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REVIEW ARTICLE

The Role of *Helicobacter Pylori* Bacterium in Cardiovascular Disease

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Abstract

Introduction Atherosclerosis, ischemic heart disease, myocardial infarction, and coronary artery disease are just a little of the cardiovascular anomalies that frame heart disease. It is the most important reason of death in any country on the globe. Heart disease has been associated to a number of general and new risk factors, counting infectious and noninfectious Infectious agents.
Objective Understand The Role of *Helicobacter pylori* bacterium in cardiovascular disease
Results and Discussion Accumulation levels indicated in this study were significantly higher in samples taken from Al-Hay brick factories than those taken from Al-Ahdeb oil fields, except for Lead (Pb), which was detected to be higher in samples of Al-Ahdeb oil fields. All concentration levels detected were high and considered hazardous to the health of organisms habituating within polluted areas since accumulation in tissues was reported to cause harmful negative effects on kidney function.
Conclusion Accumulation levels indicated in this study were significantly higher in samples taken from Al-Hay brick factories than those taken from Al-Ahdeb oil fields. All concentration levels detected were high and considered hazardous to the health of organisms habituating within polluted areas.

Keywords: Cardiovascular diseases; Helicobacter pylori; lipid profile.

1 Introduction

1.1 *H. pylori* (an over review)

The bacterium Helicobacter pylori is microaerophilic and unipolar. The exterior membrane is composed of five major protein families. The largest family is putative adhesions, which is followed by porins, iron, transporters, proteins linked to the flagellum, and proteins with unusual roles. Because it is a Gram-negative bacterium, lipopolysaccharides and phospholipids make up its exterior membrane, and it also contains these substances. The bacteria's rapid motility is partly attributed to the presence of two to six lophotrichous flagella. similar to Figure 1. These sheathed flagellar filaments are composed of two copolymerized flagellins, flagellin A and flagellin B.[1–5] Their dimensions are 0.5–1 mm in diameter and 2.5–5.0 mm in length. They desire between 5 and 10 percent carbon dioxide (CO_2) and about 5% oxygen (O_2). The enzymes urease, catalase, and oxidase are produced by *H. pylori*. Bacteria are able to withstand the harsh

conditions found in the stomach because they produce urease, a potent enzyme that breaks down urea, a substance produced by stomach cells, into carbon dioxide and ammonia. By balancing the acidity of the mucus surrounding the bacterium, this creates a protective environment for the bacteria. Multiple bacterial genome sequences account for the differences amongst *H. pylori* strains. Being one of the most genetically varied bacteria [6–10] species, it produces a wide range of toxins. Its high virulence factors have led to its association with a range of illnesses. Two of the most pathogenic factors are vacuum-occurring cytotoxin gene A (Vac A) production and Cag A production. Heart disease has been connected to *H. pylori* strains that generate cag A. These *H. pylori*-toxic nutrients increase the risk of heart disease by inducing inflammation and cellular damage in host cells and quickly inducing host factors such as interleukins (ILs; IL-1, IL-2, IL-6, IL-8, and IL-12), interferon-gamma, tumour necrosis factor-, T and B lymphocytes, and phagocytic cells [1–16].

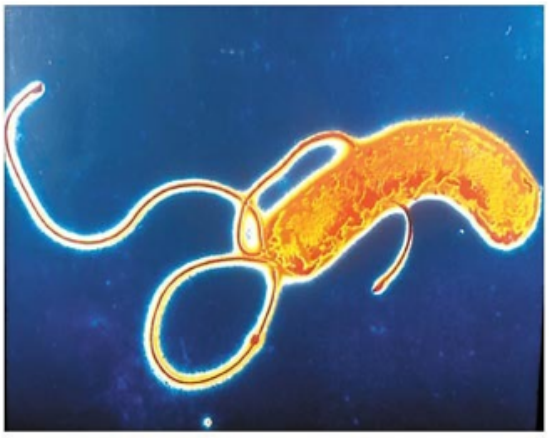

Figure 1: Helicobacter pylor [17].

