We are IntechOpen, the world’s leading publisher of Open Access books
Built by scientists, for scientists

6,600
Open access books available

177,000
International authors and editors

195M
Downloads

154
Countries delivered to

TOP 1%
Our authors are among the most cited scientists

12.2%
Contributors from top 500 universities

WEB OF SCIENCE™
Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com
Chapter

Changes in Vitamin B\textsubscript{12}, Iron, Thyroid Hormones, Thyroid Autoantibodies and Hematological Indices Levels in Patients Suffering from *Helicobacter pylori* Infection

Saleh Nazmy Mwafy, Wesam Mohammad Afana and Asma’a Ali Hejaze

Abstract

*Helicobacter pylori* infection has been recognized as a public health problem worldwide with raising prevalence in developing than the developed countries. More than 50% of the world’s population infected, and 80% of infected have no symptoms. Megaloblastic anemia can occur due to impaired DNA synthesis resulting from deficiencies of vitamin B\textsubscript{12} and folate. The development of autoantibodies to thyroid peroxidase (anti-TPO), thyroglobulin (anti-Tg), and thyroid-stimulating hormone receptor (TSH-R) is the main characteristic of autoimmune thyroid disease. *H. pylori* may decrease absorption of oral thyroxine by decreasing gastric acid secretion in the stomach. *H. pylori* has important role of in the development of autoimmune thyroid diseases, vitamin B\textsubscript{12} deficiency and malfunctions of human. The primary goal of this chapter is to observe association between *H. pylori* infection in the gastric mucosa and of autoimmune thyroid diseases vitamin B\textsubscript{12} deficiency because eradication of *H. pylori* can prevent the development of complications.

**Keywords:** vitamin B\textsubscript{12}, iron, thyroid hormones, thyroid autoantibodies, *Helicobacter pylori*

1. Introduction

*Helicobacter pylori* (*H. pylori*) is a spiral, flagellated, gram-negative bacteria, adapted to survive in the gastric lumen [1]. *H. pylori* is a bacterium that causes widespread infection, affecting over 50% of the world’s population, but 80% of infected people are asymptomatic and more common in developing countries [2].

Megaloblastic anemia can result from impaired DNA synthesis resulting from vitamin B\textsubscript{12} (cobalamin) and folic acid deficiency. Microorganisms synthesize vitamin B\textsubscript{12}, that is primarily found in low concentration levels in meals with an animal source,
humans cannot synthesize vitamin B\textsubscript{12}. Early detection of vitamin B\textsubscript{12} deficiency and rapid treatment of is important, since it is a reversible cause of demyelinated nervous system and bone marrow failure [3]. infection with \textit{H. pylori} causes gastritis and it is associated with the progress of micronutrient deficiencies, peptic ulcer, gastric carcinoma [4].

Thyroid gland is one of the crucial organs in the human body that produces basic hormones: triiodothyronine (T3) and tetra-iodothyroxine (T4) which have an essential part in control of metabolic functions, development and growth. Thyroid dysfunction affecting various vital activities; those subsequent from hypo or hyper thyroid gland action leading to increase or decrease thyroid hormones T3 and T4 [5].

Hashimoto’s thyroiditis (HT) is the most widely autoimmune thyroid disorders as one of most complications of thyroid dysfunctions. Autoimmune diseases occur when the immune system begins to attack its own self-antigens, so, that the characteristic feature of autoimmune thyroid disease is the presence of autoantibodies against thyroid antigens. Such diseases are triggered by factors including infectious agents, just like as infection with \textit{H. pylori} [6, 7]. Luther et al. (2010) found that a high prevalence of people who have been diagnosed as thyroid patients were also infected with \textit{H. pylori} which means that these bacteria play a critical part in the pathogenesis of such illnesses. \textit{H. pylori} is one of the most well-known bacterial pathogens that infect human around the worldwide, which acquired in the early childhood and is carried throughout a lifetime if not treated with antimicrobial agents [8].

The present work sought to investigates the changes in vitamin B\textsubscript{12}, iron, thyroid hormones, thyroid autoantibodies and hematological indices levels in patients suffering from \textit{H. pylori} infection.

