THE ROLE OF HELICOBACTER PYLORI IN THE DEVELOPMENT OF HELICOBACTER PYLORI IN PATIENTS WITH CHRONIC HEART FAILURE

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Abstract. This article talks about the role of helicobacter pylori in the development of anemia in patients with chronic heart failure. Chronic heart failure is a disorder of the heart (pump) function with the corresponding symptoms, which consists in the inability of the circulatory system to deliver the amount of blood necessary for the normal functioning of the organs and tissues.

Keywords: medicine, chronic heart failure, patient, helicobacter pylori, anemia, disease, healing.

Helicobacter pylori (H.pylori) infection is one of the most common infections in the world. The infection rate of the world's population exceeds 50% and is especially high in countries with a low socio-economic level of development. *H.pylori* is capable of causing gastroduodenal and extragastroduodenal diseases, in particular metabolic disorders such as obesity, diabetes mellitus and non-alcoholic fatty liver disease.

Recent epidemiological and clinical studies have also shown that the long-term persistence of *H.pylori* is associated with the development and progression of atherosclerosis and as a consequence, the occurrence of cardiovascular diseases. However, the relationship between them is ambiguous and there is no convincing evidence of this correlation in the literature. The lack of consensus may be due to differences in the diagnosis of *H.pylori* and variability in infection genotypes. Given the high prevalence of *H.pylori* and the significant incidence of metabolic and cardiovascular diseases, confirmation of the relationship between them may be of great epidemiological, preventive and clinical importance.

Helicobacter pylori is a common gastric pathogen that causes pathologies such as gastritis, peptic ulcer, adenocarcinoma and low-grade gastric lymphoma. The infection can be asymptomatic or lead to dyspepsia of varying severity.

Diagnosis is based on a urea breath test, fecal antigen testing and analysis of specimens obtained from endoscopic biopsy. Treatment is with a proton pump inhibitor along with two antibiotics.

H.pylori is a gram-negative, spiral-shaped microorganism well adapted to life in an acidic environment. In low and middle-income countries, this microorganism often causes chronic infection and infection usually occurs during childhood.

In the US, infection in children is less common and increases with age: 50% are infected by age 60. The infection is more common in black Americans, as well as Hispanic or Asian Americans.

The microorganism was cultivated from faeces, saliva, contents of the periodontal pocket, which suggests oral or alimentary (fecal-oral) transmission routes. The infection tends to "link" with family members and persons permanently residing in social care institutions. It is assumed that nurses and gastroenterologists are a high-risk group, because bacteria can be transmitted through endoscopes if they are not properly disinfected.

Infection predominantly in the antrum leads to an increase in gastrin production, possibly due to local impairment of somatostatin secretion. As a result, hypersecretion of acid predisposes to prepyloric or duodenal ulcer.

Infection predominantly of the body of the stomach leads to the development of atrophic gastritis and a decrease in acid production, possibly due to an increase in local production of interleukin-1 beta. Patients with predominantly gastric corpus infection are predisposed to gastric ulcers and gastric adenocarcinoma.

In some patients, the infection affects both the antrum and the body of the stomach, leading to different clinical effects. In many cases, *H.pylori* infection does not show any significant clinical effects.

The ammonia produced by *H.pylori* allows the organism to survive in the acidic environment of the stomach and can break down the mucus barrier. Cytotoxins and mucolytic enzymes (eg, protease, lipase) produced by the bacterium *H.pylori* may play a role in gastric mucosal injury and subsequent ulcerogenesis.

Infected individuals are 3 to 6 times more likely to develop stomach cancer. H.pylori is a group 1 (1) carcinogen. The development of adenocarcinoma of the body of the stomach and its antrum, but not cancer of the cardia of the stomach, is associated with H.pylori infection. Other H. pylori-associated tumors are gastric lymphoma and mucosal lymphoid tissue lymphoma (MLT), a monoclonal B-cell tumor.

Urease breath test and fecal antigen determination. Screening of asymptomatic individuals is not appropriate. Tests are carried out in the diagnosis of peptic ulcer or gastritis. Testing after treatment is necessary to confirm the eradication of the microorganism.

