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The Role of Chronic Helicobacter Pylori Infection as a Risk Factor for Cardiovascular Complications in Arterial Hypertension

Boboyorova Dildora Nabijonovna

Bukhara Medical University, Bukhara, Uzbekistan

Abstract This article analyzes current scientific data on the role of chronic Helicobacter pylori infection in the development of cardiovascular complications in patients with arterial hypertension. It presents the mechanisms by which the bacteria influences the vascular system, the results of clinical studies, and the potential impact of infection eradication on patient prognosis. Particular attention is paid to the systemic inflammatory and metabolic effects associated with H. pylori infection.

Keywords Helicobacter pylori, Arterial hypertension, Inflammation, Endothelial dysfunction, Cardiovascular complications

1. Introduction

Arterial hypertension (HTN) remains one of the most common chronic noncommunicable diseases and a leading cause of death worldwide. According to WHO (2023), approximately 1.3 billion people suffer from hypertension, and only half of them have adequate blood pressure control. Against this backdrop, the search for new risk factors is particularly urgent, and chronic infections, including Helicobacter pylori, are increasingly being considered. Helicobacter pylori infection is one of the most common infections worldwide. Its prevalence reaches 70% in developing countries and 40–50% in industrialized countries. Several studies have shown that H. pylori not only causes gastritis and peptic ulcers but also has systemic effects through chronic inflammation [1]. Chronic Helicobacter pylori infection is considered an important modifiable risk factor for the development of arterial hypertension due to its effects on inflammation, metabolism, vitamin status, and vascular regulation.

Epidemiological studies have shown that the risk of developing hypertension is 30–35% higher in H. pylori-infected individuals than in uninfected individuals. These data are supported by large meta-analyses including tens of thousands of patients [2]. The main pathogenetic mechanisms include systemic inflammation, endothelial dysfunction, immune responses, and metabolic disorders.

* Corresponding author:
dildora.boboyorova92@icloud.com (Boboyorova Dildora Nabijonovna)
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According to modern research, the presence of infection is associated with an increased risk of developing hypertension. Infected patients are more likely to have increased vascular stiffness, changes in the diurnal blood pressure profile, and increased sympathetic activity [3]. Chronic inflammation increases the level of C-reactive protein and cytokines (IL-6, TNF-α), activates oxidative stress processes, and reduces nitric oxide production, leading to vasoconstriction and increased blood pressure [1]. Particular attention is paid to the role of H. pylori strains containing the virulence genes cagA and vacA, which enhance the inflammatory response and endothelial damage [4]. Furthermore, infection affects lipid metabolism and homocysteine levels, increasing blood atherogenicity. Impaired vitamin B12 absorption in atrophic gastritis caused by H. pylori was demonstrated in studies by Figura and Kaptan [5]. Increased homocysteine levels in vitamin B12 deficiency are described in detail in the works of Zhang et al. B12 deficiency disrupts methionine metabolism, leading to homocysteine accumulation. Hyperhomocysteinemia causes endothelial dysfunction, decreased NO synthesis, increased oxidative stress, and contributes to elevated blood pressure. In patients with H. pylori infection, this mechanism is particularly pronounced due to impaired vitamin B12 absorption [6]. Blood pressure in adults with a positive H. pylori test was significantly higher than in those with a negative test. Notably, H. pylori infection was associated with elevated diastolic blood pressure (DBP), while no significant associations were found with systolic blood pressure (SBP), pulse pressure (PP), or mean arterial pressure (MAP). Similarly, a cross-sectional study found a positive association between H. pylori infection and DBP, but not with SBP [7].

Thus, Helicobacter pylori can be considered as an independent and modifiable risk factor for cardiovascular diseases.

2. Materials and Methods of Research

The study was conducted at the Republican Specialized Scientific and Practical Medical Center of Cardiology, Bukhara Regional Branch, and the Department of Internal Medicine of the Bukhara State Medical Institute from January 2022 to December 2024. The study included 90 patients aged 60 to 74 years (mean age 66.8 ± 3.7 years) with stage I–II arterial hypertension according to the ESC/ESH classification (2018). All patients were observed in outpatient and inpatient settings, were followed up in the dispensary, and provided written informed consent to participate in the study.

Inclusion criteria:

- stable increase in blood pressure ≥140/90 mm Hg.
- age from 60 to 74 years,
- the diagnosis of essential arterial hypertension has been confirmed,
- ability to comply with the monitoring and treatment regimen.
- voluntary written consent to participate in the study.