2. \textit{H. pylori}

2.1 General characteristics

\textit{H. pylori} is considered to be as one of the furthermost common pathogenic bacteria that colonizes human stomach, varying from 70\% in developing countries and less than 40\% in the developed countries [9]. \textit{H. pylori} is the main causative agent of gastritis and responsible for development of adenocarcinoma by stimulating cell proliferation and induces apoptosis [10]. \textit{H. pylori} is a Gram- negative spiral bacteria measuring 2–4 \upmu m in length, 0.5–1 \upmu m in width and has 2–6 sheathed flagella 3 \upmu m in length (Guo et al., 2011). \textit{H. pylori} growth at optimal rang of: temperature 34–40\degreeCelsius, pH 5.5–8.0 but can survive at pH 4 and the key feature of \textit{H. pylori} its microaerophlicity. Growth at optimal level of: 2–5\% oxygen, 5–10\% carbon dioxide and 85\% nitrogen [11].

\textit{H. pylori} is microaerophilic bacterium, requires lower oxygen concentrations than other bacteria to exist. It has a hydrogenase that may be utilized to generate energy by oxidizing the molecular hydrogen (H\textsubscript{2}) produced by intestinal bacteria, and it can generates urease, catalase, and oxidase [12]. These bacteria survive in the stomach for a long time without any indications in most of the infected people. In order to inhabit the stomach, \textit{H. pylori} must survive in acidic pH, its persistence be influenced by the production of urease enzyme, in addition to this enzyme these pathogenic bacteria produce other enzymes which damage of host epithelial cells such as catalase, protease and phospholipase. \textit{H. pylori} are vital for colonizing the stomach and make it possible for it to pass readily through the mucous layer [13].
2.2 *H. pylori* classification

*H. pylori* genus is a *Helicobacteraceae* family member and *Campylobacterales* order of proteobacteria. The digestive tracts of both humans and animals naturally contain the genus *Helicobacter*, which has more than 20 identified species and several more that are waiting proper classification [14].

2.3 Prevalence of *H. pylori* infection

*H. pylori* infection prevalence rates vary by age, place of origin, and socioeconomic position. Worldwide, *H. pylori* infection affects 50% of the population [15]. According to reports, up to 80% of people in undeveloped countries are infected with *H. pylori*. In Texas, the incidence of *H. pylori* among youngsters is 12.2%, whereas in India, it is 55.9% among people aged 11–16. [15, 16], and in northern Jordan is 82% [17] and in Gaza strip is (72.2%) [18].

2.4 Mode of transmission

2.4.1 Person-person route

Humans have been identified as a major *H. pylori* reservoir [19]. Person to-person contact is believed to be the primary route of transmission in developed and developing countries. Close personal relationships, especially those inside the family, between parents and children, siblings and siblings, and spouses and spouses, has been consistently verified as a major factor of transmission [20].

2.4.2 Oral-oral route

Using a polymerase chain reaction, *H. pylori* DNA has been found in the saliva of people who tested positive [21]. Additionally, *H. pylori* bacteria have been effectively found in infected dental plaque. However, isolation has not always been effective, possibly due to *H. pylori*’s transitory existence in the buccal cavity or low detection capabilities brought on by the co-existence of several other microorganisms.

2.4.3 Fecal-oral route

Fecal-oral is the main route of *H. pylori* transmission, *H. pylori* has been identified in human feces by culture and by PCR of its DNA. [22] Nevertheless, this has not been replicated by other researchers [23]. These findings provided evidence of the potential contribution of *H. pylori* fecal shedding into the environment.

2.4.4 Iatrogenic transmission

Endoscopes frequently utilized for upper gastrointestinal procedures, because they aren't properly disinfected in between procedures, could be the cause of an iatrogenic infection [24].

