AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 01, Issue 09, 2023 ISSN (E): 2993-2149

The Pathogenetic Analysis Relationship between Helicobacter Pylori and Arterial Hypertension

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Abstract: a literature review analyzes research in recent years devoted to the study of the role of Helicobacter pylori in the development of AG, using scientific literature data, data aimed at solving the mutual pathogenetic relationship between the two problems is important. The result of several investigations was H. people with pylori have a higher risk of developing arterial hypertension than people who are not infected. H. pylori has been found to be pathological mechanisms by which affects the body. H. pylori the basis of mutual pathogenetic dependence in the development AG is information 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, Helicobacter pylori, risk factors, endothelial damage, pathogenetic factors.

Currently, AG remains one of the pressing problems of medicine around the world. The pathogenesis of Ms-component hypertension involves primarily vascular endothelium degradation, which in turn causes a number of changes-namely,endothelial dysfunction, sympathetic nerve degradation, and renin-angiotensin-aldosterone system aberrant activation. Risk factors for hypertension have been shown to be associated with HP infection [1].HP infection increased the risk of hypertension by 32%.

In addition, the scientific literature reported that the average arterial pressure of patients with HP infection increased by 0.723 mmHg compared to patients without HP infection. Inflammatory response and inflammation caused by HP infection factors can damage the vascular endothelium and dysfunctional vascular endothelium serves as an etiological factor for hypertension. In addition, HP infection causes autoimmune reactions by affecting the immune system, which can also lead to hypertension [2]. Most AG li patients have been diagnosed with HP colonization in the arterial wall [3]. Based on data from the scientific literature, it can be said that damage to the vessel wall caused directly by HP infection disrupts the elasticity of the vessels and, as a result of its negative impact on hemodynamics, leads to hypertension. HP infection is closely related to dyslipidemia. Atherosclerosis in the outcome of long-term dyslipidemia is also an important factor for AG, which affects the appearance and development of hypertension. Scientific research studied shows that hypertension and HP infection do not have a double correlation, as observed in other diseases. One of the exceptions is Diseases of the digestive system can be an injury associated with hypertension, especially in the stomach, which increases the susceptibility to HP infection. However, this phenomenon has rarely been observed. H. the relationship between pylori and hypertension is one of the world's clinical evidence of arterial blood pressure, which, according to most hypotheses, is associated with changes in inflammation and salt intake

[4]. Chronic inflammation can exacerbate hypertension after causing endothelial dysfunction, oxidative stress. According to the results of scientific research of a number of scientists H. pylori infection to the activation of the cascade of inflammatory cytokines is caused by vasoactive substances H. pylori vit D metabolism can be directly influenced by a mixed alternative H. for the causal relationship between pylori and hypertension. The Renin-Angiotensin-aldosterone system (RAAS) has been confirmed to be Vit D, a major hormonal mechanism in blood pressure regulation [5]. H. pylori-related gastritis with multiple microelements and H. absorption of pylori-positive subjects had a low level of vit D. Shafrir et al, also known as H. individuals without pylori infection have proven that the absorption of Vit D in their diet can be effective [6]. It Is H. it can be said that pylori was able to promote the development of hypertension with its effect on the metabolism of intracellular live vitamin D [7]. H.pylori is an inflammatory mechanism of endothelial dysfunction. C-reactive protein (CRP) and intracellular adhesion molecule marker like adhesion molecule-1 (ICAM-1) are elevated in patients. H. pylori infection causes secondary dysfunction for homocysteine elevated in the middle of endothelial dysfunction. H. pylori infection also causes vitamin B12 and folic acid to be malabsorbed which in turn increases serum homocysteine levels.

H. it is important to know how pylori infection leads to endothelial dysfunction. The main pathogenic process of cardiovascular disease is atherosclerosis, a mechanism whereby hypertension and other important risk factors such as age, male sex, cigarette smoking, obesity, dyslipidemia, diabetes mellitus, high salt intake,and familial predisposition [8].