Exclusion criteria:

- secondary forms of hypertension (renovascular, endocrine, etc.), acute and chronic infectious diseases in the acute stage,
- oncological pathology,
- severe heart failure grade III–IV according to the NYHA classification.
- cognitive disorders that make it difficult to cooperate with the patient.
- Diabetes mellitus

Depending on the results of the breath test, all patients were divided into three groups:

Group I (main, n=89) – patients with hypertension and laboratory-confirmed chronic Helicobacter pylori infection;

Group II (comparative, n=56) – patients with hypertension without signs of H. pylori infection.

In addition, to compare clinical, laboratory and functional data, a control group (n=30) was formed, consisting of practically healthy people of the same age, without hypertension and H. pylori infection.

3. Research Results

The study involved 89 patients with arterial hypertension. Helicobacter pylori was diagnosed using two methods: a 13C-urease breath test and the determination of specific IgG using an ELISA.

Elevated levels of inflammatory markers such as IgG to H. pylori (0-15 U/ml) 64.29 ± 5.6,C-reactive protein(0-5 mg/l), 15.24 ± 0.4, TNF-α, (0-6 pg/ml)and interleukin-6(0-10.0 pg/ml) 13.2 ± 0.1 V. This group indicates a high activity of the inflammatory process. At the same time, in the group without H. pylori, significantly lower values of the following indicators are noted: IgG -13.1 ± 6.3U/ml, C-reactive protein -4.25 ± 0.1mg/l, TNF-α —4.76 ± 0.68pg/ml and Interleukin-6 -5.98 ± 0.98pg/ml, indicating less pronounced inflammatory activity. This allows inflammation to be considered one of the significant non-traditional factors playing a significant role in the pathogenesis of arterial hypertension. Studies by Zhang J. and Chamarthi B. et al. have shown that inflammatory factors play a significant role in the development of arterial hypertension. The mechanism by which inflammatory factors influence arterial hypertension can be explained as follows: increased levels of C-reactive protein (CRP), TNF-α, and interleukin-6 lead to damage to the vascular endothelium and a decrease in the production of nitric oxide, a substance that dilates blood vessels. At the same time, vasoconstriction increases due to activation of the sympathetic nervous system and increased sensitivity to angiotensin-II. All this causes an increase in vascular resistance and, consequently, blood pressure. In particular, increased levels of C-reactive protein, TNF-α and interleukin-6 are associated with the development and progression of hypertension, confirming the involvement of systemic inflammation in the pathogenesis of this disease [8-9]. Our scientific results also demonstrate a similar trend and confirm the significant role of inflammatory markers in the patients examined.

Table 1. Comparative characteristics of laboratory parameters and inflammatory markers in patients with arterial hypertension depending on H. pylori status

Indicator	H. pylori (+) n=89	H. pylori (–) n=56	Method	R
IgG to H. pylori, (0-15 U/ml)	64.29 ± 5.6	13.1 ± 6.3	IHLA	<0.001
C-reactive protein (0-5 mg/l)	15.24 ± 0.4	4.25 ± 0.1	Diasist	<0.05
IL-6 (0-10.0 pg/ml)	3.2 ± 0.1	5.98 ± 0.98	IFA	<0.05
TNF-α, (0-6 pg/ml)	14.93 ± 5.2	4.76 ± 0.68	IFA	<0.01

Table 2. Comparison of homocysteine and vitamin B12 levels in patients with arterial hypertension depending on H. pylori infection

Indicator	H. pylori (+) n=89	H. pylori (–) n=56	Method	R
Vitamin B12, (200-1100pg/ml)	394.5 ± 35.3	612.35 ± 26.2	IFA	<0.001
Homocysteine (4-17 μmol)	18.67 ±1.7	7.74±0.45	IFA	<0.05

According to the literature, vitamin B12 (cobalamin) plays a key role in homocysteine metabolism, acting as a cofactor for the enzyme methionine synthase, which mediates the remethylation of homocysteine into methionine. Vitamin B12 deficiency inhibits this metabolic pathway, leading to the accumulation of homocysteine in the blood plasma and the development of hyperhomocysteinemia [10]. Helicobacter pylori infection, by inducing chronic inflammation of the gastric mucosa and atrophic changes, contributes to a decrease in the production of intrinsic factor and impaired vitamin B12 absorption. This, in turn, leads to functional cobalamin deficiency and impaired homocysteine remethylation, which leads to increased homocysteinen levels in the blood and potentiates vascular damage [11].

Based on the literature data on the role of homocysteine metabolism disorders, in the present study, homocysteine and vitamin B12 levels were analyzed as one of the key pathogenetic factors of arterial hypertension.