2.4.5 Mechanisms of infection

Most *H. pylori* are located in the gastric mucosa of the stomach; however, a few are also observed adherent to the gastric mucosal epithelium. The bacteria
are well suited to survive in the harsh conditions of the stomach, where very few others organisms can. Despite the fact that *H. pylori* is thought of as an extracellular bacteria, there is evidence that the bacteria have a method of intracellular invasion [25]. The human stomach is colonized by *H. pylori*, and about 50% of the worldwide people is colonized by it. Its gastric mucosa infection has been related to a variety of upper gastrointestinal tract illnesses, including chronic gastritis, peptic ulcer, stomach cancer and mucosa-associated lymphoid tissue lymphoma [26]. *H. pylori* typically results in an asymptomatic stomach infection, and documented side effects of this infection include chronic gastritis, peptic ulcer disease, and atrophic gastritis. Most infected individuals remain asymptomatic despite the fact that the infection almost always results in stomach inflammation, whereas a small percentage of people develop atrophic gastritis [27]. During its progression, the disease can have several manifestations including acute gastritis, chronic atrophic gastritis, intestinal metaplasia, dysplasia, growth failure, malnutrition and finally cancer [28]. *H. pylori* is the major cause of histologic gastritis and also plays an important role in the development of peptic ulcers, gastric carcinoma, and primary gastric B-cell lymphoma [29].

The etiology of atrophic gastritis and gastric cancer has been rewritten since the detection of *H. pylori* during the 1980s. *H. pylori* infection, which typically develops in early childhood and lasts a lifetime if untreated, is now recognized as the primary cause of atrophic gastritis [27]. One severe consequence of atrophic gastritis is the malabsorption of cobalamin (vitamin B₁₂), which is frequent in the elderly due to hypo- or achlorhydria with subsequent bacterial overgrowth, and reduced production and secretion of intrinsic factor. Carmel et al., hypothesized that *H. pylori* infection may be crucial in the decline in acid production, the reduction in intrinsic factor secretion, and the subsequent emergence of vitamin B₁₂ insufficiency [30]. *H. pylori* inhabiting the whole gastric epithelial and has a significant urease activity that results in the creation of ammonia to protect itself against the acidity of the stomach. It also produces other enzymes, including glycosulfatase and phospholipase A2 and C, which are associated with the development of stomach mucosal injury [31]. *H. pylori* induces an inflammatory response through the gastric epithelium, with production of pro-inflammatory cytokines, such as interleukin 1β and interleukin 8. Some *H. pylori* genotypes, especially those vacuolating toxin A (Vac-A) and cytotoxin-associated gene A (Cag-A) positive, are associated with greater pathogenicity and more severe sickness. Cag-A positive strains cause the stomach mucosa to react more violently to inflammation and produce more pro-inflammatory cytokines. Even while it only phenotypically manifests in 60% of *H. pylori* strains, the VacA gene, which causes the vacuolization and death of gastric epithelial cells, is genetically expressed in all of them [32]. Gastritis, including atrophic and non-atrophic gastritis, and peptic ulcers, are etiologically linked to *H. pylori* (especially duodenal ulcer). The primary gastric B-cell lymphoma (also known as mucosa-associated-lymphatic-tissue or MALT-lymphoma) and stomach adenocarcinoma have a strong correlation with *H. pylori*. *H. pylori* has been therefore classified by IARC/WHO as “group 1 carcinogen” [33].

### 2.5 Diagnosis of *H. pylori* infection

Infections are typically diagnosed by looking for dyspeptic symptoms and performing tests that may reveal *H. pylori* infection [34]. The diagnostic tools for *H. pylori* are serology, rapid urease test (RUT), urea breath test (UBT), endoscopy and biopsy/
Changes in Vitamin B₁₂, Iron, Thyroid Hormones, Thyroid Autoantibodies and Hematological...
DOI: http://dx.doi.org/10.5772/intechopen.108036

histopathology, PCR, for DNA of *H. pylori* and *H. pylori* stool antigen (HpSA). The simplest test of *H. pylori* is serologic, including the assessment of specific IgG level in serum [27, 35].

3. Anemia

3.1 Definition of anemia

Anemia is the most common blood disorder is characterized by a decrease in the number of red blood cells or a less-than-normal quantity of hemoglobin in the blood. The most widely used standards of anemia are those set by the World Health Organization, which identify hemoglobin levels of less below 12 g/dL for women and bellow 13 g/dL for males [36]. Globally, the most prevalent type of anemia was iron deficiency. It is a major public health issue that affects both advanced and developing societies, having a significant negative impact on people's health as well as social and economic development.

