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THE RELATIONSHIP BETWEEN HELICOBACTER PYLORI AND ARTERIAL HYPERTENSION IS ANALYSIS

Boboyorova D.N., Jarilkasinova G.J., Adizova D.R.
Bukhara State Medical Institute. Uzbekistan

Abstract. The literature review analyzes research devoted to the study of the role of Helicobacter pylori in the development of AH in recent years, using data from the scientific literature, data aimed at solving the mutual pathogenetic relationship between the two problems are important. The result of several checks is H. pylori people with pylori show a higher risk of developing arterial hypertension than people who are not infected. H. pyloriri it is revealed what pathological mechanisms pylori affects the body. H. pylori the basis of mutual pathogenetic dependence in the development of pylori and AH is data on the occurrence of endothelial dysfunction, which is accompanied by an increase in the amount of inflammatory cytokines, homocysteinemia, a decrease in vitamin D.

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

Arterial hypertension is an independently developing, chronic disease, the main manifestation of which is arterial hypertension syndrome, which does not depend on other factors. Despite the development of modern medicine of our 21st century, arterial hypertension (ah) currently remains a medical and social problem due to the prevalence of the disease and the severity of complications. This is one of the reasons that we can cite as an important criterion for the prevalence of this disease is the lack of blood pressure control even with drug therapy in a large part of patients, which in turn is the most important risk factor for a number of severe complications, namely myocardial infarction (MI) and cerebral stroke, mainly recording a high. According to forecasts, by 2025, the number of AH patients in the world will increase by 15-20 percent, reaching almost 1.5 billion [1].

Recently, the number of people with hypertension and the dependence of AH on the age of the patient has also been growing from year to year. For a long time, hypertension is considered a multifactorial disease. The spread of hypertension in developing countries is seen as a result of progress and changes in the way of life.

According to the scientist Jannis Kountouras notes, smoking, genetics, in addition to diet, among other factors, H. pylori has been seen in recent years as a potential risk factor for the development of arterial hypertension. Nevertheless, traditional risk factors are not sufficient to explain the increasing rates of hypertension-which, in addition, attempts to control the problem suggest that traditional measures may never be adequate or decisive [2].

Relying on statistics, it can be said that the prevalence of Helicobacter pylori infection is forming a high indicator among all layers of the population. According to

the literature, more than half of people around the world are infected with this bacterium. In Africa, Mexico, South and Central America, the prevalence of this infection reaches 70-90% among adults as a whole. There are a number of scientific studies in the world dedicated to the study of various options for the manifestation of helicobacteriosis. Today H. pylori high levels of infection of the pylori-infected population make it the greatest latent threat to human health. H. pylori to this day was found in the human stomach and for a long time was considered exclusively associated with gastrointestinal diseases.

Currently, many studies have shown that H. pylori has been studied to be etiologically associated with many diseases in addition to gastrointestinal disorders [3].

Helicobacter pylori. H. pylori, a spiral gram-negative microaerophilic Rod. Its morphology is heterogeneous because it can have a helicoidal Helix or twisted shape, a 2-6-notch bacterium that naturally colonizes the epithelium of the human stomach. Its dimensions are 0.5 mm to 1.0 mm in diameter and 2.5 mm to 5.0 mm in length. It is characterized by the production of urease, which creates a microenvironment with a pH above the pH of the gastric mucosa through the production of ammonia, which allows it to survive. H. pylori migrates to the colon and it continues to produce ammonia, which in turn is toxic it leads to the accumulation of a large amount of ammonia, and this can lead to a large number of damage to the large intestine and strong rectal spasm.

Helicobacter pylori is somewhat difficult to grow because it requires a longer incubation period (5 days instead of 24 hours) than most bacteria, and enriched nutrients must be used.

New research on the continuation of the study of such a topical topic can be said based on the results. Many pieces of evidence began to appear that clearly show a causal relationship between Pylori and extragastral diseases, which H. pylori has been found not only to negatively affect the gastroentistinal tract, but to be inextricably linked with other extragastral diseases including metabolic disorders especially cases of psychiatric stroke [4], complications after gynecological diseases[5], cases of severe vomiting and even preeclampsia [6,7].It is worth noting that, during the study of the work of the author Izida, we can see that, Helicobacter pylori undoubtedly mentioned during his scientific research that the nose and throat ears are caused by Tumor Diseases of good quality and even of poor quality[8], laryngeal carcinoma and lung cancer [9], hematological diseases[10], idiopathic thrombocytopenic purpura caused by iron deficiency anemia ITP [11].