In addition to these traditional risk factors, new potential cardiovascular risk factors are increasingly being recognized, with Helicobacter pylori (H. pylori) bacterium is one of the most studied pathogens [9]. Helicobacter pylori is still responsible for common chronic bacterial infections, and its importance in promoting the development of atheroma is associated with chronic low-level inflammation triggered by the release of anti-inflammatory molecules, increased fibrinogen, C reagent.Protein, triglycerides, and low-density lipoproteins participate in the atherosclerotic process and develop a prothrombotic State [10]. H. pylori infection can cause lifelong inflammation [11] and persistent low-grade inflammation has played an important role in accelerating the development of hypertension [12]. Hypertension disease and H. possible connections between pylori infection include activation of the cytokine cascade with release of vasoactive substances or molecular h from the primary site of infection. inflammation expressed by mimicry endothelial cells and smooth muscle cells between Caga antigens of pylori and some peptides has been said to be the main pathogenetic factors that can cleave cardiovascular disease, where pathogenesis and inflammatory symptoms of atherosclerosis are reported to be associated with risk of atherosclerosis. They show important clinical signs, a decrease in blood pressure indicators, in particular in hypertensive patients in diastolic blood, H. there has been a decrease in pressure values after the elimination of pylori. They are possible connections between hypertensive disease and H. pylori infection may involve cytokine activation, a cascade associated with the release of primary vasoactive substances at the site of infection, or molecular mimicry between Caga antigens H. some peptides and smooth muscle, expressed by pylori and endothelial cells, have altered state. H.pylori with hypertension. the importance of this association of infection H.pylori it is emphasized by the possibility of effective intervention against infection, since this bacterium is not easily destroyed with the help of the body,therefore, a simple and reliable drug regimen is used. H.pylori infection has been noted as an important cause in the appearance of chronic gastrodinestinal ulcers, but an epidemiologist - ical study based on the discovery of bacteria over the past twenty years has been published in H. a high prevalence of pylori infection has been shown to be involved in the pathogenesis of heart disease [13]. In recent years, the theory has been proposed that the bacterium is one of the potential mechanisms that can directly and indirectly affect heart disease. H. inflammatory and immunological events triggered by pylori infection are the main causes of heart disease [14].H. pylori is a unipolar and microaerophilic bacterium. It contains five main outer membrane protein families. The largest family is putative adhesion, the other four families include purines, iron,

transporters, xivchns-associated, proteins, and proteins of unknown function. As it is a Gram-negative bacterium, its outer membrane is composed of phospholipids and lipopolysaccharides, which also contain cholesterol glucosides, which are found in several other bacteria. The high mobility of bacteria is due to the presence of two to six lophotrichous chicks. The filaments of these sheathed chicks consist of two copolymerized chicks, flagellin A and flagellin B, which are about 0.5–1 mm in diameter and 2.5–5.0 mm in length. Variability between strains H. pylori is associated with the presence of several bacterial genomes. It is one of the most genetically diverse species of bacteria, as it produces many toxins. Due to high viral factors, it is associated with many diseases. These virulent factors include the production of the vacuolating cytotoxin gene a (Vac A) and Cag A about 50% H. the production of pylori strains is associated with this heart disease. H. pylori it causes toxigenic protein cells and severe inflammation, and interleukins (ILS; IL-1, IL-2, IL-6, IL-8, and IL-12), interferon-gamma, tumor necrosis factor, t and B lymphocytes, and phagocytic cells, increase the likelihood of heart disease [15].H. pylori infection significantly increases the risk of cardiovascular disease, such as atherosclerosis, HTN, CHD, cerebrovascular disease, and peripheral arterial diseases, especially in young patients (< 65 years of age). H. pylori infection significantly reduces the production of ROS in the following mechanisms and the formation of no, which disrupts the endothelial function of blood vessels, causing disruption of several mechanisms, including an increase in the oxidation process, inflammation, which calls the vasoconstrictor. According to the results of scientific research by Migneco et al. pylori infection can lead to activation of the inflammatory cytokine Cascade, and vasoactive substance production from the site of infection increases-this is the basis for an increase in arterial hypertension [16].These inflammatory cytokines can increase insulin resistance [17]. In addition, H. high fibrinogen levels in people with pylori infections, can increase the level of cardiovascular inflammatory biomarkers which in turn causes a decrease in nitric acid (no), resulting in vasoconstriction and increased peripheral vascular tension [18]. H. endothelial dysfunction associated with pylori infection, if the infection can be eliminated in a timely manner both in the animal model and in human subjects, this process is reversible. The collected data shows that H. pylori infection is an additional risk factor for endothelial dysfunction and vascular disorders. The population of young males can be reached once a year by H. examination of pylori infection and, accordingly, treatment can be an effective approach to early prevention of vascular diseases, especially H. it was also useful for the Prevention of early atherosclerosis associated with pylori infection [19]. In addition, high salt intake and vitamin D (Vit D) metabolisms are also found in H. closely associated with pylori, H. pylori infection causes hypertension [20]. Xiong and unig disciples H. they found that eradication of pylorini infection from the body positively affects arterial hypertension, in addition to proving that hypertension is effective for sex, age, and family history in their own scientific work [21]. The cause of chronic infection is H. pylori infection, especially CagA-positive strains, can lead to the K'tarisation of permanently inflammatory metabolites, which are cytokines interleukin-1 (IL-1), interleukin-1 (IL-6), and tumor-necrotic factor (Tnf-1), which affects vascular activity and leads to endothelial dysfunction [22].Also chronic H. pylori infection can cause a decrease in the absorption of vitamin B12 and folate acidacinig, resulting in hyperhomocysteinemia, which promotes the production of free intracellular oxygen radicals and the breakdown of nitric oxide,resulting in this pathogenetic process leading to endothelial cell dysfunction [23].

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