3.2 Common causes of anemia

*Anemia from active bleeding:* Heavy menstrual bleeding or, wounds and gastrointestinal ulcers or cancers [37, 38]. *Iron deficiency anemia:* Inadequate food intake, poor health and improper care [39]. *Anemia of chronic disease:* Long-term medical condition such as a chronic infection or a cancer [40]. *Anemia related to kidney disease:* Diminsh production of renal erythropoietin which in turn diminishes the production of RBC [41]. *Anemia related to pregnancy:* Water weight gain during pregnancy dilutes the blood, which may be reflected as anemia [42]. *Anemia related to poor nutrition:* Deficiency of vitamins and minerals required to make RBC [42]. *Pernicious anemia:* A problem in the stomach or the intestines leading to poor absorption of vitamin B₁₂ [43]. *Sickle cell anemia:* Is due to a point mutation in the β globin gene, resulting in the creation of abnormal hemoglobin molecules with a hydrophobic motif that is exposed in its deoxygenated state [44]. *Hemolytic anemia:* hemolysis-related anemia, which is caused by the abnormal breakdown of RBC, blood vessels, and extravascular locations throughout the body [45]. *Thalassemia:* This is another group of hereditary anemia of hemoglobin related causes. It varies in severity from mild thalassemia minor to severe thalassemia major [46]. *A plastic anemia:* Occasionally some viral infections may severely affect the bone marrow and significantly diminish production of all blood cells chemotherapy (cancer medications) and some other medications may pose the same problems and radiation [47].

3.3 Iron deficiency anemia

3.3.1 Definition

Iron deficiency anemia (IDA) is A decrease in overall hemoglobin concentration caused on by a deficiency of iron required for maintaining normal physiologic processes. Iron deficiency anemia results from inadequate iron absorption to accommodate an increase in requirements attributable to growth or arising from a prolonged negative iron balance, one of these conditions causes a reduction in iron storage as indicated by blood ferritin levels or bone marrow iron content [48].
3.3.2 Causes

Iron-deficiency anemia may develop from a variety of conditions, including stomach ulcers, ulcerative colitis, piles, and colon cancer, which can all induce gut bleeding and result in anemia. Anemia can be brought on by bleeding brought on by kidney or bladder illness. Anemia caused by iron deficiency can be brought on by a number of illnesses, including cancer and rheumatoid arthritis. Iron deficiency anemia is correlated with long-term aspirin use [49].

3.3.3 Diagnosis of iron deficiency anemia

Iron deficiency anemia were diagnosed by the first result on a regular complete blood count is typically low hemoglobin in the context of a lowered MCV, and the ferritin level was below 1010 ng/dl [50].

3.3.4 Pathophysiology of iron deficiency by H. pylori

Common symptoms of IDA include: breathlessness, tiredness, dizziness, tachycardia, headache and paleness [51]. The pathophysiologic mechanisms by which H. pylori is associated with the development of ID and ID anemia are not fully understood. It is still not known why some patients manifest this association and why in other patients it is not present, or there are other associations; or why some of the infections are asymptomatic [15]. Over the past decade, it has been linked H. pylori and ID development with a recently discovered hormone called hepcidin [52]. This hormone is produced in the liver and regulates iron metabolism in enterocytes and releases stored iron from macrophages of the reticuloendothelial system [53]. Hepcidin increases following H. pylori infection and acts as an acute phase reactant in reaction to the inflammation created in the gastric mucosa, culminating in a condition characterized as chronic illness or inflammatory anemia [54]. According to preliminary research, serum levels of hepcidin were raised in H. pylori-infected patients but returned to normal after the infection was eradicated, allowing the iron to be absorbed by enterocytes and freed from reticuloendothelial system macrophages, where it had been trapped [55]. Other possible causes of iron imbalance in patients infected with H. pylori are chronic gastritis, which occurs in all individuals infected with H. pylori [15]. This can cause bleeding when it becomes erosive gastritis, especially in patients with active bleeding peptic ulcers [56] and in patients who chronically ingest non-steroidal anti-inflammatory drug including aspirin [43].