Further results in the study of scientific research show that H. pylori has been confirmed to be an important risk factor for the development of hypertension. Chinese scientist Xiong X. and Yue L.Lar noted that H. in addition, people with pylori have a 13.4 percent higher risk of developing hypertension than people who

are not infected, H.pylori eradication of can serve as a new way to prevent and treat hypertension [12,13].

According to the results of scientific work, H.plory is more likely than patients who have not been found to have H. significantly higher blood pressure was found in patients with arterial hypertension where pylori was found. In addition, H. pylori elimination to helps improve arterial hypertension [14,15].

H.Pylori in the scientific literature, infection has been cited as causing assoctive disorders such as metabolic syndrome, and atherosclerotic cardiovascular development. Changes in cholesterol levels in the blood are the main risk factors for cardiovascular disease and metabolic syndrome due to increased levels of low - density lipoprotein - LDH and decreased levels of high-density lipoprotein-Luce. H.pylori infections LDL increased cholesterol is considered the most important risk factor for atherosclerosis and H. the eradication of pylori plays a positive role for the Prevention of atherosclerosis [16].

Pathogenetic mechanisms. Based on existing scientific research, various mechanisms have been proposed to explain the correlation of infection.

The effects of H.pylori on the body can be seen in the following pathogenetic factors:

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

2) molecular mimicry between bacterial antigens and components of macroorganism tissue, their autoimmune damage [18];

3) interaction with fat cells, followed by secretion of biologically active substances acting on vessels, bronchi and other internal organs;

4) development of allergic reactions;

5) Toxic Products, a decrease in the barrier function of the intestine, which leads to the entry of allergens into the blood;

6) absorption of macro - and microelements, in particular iron, for their vital processes, and therefore [19];

Based on laboratory and experimental data, the hypothesis was put forward that inflammation plays a key role in atherogenesis and acute thrombosis. From an epidemiological point of view, confirmation of this hypothesis comes from a number of promising cohort studies showing that inflammatory parameters (such as fibrinogen, C-reactive protein, and serum amyloid a), cell adhesion molecules [such as intercellular adhesion molecule (ICAM)].) Patients with H.pylori infection have elevated inflammatory adgesia molecules such as C-reactive protein (CRP) and intracellular adgesia molecule-1 (ICAM-1), suggesting a link between infection and endothelial dysfunction [20] and cytokines (e.g. interleukin-6) are all initially elevated in patients at risk for future coronary occlusion. In addition, data from randomized clinical studies suggest that the effectiveness of common prophylaxis agents such as aspirin and the inhibitors of hydroxy-methylglutaryl (HMG) CoA reductase may be due in part to interactions with the inflammatory system.

Inflammation in the development of atherosclerosis et There is increasing evidence that inflammation plays an etiopathogenetic role in the development of atherosclerosis and that some signs of inflammation are associated with a greater risk of developing CAD. Markers such as C reactive protein (CRP), white blood cell levels in the blood, presence of plasma fibrinogen or heat shock protein (BTS) worsen CAD prognosis [21] and inflammation can contribute to the development of hypertension, which causes endothelial dysfunction and causes oxidative stress. Minyeko et al. it has been suggested that H. pylori infection can lead to activation of the inflammatory cytokine cascade and release vasoactive substances from the site of infection [22]. People with H.pylori infection have been observed to experience significant increases in levels of various inflammatory cytokines, including IL-1beta, IL-2, IL-6, and Tnf-alpha. H. Pylori has a low-level chronic inflammatory response that triggers the atherogenic process through changes in some risk factors for cardiovascular diseases such as blood clotting factors and lipids, releasing fibrinogen, reactive protein C, Tnf-Ai interleukin 6 (IL-6). an increase in the number of white blood cells in the blood, which can cause prothrombotic status [23]. In Adults, H.pylori induces 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 cellular and brachial in nature. The specific reaction of the cell is characterized by T-helper activation of lymphocytes, which cause an increase in the release of cytokines, especially il-1, IL-6, IL-8, Tnf-Ai interferon. The ability to excite cytokines differs in stamm ovns. H.Pylori while, CAga+ strains have been observed to secrete the strongest and induce cytokine diversity, on the other hand, soluble extracts of H.Pylori have also been observed to help build up plaque in the microcirculation of the gastric mucosa [24]. In addition, endothelial cell dysfunction associated with H.plylor, Mets activates mediators of various types [25, 26].