Along with other indicators, vitamin B12 and homocysteine were included in the analysis as one of the key factors in the pathogenesis of arterial hypertension. The level of vitamin B12 (reference values 200-1100 pg/ml) in the main group was 394.5 ± 35.3 pg/ml, while in the comparison group it was statistically significantly higher - 612.35 ± 26.2 pg/ml ($p<0.001$). The level of homocysteine (reference values 4-17 μmol/l) was found to be 18.67 μmol/l in the main group, exceeding the upper limit of the norm and significantly exceeding the same indicator in the comparison group (7.74 ± 0.45 μmol/l).

The study found that patients in the study group exhibited a significant increase in homocysteine levels, which is considered a significant pathogenetic factor in arterial hypertension and is associated with the development of endothelial dysfunction and oxidative stress. Homocysteine concentrations in the study group were 18.67 μmol/L, exceeding reference values (4–17 μmol/L) and significantly higher than those in the comparison group (7.74 ± 0.45 μmol/L; $p < 0.05$). A decrease in vitamin B12 levels, which is involved in homocysteine metabolism and the regulation of vascular homeostasis, was also detected. In the study group, this indicator was 394.5 ± 35.3 pg/ml, which was statistically significantly lower than in the comparison group—612.35 ± 26.2 pg/ml ($p < 0.001$).

The obtained data indicate a possible role for hyperhomocysteinemia and relative vitamin B12 deficiency in the pathogenesis of arterial hypertension, which may contribute to the progression of endothelial dysfunction and vascular disorders. Moreover, the concentration of C-reactive protein (reference values 0–5 mg/L) in the study group reached 15.24 ± 0.4 mg/L, which significantly exceeded the similar indicator in the comparison group (4.25 ± 0.1 mg/L). The obtained results indicate pronounced inflammatory activation, decreased vitamin B12 levels, and increased homocysteine levels in patients in the study group compared to the control group.

The study found that patients infected with Helicobacter pylori had a particularly significant increase in diastolic pressure, consistent with mechanisms of chronic inflammation, endothelial dysfunction, and increased vascular stiffness associated with H. pylori.

Table 3. Blood pressure levels in patients with arterial hypertension depending on the presence of H. pylori

Indicator	H. pylori (+)	H. pylori (–)	R
SBP, mmHg	147.80 ± 20.70	145.20 ± 22.55	> 0.05
DBP, mmHg	98.26 ± 12.44	91.97 ± 12.89	<.005

This table presents the blood pressure readings of patients at the time of presentation. It should be noted that the measurements were performed under conditions of irregular and unsystematic use of antihypertensive medications, without adherence to standard therapeutic regimens. This therapeutic status could have influenced the obtained blood pressure values, which must be taken into account when interpreting the study results. Reference blood pressure values were considered to be values less than 140/90 mmHg, while the optimal level was considered to be values less than 120/80 mmHg, in accordance with international recommendations. (ESC/ESH, Maastricht, WHO) Analysis of blood pressure readings revealed that patients with H. pylori had higher values of both systolic and diastolic blood pressure compared to the group without the infection.

Thus, the level of systolic blood pressure (SBP) in the H. pylori (+) group was 147.80 ± 20.70 mmHg, while in the H. pylori (–) group it was 145.20 ± 22.55 mmHg; however, the differences between the groups were statistically insignificant ($p > 0.05$), despite the observed tendency towards higher values in infected patients. Statistically significant differences were also found for diastolic blood pressure (DBP): in the H. pylori (+) group, this indicator was 98.26 ± 12.44 mmHg, with the differences between the groups being statistically significant ($p < 0.05$), which is significantly higher compared to the group of patients without H. pylori infection. After hospitalization, patients received standard antihypertensive therapy in accordance with current clinical guidelines.

The most significant changes were found in diastolic blood pressure (DBP). This was observed in patients who tested positive for H. pylori infection. Higher DBP values indicate increased peripheral vascular resistance and reflect impaired vasodilation characteristic of endothelial dysfunction in chronic inflammation, including that associated with Helicobacter pylori infection [12]. Chronic inflammation associated with Helicobacter pylori infection leads to impaired vascular function, activation of proinflammatory cytokines (interleukin-6, TNF-α), which leads to decreased bioavailability of nitric oxide (NO), a key vasodilator factor. NO deficiency is accompanied by a predominance of vasoconstrictor effects, including increased endothelin-1 levels, which causes persistent narrowing of resistance arteries and arterioles.

Additionally, inflammation contributes to increased oxidative stress and activation of the renin-angiotensin-aldosterone system, leading to increased vascular tone and vascular remodeling. Taken together, these changes cause

an increase in peripheral resistance, which is clinically manifested by an increase in diastolic blood pressure [13].