3.4 Vitamin B<sub>12</sub>

3.4.1 Definition and structure

Vitamin B<sub>12</sub> or cyanocobalamin is relatively large and complex water-soluble vitamin. The molecular weight of vitamin B<sub>12</sub> is equal to 1355.4 [57]. All cobalamins that may be physiologically active are represented by vitamin B12. The name “cobalamin” is used to describe a class of cobalt-containing substances known as corrinoids, each of which has a lower axial ligand that contains a cobalt-coordinated nucleotide (5,6-dimethylbenzimidazole as a base. Cyanocobalamin, which is used in most supplements, is readily converted to the coenzyme forms of cobalamin (methylcobalamin and 5-deoxyadenosylcobalamin) in the human body [58]. The partial structures
Changes in Vitamin B₁₂, Iron, Thyroid Hormones, Thyroid Autoantibodies and Hematological...
DOI: http://dx.doi.org/10.5772/intechopen.108036

of vitamin B₁₂ compounds show only those portions of the molecule that differ from vitamin B₁₂: 1: 5-deoxyadenosylcobalamin; 2, 'methylcobalamin; 3, hydroxocobalamin; 4, sulfitocobalamin; 5, cyanocobalamin or vitamin B₁₂ [57].

3.4.2 Sources of vitamin B₁₂

Vitamin B₁₂ is synthesized only in certain bacteria [59]. In the natural food chain system, more predatory organisms have larger concentrations of vitamin B₁₂ that bacteria produce. The main dietary sources of vitamin B₁₂ are thought to be animal foods (meat, milk, eggs, fish, and shellfish) rather than plant foods. [58]. Some plant foods, such as edible algae or blue-green algae (cyanobacteria), however, contain large amounts of vitamin B₁₂. Vitamin B₁₂ compounds in algae appear to be inactive in mammals [60]. Foods contain various vitamin B₁₂ compounds with different upper ligands; methylcobalamin and 5-deoxyadenosylcobalamin function, respectively, as coenzymes of methionine synthase (EC 2.1.1.13), which is involved in methionine biosynthesis and of methylmalonyl-CoA mutase (EC 5.4.99.2), which is involved in amino acid and odd-chain fatty acid metabolism in mammalian cells [61]. Humans have a complex process for gastrointestinal absorption of dietary vitamin B₁₂ [62]. The recommended dietary allowance of vitamin B₁₂ for adults is set at 2.4 μg/day in the United States and Japan; however, daily body loss of the vitamin is estimated to be between 2 and 5 μg/day [63]. According to a study by Bor et al., in 2006, a daily consumption of 6 μg of vitamin B12 is sufficient to maintain a stable level of serum vitamin B₁₂ and vitamin B₁₂-related metabolic indicators [64].

3.4.3 Vitamin B₁₂ functions

Cobalamin, or vitamin B₁₂, comes in a variety of forms, such as cyano-, methyl-, deoxyadenosyl-, and hydroxy-cobalamin. Food contains small amounts of the cyano form, which would be utilized in supplements. The other forms of cobalamin can be changed into the methyl- or 5-deoxyadenosyl forms that seem to be necessary as cofactors for L-methyl-malonyl-CoA mutase and methionine synthase. For the formation of purines and pyrimidines, methionine synthase is necessary. The reaction, in which the methyl group of methyltetrahydrofolate is transferred to homocysteine to generate methionine and tetrahydrofolate, requires folate as a co-factor and also depends on methylcobalamin. Megaloblastic anemia develops as a result of a vitamin B12 shortage and the disruption of the process that causes RBCs to mature. Megaloblastic anemia is also brought on by a folate deficit, which is unrelated to vitamin B12 [65]. Methylmalonyl CoA mutase changes methylmalonyl CoA into succinyl CoA, and it needs the cofactor 5-deoxyadenosylcobalamin to do so. The neurological consequences of vitamin B12 deficiency are assumed to be caused by a flaw in this process and the accompanying buildup of methylmalonyl CoA [65].

