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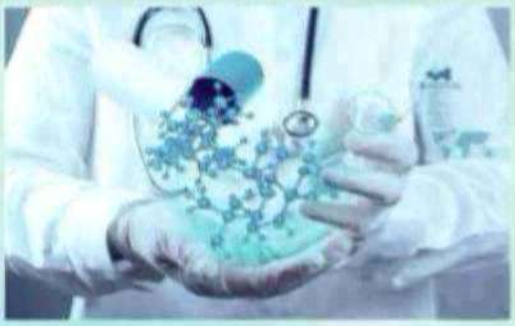

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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

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HELICOBACTER PYLORI: HISTORY AND MODERN UNDERSTANDING

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✓ ***Resume***

The bacterium was rediscovered in 1979 by Australian pathologist Robin Warren, who later conducted further research on it together with Barry Marshall, beginning in 1981. Warren and Marshall succeeded in isolating and extracting the microorganism from samples of the human stomach lining. They were also the first to successfully culture this microorganism on artificial nutrient media. In 2005, the Nobel Assembly at the Karolin Institute in Stockholm awarded the Nobel Prize in Physiology and Medicine to the Australian scientists Barry Marshall and Robin Warren for the discovery of **Helicobacter pylori** ***and its role in the development of gastritis and peptic ulcers. It is currently confirmed that*** **Helicobacter pylori** ***can cause not only non-cardia gastric cancer, but also colon cancer, MALT lymphoma, and cardia gastric cancer.***

Keywords: The bacterium was discovered in 1979 by Australian pathologist Robin Warren. Warren and Marshall conducted joint research starting in 1981. They successfully isolated and extracted the microorganism from samples of human gastric mucosa. In 2005, the Nobel Assembly of the Karolinska Institute in Stockholm awarded the Nobel Prize in Physiology or Medicine to Australian scientists Barry Marshall and Robin Warren.

ХЕЛИКОБАКТЕР ПИЛОРИ: ИСТОРИЯ И СОВРЕМЕННОЕ ПОНИМАНИЕ

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✓ ***Резюме***

Бактерия была открыта в 1979 году австралийским патологом Робином Уорреном, который позже начал совместные исследования с Барри Маршаллом, начиная с 1981 года. Уоррен и Маршалл добились успеха в изоляции и извлечении микроорганизма из образцов слизистой оболочки человеческого желудка. В 2005 году Нобелевская ассамблея Каролинского института в Стокгольме присудила Нобелевскую премию по физиологии или медицине австралийским ученым Барри Маршаллу и Робину Уоррену за открытие Helicobacter pylori и его роли в развитии гастрита и язвенной болезни. В настоящее время подтверждено, что Helicobacter pylori может вызывать не только некардиальный рак желудка, но также рак ободочной кишки, MALT-лимфому и кардиальный рак желудка.

Ключевые слова: Откритe бактерия была открыта в 1979 году австралийским патологом Робином Уорреном. Совместные исследования с Барри Маршаллом, начиная с 1981 года. Уоррен и Маршалл добились успеха в изоляции и извлечении микроорганизма из образцов слизистой оболочки человеческого желудка. В 2005 году Нобелевская ассамблея

Каролинского института в Стокгольме присудила Нобелевскую премию по физиологии или медицине австралийским ученым Барри Маршаллу и Робину Уоррену.

History. In 1875, German scientists discovered a spiral-shaped bacterium in the mucous membrane of the human stomach. The bacterium did not grow in culture (on the artificial nutrient media known at the time), and this accidental discovery was forgotten.

In 1886, Professor Walery Jaworski from the Jagiellonian University in Kraków, while studying sediment from human stomach washings, discovered not only bacteria resembling rods in shape but also a number of bacteria with a characteristic spiral shape. He named the bacterium he discovered *Vibrio rugula*. He was the first to suggest a possible etiological role of this microorganism in the pathogenesis of stomach diseases. His work on this topic was included in the Polish *Handbook of Stomach Diseases* and published in 1899. However, this work had little impact on the broader medical and scientific community, as it was written in Polish [1, 2].

In 1893, Italian researcher Giulio Bizzozero described a similar spiral-shaped bacterium living in the acidic contents of dogs' stomachs.

In 1974, Professor I. A. Morozov from Moscow discovered spiral-shaped bacteria in the material from patients after vagotomy, located in the intracellular tubules of stomach cells, as well as in ulcer patients who had not undergone vagotomy. However, microbiologists did not know how to culture these bacteria, and the discovery was forgotten for another ten years [1, 2].

