomach. Three biopsies were taken from each patient. Histopathologic study, gram staining, and rapid urease test working for each patients. Serology Test Serum Enzyme-Linked Immunosorbent Assay Testing (ELISA).

**Keywords:** Gastric ulcer; *H. pylori*; Cag A gene; Stomach ulcer; Duodenal ulcer

### 1. Introduction

*Helicobacter pylori* (*H. pylori*) affects nearly half of the world's population, thus is one of the most frequent and persistent bacterial infections (1). *H. pylori* is associated with peptic ulcer, gastric ulcers, mucosa-associated lymphoid tissue, and gastric cancer. It infects the stomach during childhood. In developing countries children are very commonly infected. *H. pylori* may be passed from person to person through direct contact with vomit, saliva, or fecal matter. Risk factors for *H. pylori* infection are related to: living in crowded conditions area, without a reliable supply of clean water, living in a developing country, or with someone who has a *H. pylori* infection. (2). The cases Vitamin B<sub>12</sub> deficiency is attributed to malnutrition. *H. pylori* infection plays an important role in the development of atrophic gastritis and related malabsorption. It is suggested that there may be a relationship between *H. pylori* infection and vitamin B<sub>12</sub> deficiency (3). A study of *H. pylori* IgG Antibodies in Iraqi Uremic patients is designed to evaluate the effect of blood urea and serum creatinine level, on gastric enzymes and *H. pylori* infection in patients with end stage renal failure before and after kidney transplantation, also to detect the association between the Human Leukocyte antigens (HLA) class I, II antigens with *H. pylori* infection (4). During the period October 2006 to July 2007 a study was done by WHO which involve Isolation and identification of *H. pylori* from drinking water in Basra governorate, Iraq. 198 samples of drinking water, from 22 districts of Basra governorate were collected. Only 14 isolates were *Helicobacter spp.*, of which 10 were *H. pylori*. These isolates, were tested for antibiotic susceptibility as well as ability to tolerate chlorine at 0.5 mg/L. They reported the occurrence of *H. pylori* in treated drinking water (5). Another study was done in Al- Sadar Teaching Hospital in Basrah in 2018 which involved 100 samples was taken from patients with peptic ulcer. The result revealed that (36%) *H. pylori* infected patients had normal vitamin B<sub>12</sub> level and (64%) were Vitamin B<sub>12</sub> deficiency in (86%) *H. pylori* infected patients (6). Aim of study: - study the relationship between *H. pylori* and vitamin B<sub>12</sub> deficiency. *H. pylori* is Gram-negative microaerophilic bacterial (7), appear spiral-shaped. The organism has 2 to 6 unipolar, sheathed flagella which often carry a distinctive bulb at the end. The flagella confer motility and allow rapid movement in viscous solutions such as the mucus layer toward the more neutral pH of the gastric mucosa. *H. pylori* are urease positive which thought to

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allow short-term survival in the highly acidic gastric lumen. The flagella confer motility and allow rapid movement in viscous solutions such as the mucus layer overlying the gastric epithelial cells (8, 9, 10). The helical shape of *H. pylori* is crucial for bacterial motility and a prerequisite for successful colonization (11). Motility has been shown to be essential for successful in vivo colonization by *H. pylori* and is provided by its sheathed flagella. In this respect, proposed that bacterial chemo sensing and motility promoted very rapid *H. pylori* colonization of injury sites, thereby contributing toward sustained gastric damage, by slowing down gastric repair (12). Chemotaxis allows a bacterium to control its swimming behavior in response to chemical signals.(13). Adherence A study was done in 2014 shows that the blood groups antigen-binding adhesion (BabA), an outer membrane protein occur commonly, driven by mutation and recombination, resulting in a complete loss of protein expression or in gradual changes in binding properties (14). Persistence A successful establishment of infection in such an acidic stomach is active intracellular urease, a major constituent of the total bacterial proteins output of *H. pylori*. This enzyme responsible for ammonium assimilation were involved in regulating either the hydrolysis of urea inside the cell of stomach, or the extent to which this ammonium was extruded versus assimilated, supporting the association between the assimilation of NH<sub>4</sub> and the primary acid resistance mechanism in *H. pylori* (15). *H. pylori* persistence has been demonstrated to be favored by the activity of (gGT), which has been demonstrated to impair T-cell proliferation (16). Cag A *H. pylori* strains are more virulent strain and are associated with gastric carcinoma, cagA, is injected by the bacterium into gastric epithelial cells and subsequently undergoes tyrosine phosphorylation. The phosphorylated cagA specifically binds SHP-2 phosphatase, activates the phosphatase activity, and induces morphological transformation of cells (17). The epidemiology of *H. pylori* has been changing over the last decades, with a decline of the prevalence of the infection in most countries. The changing epidemiology of the bacterium has been associated with decline in peptic ulcer disease and gastric cancer (18) and may have an impact on the changing epidemiology of other diseases, such as gastro esophageal reflux disease, and asthma (19, 20). The clinical features of *H. pylori* range from asymptomatic gastritis to gastrointestinal malignancy (21, 22). Vitamin B12 is an essential cofactor that is integral to methylation processes important in reactions related to DNA and cell metabolism, deficiency may lead to disruption of DNA and cell metabolism and thus have serious clinical consequences (23). Vitamin B12 cannot be synthesized in the body so must be obtained from the diet. The main dietary sources of B12 are dairy products, meat and eggs. The acidic environment of the stomach enables the release of B12 that is bound to food (24). The free B12 is rapidly bound by intrinsic factor (IF), a muco-polysaccharide secreted by gastric parietal cells that line the stomach. The binding of B12 to IF occurs in the duodenum causing the formation of the IF-B12 complex (25). This complex is resistant to digestion by stomach juices. Reaching the terminal ileum, it binds to and is absorbed by the intestinal microvilli (26, 27). These functions take place in fatty acid, amino acid and nucleic acid metabolic pathways. The deficiency of vitamin B12 is clinically manifested in the blood and nervous system where the cobalamin plays a key role in cell replication and in fatty acid metabolism (28). Vitamin B12 (Vit B12) deficiency is a common condition, which can be manifested with non-specific clinical features or with neurological or haematological abnormalities in severe cases. In systemic sclerosis (SSc) gastrointestinal involvement, nutritional status may lead to Vit B12 deficiency (29). Deficiency of vitamin B12 is cause of megaloblastic anemia. It is a reversible cause of bone marrow failure (30). Chronic gastritis owing to *H. pylori* infection (Hp-I) can lead to malabsorption of Vitamins B12 and folate, which results in failure of methylation by 5-methyl-tetrahydrofolic acid and accumulation of Hcy (30).

The studies showed that age group (40-49) and (50-59) was the most age group of people with bacteria *H. pylori* as the percentage (29.10%). From 200 biopsy samples.

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## 2. Conclusion

*H. pylori* infection is strongly related with chronic gastritis of the stomach. The presence of this genes related with chronic gastritis and stomach ulcer.

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## Compliance with ethical standards

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### *Disclosure of conflict of interest*

There is no conflict of interest with any governmental institutions.

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