2 Atherosclerosis

Heart disease, also known as cardiovascular disease, refers to a variety of heart conditions such as elevated blood pressure, blocked arteries, and coronary artery disease. Heart disease includes a large range of disorders that affect the heart and blood vessels [18]. High blood pressure, high levels of fat, obesity, a high-fat diet, lack of physical activity, diabetes and stress are all risk factors. Microorganisms such as chlamydia pneumonia, cytomegalovirus, Epstein-Barr virus, HIV, herpes simplex, and Helicobacter pylori play a role in the development of heart disease and are considered risk factors [19–22] .*H. pylori* infection has been linked to chronic gastrodigestive ulcers, but an epidemiological analysis focused on bacteria discovered in the last two decades suggests that a higher prevalence of *H. pylori* infection may play a role in the pathogenesis of heart disease [23–25] .A bacterium has been suggested as one of the possible pathways that can trigger direct and indirect effects on heart disease, according to one hypothesis. The major underlying causes of heart disease are inflammatory and immunological events triggered by the *H. pylori* infection [26, 27]. Infection may have both direct and indirect effects on cardiovascular function. Endothelial damage, dysfunction through circulating endotoxins, smooth muscle proliferation, and local inflammation are among the direct effects, while proinflammatory, hypercoagulability, atherogenic action, development of cross reactive antibodies, oxidation of low density lipoprotein, and molecular oxidation are among the indirect effects [28–33] As in Figure 2 . Recent research has linked a highly virulent strain of *H. pylori* [cytotoxin associated gene A (Cag A) strain] to an increased risk of heart disease, which was verified by the existence of anti *H. pylori* antibodies as a result of seroprevalence [34, 35]. The aim of this review is to illustrate all possible pathways that cause heart disease in people who have *H. pylori* infection.

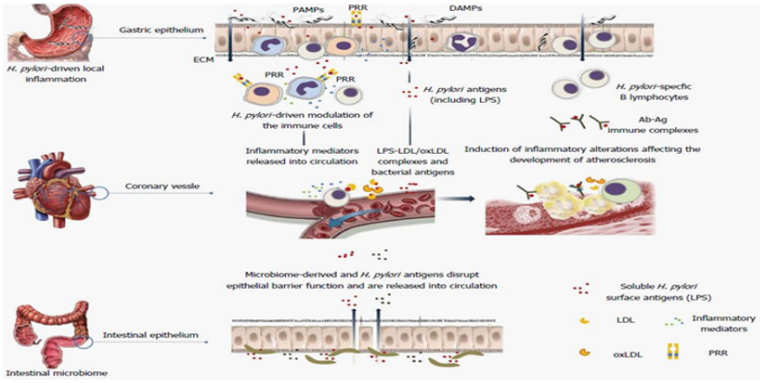

Figure 2: A possible link between local inflammation induced by Helicobacter pylori on surface of the gastric epithelium and the inflammatory response within vascular endothelium [36].

3 *H. pylori* and Acute Coronary Syndrome

Acute Coronary Syndrome (ACS) encompasses a range of clinical symptoms, namely unstable angina, ST elevation myocardial infarction (STEMI), and non-ST elevation myocardial infarction (NSTEMI), which are caused by *H. pylori*. There has been a decrease in blood flow within the coronary arteries.The number is [37] Established a correlation between the presence of H. pylori infection in the coronary artery wall and the formation of atherosclerotic plaques, which occurs through the elevation of LDL-C levels in the bloodstream. The study revealed a strong correlation between coronary heart disease (CHD) and both H. pylori infection and anti-Cag-A positive [38]. In a separate investigation, it was observed that dysfunctional angina patients exhibited elevated levels of Cag-A compared to healthy angina patients. Patients versus individuals who are considered safe. Cag-A antigens were detected in the coronary arteries. Atherosclerotic plaques affect both unhealthy and healthy individuals. Thirteen patients As per an additional report, the individual in question Another study demonstrated a positive correlation between H. pylori seropositivity [39] and mortality due to acute myocardial infarction. Individuals diagnosed with cardiac syndrome have angina and exhibit ST segment depression on a stress test, however show no abnormalities on a coronary angiography. The cause of this condition is currently not understood, however it is possible that endothelial dysfunction has a role in the development of cardiac syndrome [40,41]. H. pylori infection in individuals results in chronic inflammation, which causes an increase in C-reactive protein (CRP) and interleukin-1 (IL-1) levels.Endothelial dysfunction, which can contribute to the development of cardiac syndrome, is associated with levels of IL-1 [42]. As stated by Eskandarian et al [43].The incidence of H. pylori infection is greater in individuals with cardiac syndrome compared to those who are in good health [44]. Chronic infections typically promote the development of atherosclerosis by disrupting the metabolism of lipids and lipoproteins. Several studies have established a correlation between H. pylori seropositivity and decreased levels of high density lipoprotein cholesterol (HDL-C), as well as increased levels of triglycerides (TG) and total cholesterol.

4 Pathological Mechanism

Atherosclerosis is a chronic inflammatory disorder in the artery wall that can result in ischemia and subsequent complications in the circulatory system [45]. A relationship between H. pylori infection and atherosclerosis was identified in an animal model [46].