The bacterium was rediscovered in 1979 by Australian pathologist Robin Warren, who later conducted further research on it together with Barry Marshall, beginning in 1981. Warren and Marshall succeeded in isolating and extracting the microorganism from samples of the human stomach lining. They were also the first to successfully culture this microorganism on artificial nutrient media [2,5].

In their original publication, Warren and Marshall proposed that most stomach ulcers and gastritis in humans are caused by infection with the microorganism *Helicobacter pylori*, rather than by stress or spicy food, as was previously believed.

In 1979, R. Warren noted that curved bacteria were often seen in gastric biopsy samples during histological examination. These microorganisms were present in the layer of gastric mucus, but not in the mucous membrane itself. Warren, according to his own account, discovered the "curved rods" on his birthday (June 11, 1979), and it appears to have happened by chance: during a routine diagnostic histological examination, he noticed an unusual blue line on the surface of the gastric mucosa in a patient with active chronic gastritis. After studying a considerable number of biopsy samples using various staining methods, Warren suggested that the development of gastritis was linked to a certain bacterium closely interacting with the surface of the stomach epithelium [3,4].

Warren's two years of perseverance—enduring skepticism, distrust from colleagues, and outright refusals to collaborate—were ultimately rewarded when he met Barry Marshall, a medical intern who became interested in the pathologist's effort to classify gastritis as a bacterial infection. Together, they began searching for a way to isolate the microorganism from biopsy material. The researchers inoculated biopsy samples into selective media and incubated them under microaerophilic conditions. Since most *Campylobacter* species would die under these conditions within 48 hours, plates without visible growth were typically discarded after three days [1].

Initially, cultures from about 30 patients were negative, but by chance, one sample was incubated for five days over the Easter holiday—and colonies appeared! Later, the microorganism was successfully isolated from 11 patients. It was described and named *Campylobacter pyloridis*, now known as *Helicobacter pylori* [2].

Warren and Marshall continued their clinical research, gathering more and more evidence linking *H. pylori* to gastritis and stomach ulcers. They proposed that the disease should be treated with antibacterial drugs. The scientists concluded that the bacteria could be transmitted from person to person through dirty dishes or even kissing. Once in the stomach, the bacteria protect themselves from its harsh environment by surrounding themselves with a special enzyme. They then bore through the stomach's mucous membrane and attach to the epithelial cells. Beneath the mucus layer, they thrive, as they are shielded from hydrochloric acid [1].

In these comfortable conditions, the bacteria produce endotoxins, which in turn lead to inflammation of the cells. With prolonged presence, this can result in necrosis of the epithelial cells and the development of peptic ulcers.

However, the researchers' new evidence continued to be met with skepticism in the scientific community. Even when Barry Marshall, in the best traditions of the 19th century, conducted a classic self-experiment, the Australian scientists were still not believed. After pharmacologically suppressing his stomach's hydrochloric acid secretion, he swallowed a concentrated suspension of bacterial cells isolated from a patient with active chronic gastritis.

Soon afterward, Marshall developed histologically confirmed acute gastritis, and from a biopsy of his stomach lining, the same “curved rod” was isolated. After undergoing a course of therapy planned according to the antibiotic sensitivity of the experimental strain, the experimenter recovered. His recovery was confirmed through endoscopic, histological, and bacteriological methods.

Interestingly, a second self-infection experiment, carried out independently of Marshall, was even more dramatic: New Zealand gastroenterologist A. Morris, who infected himself with a strain isolated from a patient with peptic ulcer disease, ended up battling chronic gastritis for several years.

In 2005, the Nobel Assembly at the Karolin Institute in Stockholm awarded the Nobel Prize in Physiology and Medicine to the Australian scientists Barry Marshall and Robin Warren for the discovery of *Helicobacter pylori* and its role in the development of gastritis and peptic ulcers.

In 1991, four reports for the first time demonstrated a link between *Helicobacter pylori* infection and the presence and development of stomach cancer.

H.pylori infection and gastric cardia cancer. Helicobacter pylori (HP) has been demonstrated the causative factor of various gastrointestinal diseases; nevertheless, the relationship between HP infection and gastroesophageal reflux disease (GERD) is still debated. To date, different studies have examined the relation ship between atrophic gastritis due to HP infection and reflux oesophagitis with conflicting results. Between January 2001 and January 2003, 146 consecutive patients with daily reflux symptoms for at least one year were evaluated at the Department of Surgery, Tor Vergata University Hospital, Rome and were included in this prospective study. The study had been approved by the Institutional Committee of the Tor Vergata University of Rome. Exclusion criteria were the following: 1. Previous therapy to eradicate HP. 2. Concomitant assumption of aspirin and non-steroidal anti-inflammatory drugs 3. Previous surgical procedures on digestive tract. All patients underwent a pre-treatment evaluation, which included anamnesis, clinical examination, EGDS with biopsy, oesophageal manometry and 24 hours pH-metry. The exact association between HP and reflux disease continues to be debated. This clinical, endoscopic manometric and pH-metric data shows significant role of HP infection neither in the development of GERD nor in the pathogenesis of reflux esophagitis. Nevertheless, current data do not provide sufficient evidence to define the relationship between HP and GERD. However, this is an evolving area with ongoing research and further assessments in prospective large studies are warranted.