Non-invasive tests. Laboratory and outpatient serological tests for H.pylori antibodies have greater than 85% sensitivity and specificity and have previously been considered the non-invasive tests of choice for the initial confirmation of H.pylori infection. However, as the prevalence of infection has declined, the false positive rate in serological tests has increased significantly, making these tests too unreliable in most countries and regions. As a result, urease breath testing and fecal antigen testing are preferred for primary diagnosis. Qualitative methods give a positive result up to 3 years after successful treatment, and since quantitative antibody levels do not change significantly within 6-12 months after treatment, serological tests are usually not used to evaluate the effectiveness of treatment.

The urea breath test uses oral administration of a specific dose of 13C or 14C labeled urea. In an infected patient, the microorganism metabolizes urea, which releases labeled CO2, which

is exhaled and can be quantified in an exhaled breath sample obtained 20–30 minutes after ingestion of urea. Sensitivity and specificity are > 95%. Urea breath tests have been well studied to confirm the eradication of the microorganism after treatment. False-negative results are possible with recent use of antibiotics or concomitant use of proton pump inhibitors, so repeat tests should be performed no earlier than 4 weeks or more after antibiotic therapy and one week after treatment with proton pump inhibitors. H2 blockers do not affect the test.

The fecal antigen test has similar sensitivity and specificity to the urea breath test, especially at initial diagnosis; a technique for an appropriate stool test that can be performed on an outpatient basis is under development.

Invasive methods. Endoscopy is used to obtain biopsy samples of the mucosa for rapid urease test (RUT) or histological staining. Bacterial seeding has limitations in use due to the difficulty of obtaining a culture of a "fastidious" microorganism. Endoscopy is not the only recommended means of diagnosing *H.pylori* infection; non-invasive tests are preferred unless endoscopy is indicated for other reasons.

RUT, in which the presence of bacterial urease in the biopsy specimen causes a color change in the special medium, is the diagnostic method of choice for the tissue sample. Histological staining of biopsy material should be performed for patients with a negative RUT result, but clinical signs suggestive of *H.pylori* infection, recent antibiotic use, or treatment with proton pump inhibitors. BUT and histological staining have a sensitivity and specificity of over 90%.

Treatment of H.pylori infection.

Antibiotics (various regimens) in combination with a proton pump inhibitor

Eradication can be confirmed by urease breath test, fecal antigen testing, or esophagogastroduodenoscopy.

(See also American College of Gastroenterology's 2017 guidelines for the treatment of Helicobacter pylori infection.)

Patients with complications (eg, ulcers, cancer) require eradication of the microorganism. *H.pylori* eradication can even eliminate LTSO lymphoma (but not other tumors associated with infection) in some cases. The treatment of asymptomatic infection is controversial, but recognition of the role of *H.pylori* in cancer development has led to the development of treatment recommendations. Vaccines, both prophylactic and therapeutic (i.e. supplementing the treatment of an infected patient) are under development.

Eradication of *H.pylori* requires multicomponent therapy, usually with antibiotics in combination with drugs that suppress gastric acid secretion (1). Proton pump inhibitors inhibit *H.pylori* infection and the increase in gastric pH that accompanies their use can increase tissue concentration and antimicrobial efficacy, creating a hostile environment for *H.pylori.*

Quadrotherapy is the best initial therapy in areas where clarithromycin resistance is >15%. In quadruple therapy, the following oral medications are given for 14 days (2):

Proton pump inhibitors (lansoprazole 30 mg twice daily, omeprazole 20 mg twice daily, pantoprazole 40 mg twice daily, rabeprazole 20 mg twice daily, or esomeprazole 40 mg once daily)

Bismuth subsalicylate (524 mg 4 times a day)

Metronidazole 250 mg 4 times a day

Tetracycline 500 mg 4 times a day

Triple therapy was the most commonly prescribed treatment regimen for H.pylori infection. The following oral medicines are used for 10 to 14 days:

Proton pump inhibitors (lansoprazole 30 mg twice daily, omeprazole 20 mg twice daily, pantoprazole 40 mg twice daily, rabeprazole 20 mg twice daily, or esomeprazole 40 mg once daily)

Amoxicillin (1 g 2 times a day) or metronidazole 250 mg 4 times a day

Clarithromycin (500 mg twice daily)

However, in many regions of the world, the rate of development of resistance to clarithromycin is increasing and failure of triple therapy is more likely. Therefore, this regimen is not recommended for initial therapy unless ≥ 85% of local *H.pylori* strains are susceptible or the regimen is still clinically effective in the locality. For multidrug-resistant strains of H. pylori, triple therapy with proton pump inhibitors, rifabutin and amoxicillin (3) appears to be effective.