Additionally, it was found that CagA has the ability to induce foam formation in macrophages, hence exacerbating the growth of atherosclerotic plaques and causing dysfunction in the arteries. In addition, plaques assimilate exosomes formed from H. pylori-infected gastric epithelial cells (Hp-GES-EVs), resulting in the release of CagA within them. This occurrence intensifies the obstructive inflammatory process, leading to the formation of lesions both in laboratory settings (in vitro) and in living organisms (in vivo) [46]. According to a study examining the relationship between H. pylori and cardiovascular risk factors, this bacterial infection has the ability to promote the development of atherosclerosis by impacting the patient's lipid profile.The results indicated that individuals infected with H. pylori had elevated levels of total cholesterol and low-density lipoprotein (LDL), but their levels of high-density lipoprotein (HDL) were decreased [47]. Another recent study, within this environment, supported the theory.The study showed that eliminating H. pylori had a significant positive effect on lipid levels in persons with dyslipidemia. It increased HDL levels and reduced the LDL/HDL ratio, which is an important indicator of atherosclerosis risk [48].

Given that infection is a complex occurrence, it is not possible to attribute infection-related cardiac disease to a single component.

Cardiovascular issues resulting from infection are produced by various potential processes and pathways associated with *H. pylori*. The mechanisms via which *H. pylori* contributes to cardiac disease are briefly reviewed in Figure 3.

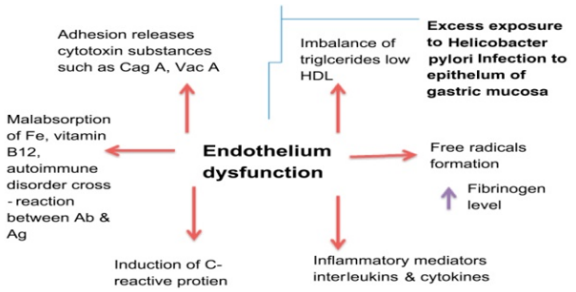

Figure 3: *Helicobacter pylori* infection induced immune response (29).

Inflammation is a complex process involving multiple steps that serves as a defence mechanism for the immune system. Chronic *H. pylori* infection leads to a breakdown in the body's inflammatory response and its ability to fight against infections, which can result in several diseases including heart disease and cancer. Continuous exposure to excessive stress leads to a deterioration of the body's strength and vital-

ity [49–51].Persistent activation of inflammatory responses in the gastrointestinal tract and stomach leads to dyslipidemia, increased levels of fibrinogen, release of C-reactive protein, elevated blood leukocytes and homocysteine, heightened blood clotting tendency, stimulation of immune cross-reactivity, and elevated levels of proinflammatory cytokines (interleukins, lymphocytes) and other cytotoxins [19, 51, 52].Chronic *H. pylori* infection causes disruption of the immune system, resulting in cardiovascular abnormalities, specifically coronary artery issues [53–55] as seen in Figure 4.

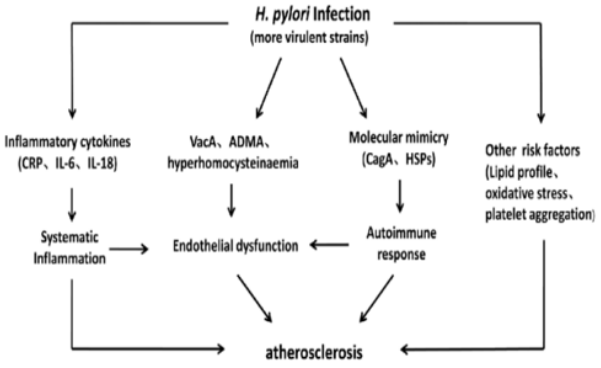

Figure 4: Postulated mechanisms by which Helicobacter pylori infection may contribute to the development of atherosclerosis [56].

5 Conclusions

The presence of *H. pylori* infection is associated with the occurrence of cardiovascular illnesses, either through a direct or indirect mechanism. The active component has been identified by numerous serological studies. The involvement of *Helicobacter pylori* (*H. pylori*) in the pathogenesis of cardiovascular disease Inflammation is triggered by the activation of inflammatory mediators, which include proinflammatory factors and toxin-free substances.The primary mechanisms by which *H. pylori* contributes to cardiovascular abnormalities are disrupted lipid metabolism, modified iron metabolism, and autoimmune response. The chief executive Inadequate dietary choices significantly contribute to the onset of cardiovascular disease.The functionality of the immune system at both the cellular and systemic levels. The majority of these conclusions mostly pertain to serological data. Further investigation is necessary to ascertain the precise function of *H. pylori* in these conditions.

Conflict of Interest: No conflicts of interest exist between the authors and the publication of this work.
Ethical consideration: The ethical committee approved the study at University of Thi-Qar, Nassiryhia, Iraq.

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