According to the theses published by researchers from Uzbekistan at the 2023 Congress of Russian Oncologists, **153 patients diagnosed with cardioesophageal and esophageal cancer underwent anti-*Helicobacter pylori* treatment**. The treatment made it possible to reduce severe dyspeptic symptoms in 113 patients (73.8%) and eliminate pain syndrome in 133 patients (86.9%), which contributes to a more favorable course of chemotherapy. Considering the results of the study, the administration of anti-*Helicobacter* therapy is deemed appropriate, as it contributes to the improvement of patients' medical rehabilitation [8].

Marlene Cavaleiro-Pinto, Ba´rbara Peleteiro, Nuno Lunet , Henrique Barros systematically reviewed published studies addressing the association between H. pylori infection and gastric cardia cancer (up to June 2009), and extracted relative risk (RR) estimates for the association with cardia and non-cardia cancers. Summary RR estimates and 95% confidence intervals (95% CI) were computed using random-effects

models. Subgroup analyses were conducted, namely according to gastric cancer risk settings. In conclusion, the present study is the most comprehensive assessment of the association between H. pylori infection and gastric cardia cancer, adding to previous knowledge an update in understanding the role of low- and high-gastric-cancer-risk settings on this subject. Our results support the hypothesis of different etiologies for gastric cardia cancer, but the achievement of more robust and definitive explanations for the heterogeneous findings on this topic requires the conduction of prospective investigations able to classify accurately the distinct subtypes of cardia tumors taking into account the presence of gastric atrophy in the non-neoplastic mucosa, cancer histologic type, and reflux symptoms, both in high- and low-gastric cancer-risk populations [8].

MALT lymphoma. Low grade B cell mucosa associated lymphoid tissue (MALT) lymphoma of the stomach is usually an indolent tumour that remains localised for a long time before dissemination occurs. MALT appears in the stomach in response to infection by Helicobacter pylori, which is present in 80–90% of cases. The pathogenesis of the evolution from chronic gastritis to malignant lymphoma has not yet been fully explained and the exact role of H.pylori in the pathogenesis and progression of gastric lymphoma remains unclear. Case report from D Sandmeier, J Benhattar, H Bouzourene, Institute of Pathology, Bugnon 25, CH 1011 Lausanne, Switzerland concludes a patient with a lymphoid proliferation in the gastric mucosa that fulfilled the criteria for a B cell low grade MALT lymphoma, but who did not progress during 11 years, despite the absence of H pylori eradication. The detection of immunohistochemistry rearrangements remains a useful tool for the diagnosis of gastric MALT lymphoma. PCR findings suggest that coexisting or new lymphomatous clonal independent foci may emerge during the follow up of MALT lymphomas. This case report illustrates the indolence of low grade MALT lymphoma of the stomach and supports the hypothesis that sustained H.pylori infection is, by itself, not sufficient for tumour progression.

G.S. Dzhulay, T.E. Dzhulay Tver State Medical University; 4 Sovietskaya Str., Tver, Russian Federation reported several patients examination and treatment results. Clinical observation demonstrates an etiological link between gastric MALT lymphoma and the persistence of *Helicobacter pylori* infection in the upper gastrointestinal tract, as well as the positive effect of immunochemotherapy and persistent eradication therapy, which contributed to the regression of both the gastric MALT lymphoma itself and the involvement of intra-abdominal lymph nodes [7].

Helicobacter pylori and colon cancer. There is evidence that bacteria are linked to a number of extragastric disorders. Since it was initially hypothesized that the link between extragastric illnesses and colon cancer. Batool Ali Khorsheed, Raghdah Maythem Hameed1 , Zahraa Hussein Khorsheed published article aboaut correlation og H.pylori and colon cancer. The study was to obtain the correlation between colon cancer and H. pylori infection. In this study, blood samples were collected from the patients with colon cancer to assess the presence of anti-H. pylori infection antibodies in 10 patients with colorectal cancer and 10 with no specific pathologies as a control group. Current evidence suggested that H. pylori infection was associated with colorectal cancer. These findings suggested that H. pylori infection may be a possible risk factor for colorectal cancer, which is important for the prevention of colorectal cancer in the adult population [10].

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