3.4.4 Deficiency of vitamin B₁₂

Vitamin B₁₂ deficiency is usually caused by the malabsorption of vitamin B₁₂ although dietary inadequacy is common in the elderly, vegans or ovo-lacto vegetarians with poor diets. Other contributing factors include insufficient intrinsic factor synthesis, atrophic gastritis, disease-related disruption of vitamin B12 absorption in the ileum, bacterial overgrowth, resection, drug-nutrient interactions, and other less prevalent genetic abnormalities [66]. Pernicious anemia is the end stage of an
auto-immune gastritis and results in the loss of synthesis of IF. It is this loss of IF that causes vitamin B\textsubscript{12} deficiency and if untreated, megaloblastic anemia and neurological complications develop [66].

3.4.5 Mechanism of vitamin B\textsubscript{12} deficiency

A mechanism that has been proposed to explain this association is that the action of \textit{H. pylori} decreases gastric acid secretions which leads to hypochlorhydria [67]. On the one hand, protein-bound vitamin B\textsubscript{12} must be released by the action of gastric acid in the stomach, yet hypochlorhydria itself increases the bacteria of the stomach and intestines. These bacteria may in turn make use of the vitamin B\textsubscript{12} themselves [68]. This mechanism is supported decreased vitamin B\textsubscript{12} levels secondary to chronic use of PPIs [69]. In addition, it has been proposed that vitamin B\textsubscript{12} deficiency is secondary to decreased production of intrinsic factor due to atrophic gastritis (pernicious anemia) which results from chronic \textit{H. pylori} infections [70]. However, one study has concluded that the association between \textit{H. pylori} and vitamin B12 deficiency is independent of atrophic gastritis [71].

3.5 Thyroid hormones and autoantibodies

3.5.1 Thyroid hormones

The thyroid gland, which is shaped like a butterfly and is located at the base of the neck right behind the larynx, generates the essential hormones T4 and T3 [72]. Thyroid hormones are essential for numerous functions including: brain development, growth, fuel metabolism, reproduction, regulate body temperature and blood pressure [73]. TSH, which is made by the pituitary gland and regulates the production of T3 and T4, was responsible for controlling T3 and T4 levels. TSH production controlled by thyroid releasing hormone (TRH) produced by the hypothalamus [74]. This means that thyroid gland regulates its hormonal secretion with the aid of hypothalamus and the pituitary gland in a way that TRH is triggered pituitary to secrete TSH which in turn tells thyroid gland to capture iodine from the blood to synthesized and produced T4 and T3. Hypothalamus and pituitary gland decrease TRH and TSH when T4 is reach to a satisfactory level in circulation [75].

3.5.2 Thyroid autoantibodies

Auto-antibodies cause cellular damage and modify thyroid gland function. Sensitized T-lymphocytes and/or autoantibodies that attach to thyroid cell membranes result in cell lysis and inflammatory responses, causing cellular damage. Alterations in thyroid gland function result from the action of stimulating or blocking auto-antibodies on cell membrane receptors. TPO, Tg, and the TSH receptor are the three main thyroid auto-antigens involved in autoimmune thyroid disease (ATD). [75]. Thyroid peroxidase is the key enzyme catalyzing both the iodination and coupling reaction for the synthesis of thyroid hormone. It is membrane-bound and found in the cytoplasm of thyrocyte. It was earlier known as thyroid microsomal antigen. Anti-TPO autoantibodies are found in patients with autoimmune hypothyroidism and Graves’ disease (GD). Together with Tg antibodies, these are the predominant antibodies in Hashimoto’s thyroiditis. Anti-TPO antibodies are mainly of the IgG class with IgG1 and IgG4 subclasses in excess [76].
Thyroglobulin made out of two identical subunits. It is discharged by the thyroid follicular cells into the follicular lumen and stored as the colloid. Each Tg molecule has around 100 tyrosine residues. These deposits were coupled to form the thyroid hormones T3 and T4. The sequence of human Tg has been determined [77]. Thyroglobulin autoantibodies are found in patients with lymphocytic thyroiditis and Graves’ disease patients. They are polyclonal and mainly of IgG class with all four subclasses represented. TSH controls the cell surface expression of TPO and Tg altering the mRNA transcription of these two proteins. Both blocking and stimulating Autoantibodies are found in the sera of GD patients replicate these effects [78].

