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THE RELATIONSHIP BETWEEN HELICOBACTER PYLORI AND ARTERIAL HYPERTENSION WAS ANALYZED

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Abstract
The literature review analyzes studies on the role of Helicobacter pylori in the development of hypertension in recent years, using data from the scientific literature, important data aimed at solving the mutual pathogenetic relationship between the two problems. The result of several tests is H. pylori: people with pylori have a higher risk of developing hypertension than people who are not infected. H. pylori has been shown to be the pathological mechanisms of pylori's effect on the body. H. pylori The mutual pathogenetic dependence in the development of pylori and hypertension is based on data on the occurrence of endothelial dysfunction, which is accompanied by an increase in the number of inflammatory cytokines, homocysteinemia, and a decrease in vitamin D.

Keywords: arterial hypertension, Helicobacter pylori, risk factor, endothelial dysfunction, pathogenetic dependence, homocysteinemia.

Introduction

Arterial hypertension is an independent, chronic disease, the main manifestation of which is the syndrome of arterial hypertension, which is not associated with other causes. Despite the development of modern medicine, arterial hypertension (AH) remains a medical and social problem due to its wide prevalence and severity of complications. This is due to the wide spread of this disease, the lack of blood pressure control even with drug therapy in a significant part of patients, and the fact that hypertension is the most important risk factor for the main cardiovascular diseases - myocardial infarction (MI) and cerebral stroke, which mainly determine the high mortality rate in the world. According to the forecast, by 2025, the number of hypertension patients in the world will increase by 15–20% and reach almost 1.5 billion [1].

Recently, the number of people suffering from hypertension has been increasing from year to year, and their age is becoming younger. For a long time, hypertension was considered as a multifactorial disease. The prevalence of hypertension in developing countries is seen as a consequence of progress and changes in lifestyle. In addition to smoking, genetics, diet, and other factors, H. pylori has been considered a potential risk factor for hypertension in recent years. Despite this, traditional risk factors do not appear to be sufficient to explain the rising rates of hypertension, which further indicates that attempts to control the problem with traditional measures may never be adequate or decisive [2].

The prevalence of Helicobacter pylori among all segments of the population is very wide. According to the literature, more than half of people worldwide are infected with this bacterium. In Africa, Mexico, South and Central America, the prevalence of this infection reaches 70-90% among the entire adult population. A number of scientific studies are being conducted around the world to study various variants of the manifestation of Helicobacteriosis. Today, the high rate of infection in the population with H. pylori makes it the biggest hidden danger to human health. Ever since H. pylori was discovered in the human stomach, it has long been thought to be associated only with gastrointestinal diseases. Currently, many studies have shown that H. pylori is etiologically associated with many extraintestinal diseases.

Helicobacter pylori (H. pylori) is a spiral-shaped, gram-negative micro-aerophilic bacillus. Its morphology is heterogeneous in the sense that it can take a helicoidal, spiral or curved shape, with 2-6 flagella, a bacterium that naturally colonizes the epithelium of the human stomach.Its dimensions range from 0.5 mm to 1.0 mm in diameter and from 2.5 mm to 5.0 mm in length. It is characterized by the production of urease, which, through the production of ammonia, creates a microenvironment with a pH higher than the pH of the gastric mucosa, which allows it to survive. Cultivation of *helicobacter pylori* is somewhat difficult because it requires a longer incubation period than most bacteria (5 days instead of 24 hours). and enriched culture media should be used.

In the course of studying over this topical topic, the results of the latest new research have emerged, a large body of evidence strongly suggesting a causal, causal relationship between H. pylori and extragastric disorders, this information give us Helicobacter plori affect not only the gastroentinal tract can specific negative effects other extragastric disorders for example such as metabolic disorders including, disorders especially cases stroke psychiatric,[4] complications of gynecological disorders,[5] from severe vomiting to preeclampsia,[6,7] and especially it can be pointed out that during the research of the work of the author Izadi we can see Helicobacter plori undoubted cause of such diseases of the ears, nose and throat from benign diseases to malignancies[8] such as, laryngeal carcinoma and lung cancer[9], haematological disorders[10] caused by iron deficiency anemia to idiopathic thrombocytopenic purpura ITP [11].

Based on several studies, the results confirmed that H. pylori is a vital risk factor for hypertension. People infected with H. pylori had a 13.4% higher risk of developing hypertension than uninfected individuals.