Based on scientific research, it can be said that the mechanism of endothelial dysfunction is the pathogenesis of heart syndrome. As a result of inflammatory and proliferative changes by pylori, the structural and as a result of functional disorders, it leads to changes in the elastic properties of blood vessels, anti-inflammatory cytokines, cell adhesion molecules, growth factors and acute phase proteins, which caused mediators such as active formation [27]. Indeed, H. pylori increases the level of inflammatory mediators such as the MetS-associated factor of tumor necrosis (TNF)-a, interleukin (IL)-1, IL-6, IL-8, interferon (IFN) - γ, fibrinogen, thrombin, intercellular adhesion molecule, and blood stem cell adhesion molecule; it is associated with MetS inflammatory mediators that cause direct or indirect damage to the walls of blood vessels and thus cause atherosclerosis [28]. H. pylori mediated-inflammation has been associated with atherosclerosis, and the aforementioned inflammatory mediators have been involved in the pathophysiology of arterial hypertension associated with MetS H. Pylori infection causes an increase in

cholesterol and triglycerides when Lewis lowers cholesterol levels and promotes the development of dyslipidemia, a known risk factor for cardiovascular disease.

Changes in lipids in the blood H. pylori infection leads to an increase in cholesterol and triglycerides, promotes the development of dyslipidemia with a decrease in the level of Luce, which is known to be a cardiovascular risk factor. A number of authors assume that the formation of oxidizing agents is also important, in patients with H.plylori, a decrease in antioxidants has been observed, which can lead to the activation of lipid peroxidation and therefore the development of atherogenesis, since the oxidation of low-density lipoprotein (LDL) is the first of the main stages of the atherogenic process. Another theory of cross-reactivity with heat shock protein antibodies (BTS) is anti-BTS antibodies with cross-reactivity. H. pylori has been found to produce BTSH - 60 as a sufficiently novel cardiovascular risk factor, as increased homocysteine levels have been observed to be associated with increased risk of cardiovascular disease. In this context, patients with chronic gastritis usually have reduced absorption of vitamin B12 and folic acid due to Helicobacter infection, thus causing secondary hypergomocysteinemia [29].

To date, there has been a growing interest in homocystei, a "new" risk factor for scientific isolators in addition to traditional risk factors. Homocysteine is an intermediate product of the metabolism of the amino acids methionine and cysteine proteinogen. The relationship between H.pylori infection and hyperhomocysteinemia is one of the ways in which this organism can be associated with the development of coronary disease. Studies have shown that there is a strong relationship between hyperhomocysteinemia and insufficient vitamin intake, as well as the plasma concentration of vitamins, especially vitamin B6, vitamin B12 and folic acid levels. In this regard, patients with chronic gastritis, usually caused by Helicobacter pylori infection, experience a decrease in the absorption of vitamin B12 and folic acid, which causes secondary hyperhomocysteinemia [30,31].

Numerous retrospective and prospective studies have consistently found independent correlations between moderate hyperhomocysteinemia and cardiovascular disease or all-cause death. Starting with a concentration of homocysteine in approximately 10 mkmol/l blood plasma, increased risk occurs depending on the linear dose-effect without certain threshold levels. Hyperhomocysteinemia is responsible for about 10% of the total risk as an independent risk factor for cardiovascular disease. Increased blood plasma homocysteine levels (>12 mkmol/l; moderate hypergomocysteinemia) is cytotoxic and occurs in 5-10% of the general population and up to 40% of vascular patients. Hypergomocysteinemia is associated with changes in vascular morphology, loss of antithrombotic function of the endothelium, and induction of the procoagulant environment. The most popular forms of injury are caused by homocysteine-mediated oxidative stress. A large number of agents, drugs, diseases and lifestyle factors, especially cofactors and direct or indirect antagonists of enzymatic activity, affect