Our previous experimental results [79] indicated statistically significant positive correlation between TSH levels and anti TPO and anti Tg at baseline. Also, there were statistically significant negative correlations between fT3, fT4 levels and anti-TPO and anti-Tg. This result agrees with a previous study in which statistically significant positive correlations between TSH levels and anti TPO and anti-Tg were reported. Also these findings are in agreement with that obtained by [80]. Lin et al. (2014) who found elevated levels of both anti-Tg and anti-TPO in patients with radiation-induced hypothyroidism, in addition to positive correlation between TSH and anti-Tg and a negative correlation between fT4 and anti-TPO [80].

Hou et al. (2017) who observed reduction of thyroid autoantibodies in patients with GD and HT after pharmaceutical eradication of H. pylori infection [81]. H. pylori plays role in ATD pathogenesis. Genetic factors include thyroid specific genes and immune regulatory genes while none genetic factors include: smoking, stress, iodine intake, medication, pregnancy and bacterial and virus infection that have been implicated with etiology of ATD [82]. Strong correlation between IgG anti–H. pylori antibodies and thyroid auto-antibodies as well as the observation that eradication of H. pylori infection is followed by gradual decrease in the levels of thyroid auto-antibodies, supposed that H. pylori antigens might be involved in the development of autoimmuno atrophic thyroiditis or that autoimmuno function in this disease may increase the likelihood of H. pylori infection [83]. El-Eshmawy et al., (2011) who found that a correlation between H. pylori infection and the presence of autoantibodies against thyroid antigens, and highly significant prevalence of H. pylori infection in the ATD patients when compared with healthy individuals [84].

4. Conclusions

H. pylori appears to play a role in the onset of IDA and vitamin B12. H. pylori patients had significant decreases in vitamin B12, serum iron and hemoglobin levels. An insufficient response to the medication may be caused by H. pylori gastritis. A raise in H. pylori IgG, anti-TPO, anti-TG, and TSH levels and a decrease in fT4, fT3, and other hematological markers appear to before H. pylori treatment in hypothyroidism Palestinian females. Patients receiving triple therapy for H. pylori infection OAC may help patients feel better overall by restoring their vitamin B12, serum iron, and hemoglobin levels. In people with gastritis, vitamin B12 levels are highly associated with Hb and RBCs. As a result, it could be thought of as a helpful indicator for patients with anemia with gastritis. H. pylori is responsible for hypothyroid patients getting large doses of L-T4 poor response. However, these parameters were nearly improved by 14-day conventional triple treatment with omeprazole, amoxicillin, and clarithromycin. Routine testing of vitamin B12, iron, ferritin and total iron binding.
capacity level and immunological thyroid alterations were recommended for *H. pylori* patients and gastroscopy confirmation of *H. pylori* infection.

**Conflict of interest**

The authors declare no conflict of interest.
References


[17] Bani-Hani KE, Hammouri SM. Prevalence of *Helicobacter pylori* in Northern Jordan. Endoscopy based...


[35] Tiwari SK et al. Rapid diagnosis of Helicobacter pylori infection in dyspeptic patients using salivary secretion: A
Changes in Vitamin B<sub>12</sub>, Iron, Thyroid Hormones, Thyroid Autoantibodies and Hematological... DOI: http://dx.doi.org/10.5772/intechopen.108036


[54] Cherian S et al. An insight into the relationships between hepcidin, anemia, infections and inflammatory cytokines


[64] Bor MV et al. A daily intake of approximately 6 μg vitamin B-12 appears to saturate all the vitamin B-12-related variables in Danish postmenopausal women. The American Journal of Clinical Nutrition. 2006;83(1):52-58


[70] Lahner E, Persechino S, Annibale B. Micronutrients (other than iron) and Helicobacter pylori infection: A systematic review. Helicobacter. 2012;17(1):1-15


University of Science & Technology; 2018


[80] Lin Z et al. Longitudinal study on the correlations of thyroid antibody and thyroid hormone levels after radiotherapy in patients with nasopharyngeal carcinoma with radiation-induced hypothyroidism. Head & Neck. 2014;36(2):171-175