According to the results of scientific studies, it can be said that patients with hypertension who are positive for H. pylori have significantly higher blood pressure than patients with hypertension who do not suffer from infection. In addition, eradication of H. pylori has been reported to improve hypertension. H. pylori infection has been found to be an independent risk factor for carotid plaque formation and stroke. A number of studies have indicated an association between H. pylori and acute coronary syndrome, suggesting that H. pylori infection is a risk factor for cardiovascular CVD disease, moreover, patients with H. pylori

infection have an approximately 3 times higher risk of coronary heart disease than healthy individuals [14,15].

A number of studies have shown that mild systemic inflammation by provocations of H. pylori infection is associated with metabolic syndrome and atherosclerotic cardiovascular disease. Altered blood cholesterol levels, such as elevated levels of low-density lipoprotein (LDL) and decreased levels of high-density lipoprotein (HDL), are major risk factors for cardiovascular disease and metastatic N. pylori syndrome, infections may be associated with elevated LDL cholesterol levels, the most significant risk factor for atherosclerosis, and that eradication of H. pylori may play a positive role in the treatment of prophylactics atherosclerosis[16].

Pathogenic Mechanisms

Based on existing scientific research, various mechanisms have been proposed to explain the association of *H. pylori infection with cardiovascular disease.*

Possible mechanisms of H. pylori's effects on the body are:

1) activation of the inflammatory process with the production of cytokines, eicosanoids and other mediators[17];

2) molecular mimicry between bacterial antigens and components of macroorganism tissues with their further autoimmune damage[18];

3) interaction with mast cells with subsequent secretion of biologically active substances acting on blood vessels, bronchi, and other internal organs;

4) development of allergic reactions, predominantly of the immediate type;

5) reduction of the intestinal barrier function, leading to the entry of toxic products and allergens into the blood;

6) the absorption of macro- and microelements, in particular iron, for the processes of its vital activity and, consequently, the theft of the macroorganism[19].

Based on laboratory and experimental data, it has been hypothesized that inflammation plays a fundamental role in atherogenesis and acute thrombosis. From an epidemiological point of view, support for this hypothesis has been obtained in a series of prospective cohort studies that demonstrate that inflammatory parameters (such as fibrinogen, C-reactive protein, and serum amyloid A), cell adhesion molecules [such as intercellular adhesion molecule (ICAM)-1], and cytokines (such as interleukin-6) are all elevated baseline in patients at risk of future coronary occlusion. In addition, evidence from randomized clinical trials suggests that the efficacy of common prophylactics such as aspirin and hydroxy-methylglutaryl (HMG) CoA reductase inhibition may depend in part on interactions with the inflammatory system. Taken together, these findings raise the possibility that therapies that target low-grade chronic inflammation could provide new future strategies for preventing cardiovascular disease.

There is increasing evidence that inflammation plays an etiopathogenetic role in the development of atherosclerosis and that certain markers of inflammation are associated with a greater risk of coronary artery disease. Markers such as C-reactive protein (CRP),

blood leukocyte count, plasma fibrinogen, or the presence of heat shock proteins (TSS) worsen the prognosis of coronary artery disease[21].

Inflammation can also contribute to the development of hypertension by causing endothelial dysfunction and inducing oxidative stress. Minieco et al. hypothesized that H. pylori infection may lead to activation of a cascade of inflammatory cytokines with the release of vasoactive substances from the site of infection. Levels of various inflammatory cytokines, including IL-1beta, IL-2, IL-6, and TNF-alpha, were significantly elevated in individuals with H. pylori infection.These inflammatory cytokines can contribute to the development of insulin resistance. Insulin resistance can then further increase overall peripheral vascular tension. In addition, people with H. pylori may have elevated levels of fibrinogen, a biomarker of vascular inflammation that could inhibit a decrease in nitric acid (NO) levels, which in turn would cause vasoconstriction and increased peripheral blood vessel tension. Due to such a long, prolonged inflammatory reaction due to *H. pylori*, a chronic inflammatory reaction of low severity occurs, provoking an atherogenic process through changes in some cardiovascular risk factors, such as coagulation factors and lipids, with the release of fibrinogen, reactive protein C, TNF-α, and interleukin 6 (IL-6), in addition to an increase in the number of white blood cells in the blood, which can cause prothrombotic condition[23]. In adults, *H. pylori causes an active chronic inflammatory process with the presence of neurophils, T-lymphocytes, B-lymphocytes and plasma cells;* In other words, it triggers a reaction that is both cellular and brachial in nature. The specific cellular response is characterized by the activation of 1 helper T lymphocytes, causing an increase in the release of cytokines, especially IL-1, IL-6, IL-8, TNF-α, and interferon. The ability to induce cytokines differs in H. pylori strains, with CagA+ strains being observed to produce the most intense release and the greatest variety of cytokines, on the other hand, it has also been observed that soluble extracts of H. pylori *promote plaque aggregation in the gastric mucosal microcirculation[24].*