homocysteine metabolism. Folate deficiency is the most common cause of hyperhomocysteinemia. Adequate intake of at least 400 mcg of folic acid per day is difficult to maintain, even with a balanced diet, and high risk groups often fail to meet the need for folic acid. Based on the available evidence, there is an increasing need to diagnose and treat high levels of homocysteine in high-risk people in general and in patients with vascular diseases in particular. Subjects of both populations must first undergo a basic analysis for homocysteine. With the exception of cases where the manifestations are already present, if any, the intervention should be based on the severity of hypergomocysteinemia. As recommended by other working groups and consensus groups, we recommend a target level of homocysteine in the blood plasma of <10 mkmol/L. Based on various design models, a decrease in plasma homocysteine concentration theoretically prevents up to 25% of cardiovascular complications. Food supplements are cheap, potentially effective and free of side effects and therefore have a very beneficial benefit-risk ratio. The results of current randomized controlled intervention studies are available before screening for hypergomocysteinemia and recommending treatment in the overall healthy population must be [32]. Hypergomocysteinemia is a novel cardiovascular risk factor, as it has been observed that increased homocysteine levels are associated with increased cardiovascular risk. In this context, patients with chronic gastritis (usually caused by H.pylori infection), which can lead to decreased absorption of B12vitamine [33].

H.pylori infection, thinning the secretion of nitric oxide by endothelial cells, which in turn causes platelet aggregation and vasoconstriction which leads to damage to endothelial cells and weakening of vasodilation factors and protective effects derived from endothelial cells. H.pylori also promotes the linking of lipoproteins to fibrinogen, promotes the appearance of arteriosclerosis and hypertension. NO leads to vasoconstriction and increases the tension of peripheral blood vessels [34].

According to researchers the South US 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 can be especially useful in the treatment of drug-resistant hypertension.

Umaine psychology professor Merrill Elias has also published a peer-reviewed Editorial on drug-resistant treatment of hypertension by lowering homocysteine levels with B vitamins in the American Journal of Hypertension in collaboration with Dr. Craig Brown, professor of honorary cooperation at the Higher School of Biomedical Sciences and engineering, associate professor of the Department at the University of Arkansas. Homocysteine is an intermediate involved in regulating vitamin levels. Increased homocysteine levels are the result of genetic mutations or insufficient supply of vitamins B6, B12, folic acid and riboflavin (B2). High levels of homocysteine are associated with impaired nitric oxide synthesis, which is small it is associated with narrowing of the vessels and is a risk factor for hypertension,

cardiovascular disease, stroke and neurological diseases. Lowering homocysteine levels is relatively inexpensive, as it can be used by taking vitamin supplements. So while recent literature has confirmed the efficacy and safety of lowering homocysteine levels in the treatment of hypertension, the validity of this generalization has been questioned, which has been the subject of controversy for over 15 years, and the researchers claim has slowed the use of lowering homocysteine levels as a treatment for hypertension.

Elias and Brown, analyzing the literature on both sides of the conflict, concluded that initial criticisms of lowering homocysteine levels were premature and that sufficient amounts of vitamin B2 (riboflavin), B6, folic acid and B12 supplements could safely lower blood pressure to 6-13 mm HG [35].

The updated control value for normal homocysteine levels is ≤10 mkmol/L.However, many laboratories determine normal homocysteine levels at 11.4 mkmol/L. Elias and Brown argue that it is necessary to update laboratory indicators of normal homocysteine levels and determine whether the risk-protecting values should be even lower.

The introduction of vitamin injections into vitamin treatment standards is a potentially important supplement for the treatment of arterial hypertension, but such therapy should be guided by a doctor or a qualified health care provider, the researchers say.

About 12.8 percent of the world's population suffers from drug-resistant hypertension, which is defined as an inability to reach the target level of blood pressure of 140/90 mmHg.When using three classes of antihypertensive drugs. Re-detection of Arterial hypertension - 130/80 mmHg-makes it even more difficult to achieve successful treatment.

Based on some studies, which directly affects vitamin D metabolism H.pylory an alternative explanation may be for the causal relationship between pylori and hypertension. It has been confirmed that vitamin D may regulate the renin-angiotensin-aldosterone system (RAAS), one of the main hormonal mechanisms for regulating blood pressure [36]. H. pylori-related gastritis can be an inhibitor of the absorption of many trace elements, and patients with H.pylori-positive vitamin D levels have low levels [37]. Shafrir et al. also say, H. pylori has been shown to be able to effectively absorb vitamin D from a diet without infection, it can be assumed that H.pylori can contribute to the development of hypertension in vivo due to its effect on vitamin D metabolism [38].

Conclusion. We have tried to study the link between H.pylori and AH from the analysis of the results of scientific research published in above. The presence of information in various literature that denies one and has not yet been fully clarified indicates the need for a wide study of the front and assumes the conduct of scientific research.

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