Infected patients with duodenal or gastric ulcers need to continue acid-suppressive therapy for at least 4 weeks. Eradication can be confirmed by a urease breath test, stool antigen test, or esophagogastroduodenoscopy done 4 weeks or more after completion of therapy. Confirmation of eradication is reasonable in all patients, but is mandatory in patients with severe manifestations of *H.pylori* infection (eg, ulcer complicated by bleeding). Without eradication of the infection, there is a possibility of recurrence of ulcerative bleeding.

If neither quadruple nor triple therapy results in eradication of H. pylori, the treatment is repeated. If two courses are unsuccessful, some experts recommend endoscopy for culture and antibiotic susceptibility testing. If quadruple bismuth therapy fails, clinicians should make joint decision-making with patients to determine whether they should receive levofloxacin triple therapy (with amoxicillin), rifabutin triple therapy or bismuth alternative therapy.

Conclusion. H.pylori is a Gram-negative microorganism, highly adapted to existence in an acidic environment, most often affecting the stomach; the incidence of this infection increases with age - by the age of 60, about 50% of people are infected.

Infection provokes the development of gastric, duodenal and prepyloric ulcers and also increases the risk of adenocarcinoma and gastric lymphoma.

Initial diagnosis should be made with a urea breath test or stool antigen analysis; if endoscopy is performed for other indications, tissue samples taken for biopsy should be analyzed using a rapid urease test or histological staining.

Patients with complications (eg, peptic ulcer, cancer) should receive eradication therapy; a typical regimen includes quadruple therapy in regions with >15% clarithromycin resistance or proton pump inhibitors plus 2 antibiotics (eg, clarithromycin plus either amoxicillin or metronidazole).

Confirmation of the result of treatment is carried out using a urease breath test, determination of antigen in feces or esophagogastroduodenoscopy.

Thus, this is an imbalance between the state of blood circulation and metabolism, which increases with the increase in the activity of vital processes. From modern clinical positions, Chronic heart failure is a set of characteristic symptoms (shortness of breath, fatigue and decreased physical activity, edema and others) disease, body fluid retention.

Chronic heart failure Chronic heart failure is characterized by repeated episodes of exacerbation (decompensation) characterized by a sudden or often gradual increase in symptoms and signs of heart failure.

Causes. Chronic heart failure can develop against the background of almost any disease of the cardiovascular system, but the main three are the following supranosological forms: coronary artery disease, arterial hypertension and heart defects.

Heart failure (Latin: insufficientia cordis) is a syndrome resulting from decompensated myocardial dysfunction. This is manifested by an increase in the volume of intercellular fluid, a decrease in the perfusion of organs and tissues. The pathophysiological basis of this syndrome is that the heart cannot meet the metabolic needs of the body due to impaired pumping function, or it does so by increasing end-diastolic pressure in the ventricles. In some patients with heart failure, when the pumping function is impaired, the clinical manifestations are caused by impaired filling or emptying of the heart chambers. Myocardial dysfunction (systolic or diastolic) is initially asymptomatic, and only then does heart failure begin to manifest.

Left ventricular heart failure (LVH) is a heart failure that occurs when the left heart is damaged and overloaded, and is characterized by clinical signs of advanced venous congestion in the pulmonary circulation. Left ventricular failure is manifested by a decrease in cerebral circulation (dizziness, darkening of the eyes, fainting) and a decrease in coronary circulation (angina), which is characteristic of aortic malformation, coronary heart disease, arterial hypertension, obstructive cardiomyopathy.

According to the type of right ventricle, heart failure is a type characterized by insufficient blood flow from the right ventricle to the pulmonary artery and stagnation of blood in the systemic circulation.

Depending on how quickly heart failure develops, it is divided into acute and chronic. Acute heart failure can be related to trauma, toxins, heart disease, and can quickly lead to death if left untreated.

Chronic heart failure develops over a long period of time and is manifested by a complex of characteristic symptoms (shortness of breath, fatigue and decreased physical activity, edema, etc.), associated with insufficient perfusion of organs and tissues during rest or physical exercise. is fluid and is often associated with fluid retention in the body. Currently, there are more than 25 million people with heart failure syndrome in the world.