By focusing on inflammatory processes associated with H. pylori, chronic inflammation caused by H. pylori infection [25,26] activates various mediators that have been associated with endothelial cell dysfunction associated with MetS[25,26].The studies of Rasmi Y. and Raeisi S. studied the mechanism of endothelial dysfunction in the pathogenesis of cardiac syndrome X, caused by structural and functional disorders of endotheliocytes as a result of inflammation and proliferative changes from H. pylori, leading to a change in the elastic properties of blood vessels through pro-inflammatory cytokines, cell adhesion molecules, growth factors, and acute-phase proteins. Indeed, H. pylori increases levels of inflammatory mediators such as MetS-related tumor necrosis factor (TNF)-α, interleukin (IL)-1, IL-6, IL-8, interferon (IFN)-γ, fibrinogen, thrombin, intercellular adhesion molecule, and vascular cell adhesion molecule; these MetS-related inflammatory mediators directly or indirectly damage vascular walls, thereby causing atherosclerosis[28] H. pylori-mediated inflammation, has been associated with atherosclerosis and the above-mentioned inflammatory mediators have been implicated in the pathophysiology of MetS-associated hypertension.

Change in blood lipids H.pylori infection causes an increase in cholesterol and triglyceride levels with a decrease in HDL levels, contributing to the development of dyslipidemia, a known cardiovascular risk factor. A number of authors suggest that the formation of oxidants is also important. A decrease in antioxidants has been observed in patients with H. pylori, which may lead to the activation of lipid peroxidation and therefore to the development of atherogenesis, since the oxidation of low-density lipoprotein (LDL) is the first of the major steps in the atherogenic process. Cross-reactivity with antibodies of heat shock proteins (BTS) Another theory is anti-BTS antibodies with cross-reactivity. H. pylori has been shown to produce BTS-60 with a fairly new cardiovascular risk factor, as it has been observed that increased homocysteine levels are associated with an increased risk of cardiovascular disease. In this regard, patients with chronic gastritis, usually caused by Helicobacter pylori infection, have decreased absorption of vitamin B12 and folic acid, thereby causing secondary hyperhomocysteinemia.

Since traditional risk factors do not account for a subset of cases, homocysteine, a "new" risk factor, is being considered with increasing interest. Homocysteine is an intermediate in the metabolism of the proteinogenic amino acids methionine and cysteine. Numerous retrospective and prospective studies have consistently found an independent association between mild hyperhomocysteinemia and cardiovascular disease or all-cause mortality. Starting with a plasma homocysteine concentration of approximately 10 µmol/L, the increase in risk occurs according to a linear dose-response relationship without a defined threshold level. Hyperhomocysteinemia, as an independent risk factor for cardiovascular disease, is thought to be responsible for about 10% of the overall risk. Elevated plasma homocysteine levels (>12 µmol/L; mild hyperhomocysteinemia) are considered cytotoxic and occur in 5-10% of the general population and up to 40% of patients with vascular disease[30,31]. Hyperhomocysteinemia is associated with changes in vascular morphology, loss of endothelial antithrombotic function, and induction of the procoagulant medium. The most well-known forms of damage occur due to homocysteine-mediated oxidative stress. Numerous agents, drugs, diseases and lifestyle factors, especially acting as direct or indirect antagonists of cofactors and enzymatic activity, have an impact on homocysteine metabolism. Folic acid deficiency is considered the most common cause of hyperhomocysteinemia. Adequate folic acid intake of at least 400 mcg per day is difficult to maintain even with a balanced diet, and high-risk groups are often unable to meet these folic acid needs. Based on the available evidence, there is a growing need to diagnose and treat elevated homocysteine levels in high-risk individuals in general and patients with manifest vascular disease in particular. Subjects in both populations should first undergo a baseline homocysteine test. Unless manifestations are already present, intervention, if any, should be based on the severity of hyperhomocysteinemia. In line with the recommendations of other working groups and consensus groups, we recommend a target plasma homocysteine level of <10 µmol/L. Based on various calculation models, a reduction in elevated plasma homocysteine concentrations could theoretically prevent up to 25% of cardiovascular events. Dietary supplements are inexpensive, potentially effective, and free of side effects, and

therefore have an exceptionally favorable benefit-risk ratio. Results from current randomized controlled intervention trials should be available before screening for hyperhomocysteinemia and its treatment in an apparently healthy population can be recommended.