4. Discussion

Additionally, it was found that patients with Helicobacter pylori exhibit a significant increase in homocysteine levels, accompanied by decreased vitamin B12 concentrations ($p<0.05–0.001$). Given the key role of cobalamin in homocysteine remethylation, its relative deficiency, likely due to impaired absorption in chronic gastritis associated with H. pylori, contributes to the development of hyperhomocysteinemia. Elevated homocysteine levels, in turn, increase endothelial dysfunction, oxidative stress, and vascular damage, which may be an additional pathogenetic mechanism in the development and progression of arterial hypertension.

The identified changes can be explained by pathophysiological mechanisms associated with chronic inflammation. As previously shown, patients with H. pylori infection exhibit elevated levels of proinflammatory markers (C-reactive protein, TNF-α, interleukin-6), which contribute to the development of endothelial dysfunction. Reduced nitric oxide (NO) bioavailability and increased production of vasoconstrictor factors, such as endothelin-1, lead to a persistent increase in resistance vascular tone.

Thus, the study results confirm that Helicobacter pylori infection can be considered an additional factor contributing to the deterioration of the circadian blood pressure profile, primarily due to an increase in diastolic pressure and a disruption of its nocturnal decline. This is consistent with the concept of the involvement of chronic inflammation, endothelial dysfunction, and metabolic disturbances (including hyperhomocysteinemia and vitamin B12 deficiency) in the pathogenesis of arterial hypertension.

5. Conclusions

Our results show that *H. pylori* Infection is associated with an increase in diastolic blood pressure (DBP), but no significant associations were observed with systolic blood pressure (SBP), pulse pressure (PP), or mean arterial pressure (MAP). Consistent with this, previous cross-sectional studies have demonstrated a positive correlation with DBP but not with SBP. Chronic Helicobacter pylori infection is considered a potential modifiable risk factor that can accelerate the progression of hypertension. Systemic inflammation, oxidative stress, and endothelial dysfunction play a key role in its impact on the vascular system. H. pylori eradication therapy can improve vascular reactivity and reduce the risk of cardiovascular complications, making it an important element of comprehensive treatment for patients with hypertension.

REFERENCES

[1] Kuijper E. J. et al. Helicobacter pylori infection and systemic diseases. // Gut Microbes. 2020; 12(1): 1–15.

[2] Figura N. et al. The role of Helicobacter pylori in extragastric diseases. // Helicobacter. 2019; 24(2): e12-538. (Electronic article, without traditional pages - e-number is used).

[3] Zhang C, Li H, et al. Association between Helicobacter pylori infection and hypertension: a meta-analysis. // Journal of Hypertension. 2022; 40(6): 1042–1050.

[4] Chen Y., Blaser M. Helicobacter pylori and cardiovascular diseases: epidemiological links and potential mechanisms. // Clinical cardiology. 2021; 44(3): P 322–330.

[5] Fang L et al. The relationship between Helicobacter pylori and hypertension: a systematic review. // BMC Cardiovascular Disorders. 2022; 22(1): P 312.

[6] Yue J et al. Helicobacter pylori infection and blood pressure: a comprehensive meta-analysis. // PLoS One. 2023; 18(7): e0289021. (electronic format - number e instead of the number of pages).

[7] Wang C., Hu L., et al. Helicobacter pylori infection and the prevalence of high blood pressure among Chinese adults. // Journal of Human Hypertension. 2018; 32(2): P-158-164.

[8] Chamarthi B., et al. Inflammation and Hypertension: //The Interplay of Interleukin-6 and the Renin–Angiotensin System in Humans. // American Journal of Hypertension, 2011.

[9] Zhang J., et al. Correlations of CRP and IL-6 with complications in hypertensive patients. // Medical Science Monitor, 2019.

[10] Cai X., Li X., et al. Vitamins and Helicobacter pylori: // An Updated Comprehensive Meta-Analysis and Systematic Review. Frontiers in Nutrition. 2022; 8: 781333.

[11] Dierkes J., Ebert M. Helicobacter pylori infection, vitamin B12 and homocysteine. // A review Digestive Diseases. 2003; 21(3): 237-44.

[12] Franceschi F,. et all. // Clinical effects of Helicobacter pylori outside the stomach. // Nature Reviews Gastroenterology Hepatology. 2014; 11(4): P. 234–242.

[13] O'Brien E., Parati G., et al. European Society of Hypertension position paper on ambulatory blood pressure monitoring. // Journal of Hypertension. 2013; 31(9): P. 1731–1768.