Anemia is a disease characterized by a decrease in the number of erythrocytes and the amount of hemoglobin in the blood, and a change in its quality. Anemia can be caused by a violation of the process of blood formation, the bone marrow, the main blood-forming tissue, cannot perform its function sufficiently. Anemia caused by iron and vitamin B12 deficiency is quite common. Anemia is often observed in the case of long-term bleeding, gastric ulcer or stomach and duodenal ulcer. Iron deficiency anemia is common in women with long and heavy periods. Anemia related to iron deficiency is caused by frequent pregnancy and long-term breastfeeding, because during pregnancy and breastfeeding, part of the iron reserve in the mother's body is transferred to the child. Anemia observed in young children occurs as a result of improper feeding, as well as lack of food. A decrease in the amount of hemoglobin in the blood, while the number of erythrocytes is slightly reduced or equal, is one of the main symptoms of anemia related to iron deficiency. The patient is discolored, often complains of rapid fatigue, headache, dizziness, blurred vision, hair loss, and brittle nails. Sometimes it becomes difficult to swallow, the patient wants to eat things that are not usually eaten (chalk, lime, chalk, etc.), likes spicy, salty foods. In the prevention and treatment of anemia related to iron deficiency, it is necessary to identify and eliminate possible sources of blood loss in time, achieve a certain level of planning of pregnancy and childbirth, and follow a balanced diet. Anemia caused by vitamin B12 or folic acid deficiency is much rarer. This type of anemia has specific symptoms: sore tongue, signs of damage to the nervous system (funicular myelosis) when the disease is missed. In order to prevent this type of anemia, it is very important to timely identify and treat chronic diseases of the gastrointestinal tract, especially those with diarrhea. In places where worms are spread, it is necessary to take measures to prevent infection from them, and when the disease appears, it is necessary to treat it in time. There are many types of hemolytic anemia associated with extensive destruction of erythrocytes. They can be hereditary or acquired, and are usually characterized by yellowing of the skin and mucous membranes, and a decrease in the number of red blood cells and hemoglobin. In all types of anemia, it is necessary to consult a doctor and get the right treatment in time.

Every adult has about 5 liters of blood in their body. If you're wondering how much blood is in your body, remember that it makes up 7% of your body weight. Blood is basically water with salts in it, but its most important component is the cells that circulate in the blood. These cells are formed in the bone marrow, and their value is difficult to overestimate. Erythrocytes, i.e. red blood cells, deliver oxygen from the lungs to all tissues of our body and participate in respiration. It is they who make the blood red. Others, such as white blood cells, protect us

from germs and viruses. Finally, a third type of cell, platelets, stop bleeding when we are injured. If the number of any cell is too low or if it stops performing its function, our health deteriorates.

The development of anemia is related to the lack of one of these cells - red blood cells. However, sometimes, while the number of red blood cells is preserved, the number of their main component, the hemoglobin protein that carries oxygen, decreases. As a result, red blood cells become "poor" and their functions fail, even if the number of red blood cells does not change. Thus, we learned what anemia is - a condition that occurs when the amount of red blood cells or hemoglobin in them decreases. These cells are responsible for delivering the oxygen we breathe to all tissues and cells in the body, so with anemia, the body begins to "suffocate" gradually.

Fortunately, our bodies are perfectly adaptable and over time can compensate for the lack of red blood cells or hemoglobin (just remember how people living in mountainous areas have adapted to thin air with low oxygen pressure). However, sooner or later, the body loses its ability to fight anemia and hide its consequences. In this case, symptoms of anemia begin to appear.

References

1. Yang J.C, Lin C.J, Wang H.L, et al: High-dose dual therapy is superior to standard first-line or rescue therapy for *Helicobacter pylori* infection. *Clin Gastroenterol Hepatol* 13(5):895–905.e5, 2015. doi: 10.1016/j.cgh.2014.10.036

2. Fallone C.A, Chiba N, van Zanten S.V, et al: The Toronto consensus for the treatment of *Helicobacter pylori* infection in adults. *Gastroenterology* 151(1):51–69, 2016. doi: 10.1053/j.gastro.2016.04.006

3. Fiorini G, Zullo A, Vakil N, et al: Rifabutin triple therapy is effective in patients with multidrug-resistant strains of *Helicobacter pylori*. *J Clin Gastroenterol* 52(2):137–140, 2018. doi: 10.1097/MCG.0000000000000540

4. Shah S.C, Iyer P.G, Moss S.F: AGA clinical practice update on the management of refractory *Helicobacter pylori* infection: Expert review. *Gastroenterology* 160(5):1831–1841, 2021. doi: 10.1053/j.gastro.2020.11.059