Hyperhomocysteinemia is an emerging cardiovascular risk factor as it has been observed that an increase in homocysteine levels is associated with an increase in cardiovascular risk. In this regard, in patients with chronic gastritis (usually caused by H. pylori infection), it may lead to decreased absorption of vitamin B12 [33] **.**

Deficiency of these substances can lead to impaired methionine metabolism and methylation deficiency causing elevated serum levels in patients with H. pylori infection. This is consistent with our finding that with or without hypertension in individuals with Hcy Hcy infection. However, Hcy (NO) inhibits the secretion of nitric oxide by endothelial cells, causing platelet aggregation and vasoconstriction [34]. Hcy may also promote the binding of lipoproteins to fibrinogen and contribute to the onset of arteriosclerosis and hypertension. DBP is highly dependent on peripheral resistance, while SBP is mainly dependent on cardiac output. Patients infected with Hylori had significantly higher levels of fibrinogen, a marker of vascular inflammation that inhibits the decline leading to vasoconstriction of NO, and increased peripheral blood vessel tension. 33. As mentioned above, may inhibit the release and promote the binding of Hcy NO lipoproteins to fibrinogen This may explain why. , infection with H pylori , . was associated with DBP but not SBP It was also consistent with characteristics according to which participants infected with H pylori had higher levels of PP. According to researchers at the University of Maine and the University of Arkansas, the use of B vitamins to lower homocysteine levels is an effective means of lowering blood pressure and may be particularly useful in the treatment of drug-resistant hypertension.

UMaine Professor Emeritus of Psychology Merrill Elias, who is also an Honorary Collaborating Professor in the Graduate School of Biomedical Sciences and Engineering, collaborated with Dr. Craig Brown, associate professor of ophthalmology at the University of Arkansas, to publish a peer-reviewed editorial in the American Journal of Hypertension on the treatment of drug-resistant hypertension by lowering homocysteine levels with B vitamins.Homocysteine is an intermediate compound involved in the regulation of vitamin levels. Elevated homocysteine levels are the result of genetic mutations or an insufficient supply of vitamins B6, B12, folic acid, and riboflavin (B2). High homocysteine levels are associated with impaired nitrous oxide synthesis, which is associated with constriction of small vessels and is a risk factor for hypertension, cardiovascular disease, stroke, and neurological diseases. Decrease in levels Homocysteine is relatively inexpensive because it is achieved through vitamin supplements. While recent literature supports the efficacy and safety of homocysteine reduction in the treatment of hypertension, the validity of this generalization has been questioned, sparking a controversy that lasted more than 15 years and, according to the researchers, slowed the use of homocysteine lowering as a treatment

for hypertension. Elias and Brown reviewed the literature on both sides of the conflict and concluded that early criticisms of lowering homocysteine levels were premature and that supplementation with adequate amounts of vitamins B2 (riboflavin), B6, folic acid, and B12 can safely lower blood pressure by as much as 6–13 mmHg [35].

The updated reference value for normal homocysteine levels is ≤10 μmol/L. However, many laboratories define normal homocysteine levels at 11.4 μmol/L. Elias and Brown argue that there is a need to update laboratory values for normal homocysteine levels and determine whether risk-protective values should be even lower.

Vitamin treatment is a potentially important adjunct to the medical treatment of drug-resistant hypertension, but therapy should be led by a physician or a qualified health care provider, the researchers said.

Approximately 12.8% of the world's population suffers from drug-resistant hypertension, defined as the inability to achieve a target blood pressure level of 140/90 mmHg with the use of three classes of antihypertensive drugs. The new definition of hypertension - 130/80 mmHg - makes it even more difficult to achieve successful treatment.Some research has confirmed that H. pylori, which directly affects vitamin D metabolism, may be an alternative explanation for the causal relationship between H. pylori and hypertension. It has been confirmed that vitamin D can regulate the renin-angiotensin-aldosterone system (RAAS), one of the main hormonal mechanisms for blood pressure regulation [36] Gastritis associated with H. pylori may interfere with the absorption of many trace minerals, and in patients with H. pylori-positive vitamin D levels had lower levels [37]. Shafrir et al. It has also been shown that people without H. pylori infection can effectively absorb vitamin D from their diet, and it can be hypothesized that H. pylori may contribute to the development of hypertension due to its effect on vitamin D metabolism in vivo.

Conclusion

We have attempted to investigate the association between H. pylori and AH based on the analysis of the results of the scientific study published above. The presence of information in various literature that refutes each other and has not yet been fully clarified indicates the need for a broad study of this front and requires scientific research.

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