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# Erosive-Ulcerative Gastrointestinal Lesions in Coronary Artery Disease: The Role of *Helicobacter pylori* and Prevention Strategies in Different Clinical Scenarios

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**Aim:** to assess the prevalence and characteristics of erosive-ulcerative lesions of the gastrointestinal tract in patients with chronic coronary artery disease and acute myocardial infarction, determining the role of *Helicobacter pylori* (*H. pylori*) and other independent risk factors in their pathogenesis.
**Materials and methods.** A single-center prospective cohort study analyzed data from 110 patients divided into two groups based on the clinical form of coronary artery disease (CAD). Group 1 ($n$ = 56) included patients with acute myocardial infarction (AMI) after primary percutaneous coronary intervention (mean age — 62.0 years) and consisted predominantly of men ($n$ = 41; 73.21 %). Group 2 ($n$ = 54) included patients with stable CAD who had undergone percutaneous coronary intervention within the preceding year (mean age — 67.5 years), and there was also a predominance of men ($n$ = 37; 72.55 %). All patients underwent a comprehensive examination, including the collection of clinical and anamnestic data and laboratory diagnostics. Esophagogastroduodenoscopy (EGD) was performed at different time points depending on clinical status: patients with stable CAD underwent EGD at the time of hospitalization, while for patients with AMI, the examination was conducted in a delayed manner to minimize risks. *H. pylori* infection was diagnosed using the $^{13}$C-urea breath test in AMI patients and via biopsy during EGD in stable CAD patients. The one-month follow-up examination included: esophagogastroduodenoscopy to assess the condition of the upper gastrointestinal mucosa, and the $^{13}$C-urea breath test for patients with baseline *H. pylori* infection to evaluate the efficacy of eradication therapy.
**Results.** The prevalence of *H. pylori* was 46.4 % in the AMI group and 31.5 % in the stable CAD group. Planned EGD in patients with stable CAD before treatment revealed erosive-ulcerative lesions of the upper gastrointestinal tract in 51.9 % of cases, with 46.4 % of these lesions being associated with active *H. pylori* infection ($p$ = 0.012). The application of active screening and preventive *H. pylori* eradication strategy in AMI patients resulted in successful eradication in 92.3 % of cases. At the 1-month follow-up EGD, mucosal changes persisted in 30.9 % of AMI patients; however, the majority of these findings were minimal and clinically insignificant (superficial gastritis, catarrhal bulbitis). True erosive-ulcerative lesions were detected in only 18.2 % of patients, indicating the role of the ulcerogenic effect of antiplatelet therapy in their pathogenesis. In patients with stable CAD, targeted therapy (eradication of *H. pylori* and prescription of proton pump inhibitors) led to a significant reduction in the frequency of erosive-ulcerative lesions from 51.9 to 7.4 % ($p$ < 0.001). Multivariate analysis confirmed that in stable CAD, active *H. pylori* infection is an independent risk factor for gastrointestinal lesions (adjusted odds ratio [OR] — 4.32; 95 % CI: 1.22–18.20; $p$ = 0.023). In contrast, in the AMI group after preventive eradication, this association completely lost statistical significance (OR = 0.84; 95 % CI: 0.22–3.20; $p$ = 0.79).
**Conclusions.** Erosive-ulcerative gastrointestinal lesions in patients with CAD are closely associated with *H. pylori* infection and can be effectively prevented through its early diagnosis and eradication. In patients with AMI, preventive *H. pylori* eradication is a mandatory but insufficient element of prophylaxis and must be complemented by measures to mitigate the risks associated with dual antiplatelet therapy. Thus, an individualized approach to gastroprotection, tailored to the clinical form of coronary artery disease, enhances the safety and efficacy of pharmacotherapy in these patient groups.
**Keywords:** acute myocardial infarction, ischemic heart disease, erosive and ulcerative lesions, gastrointestinal tract, *Helicobacter pylori*, risk factors for antiplatelet therapy
**Conflict of interest:** the authors declare no conflict of interest.

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# Эрозивно-язвенные поражения желудочно-кишечного тракта при ишемической болезни сердца: роль *Helicobacter pylori* и антитромбоцитарной терапии

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**Цель:** оценить распространенность и характер эрозивно-язвенных изменений желудочно-кишечного тракта у пациентов с хронической ишемической болезнью сердца и острым инфарктом миокарда, а также определить роль *Helicobacter pylori* (*H. pylori*) и других независимых факторов риска в их патогенезе.

**Материалы и методы.** В одноцентровом проспективном когортном исследовании проанализированы данные 110 пациентов, разделенных на две группы в зависимости от клинической формы ишемической болезни сердца (ИБС). Первая группа ($n$ = 56) включала пациентов с острым инфарктом миокарда после первичного чрескожного коронарного вмешательства (средний возраст — 62,0 года) и состояла преимущественно из мужчин ($n$ = 41; 73,2 %). Вторая группа ($n$ = 54) включала пациентов со стабильной ИБС, перенесших чрескожное коронарное вмешательство в течение предшествующего года (средний возраст — 67,5 года), в ней также преобладали мужчины ($n$ = 37; 72,6 %). Всем пациентам проведено комплексное обследование, включавшее сбор клинико-анамнестических данных и лабораторную диагностику. Эзофагогастродуоденоскопия (ЭГДС) выполнялась в различные сроки в зависимости от клинического состояния: пациентам со стабильной ИБС — в момент госпитализации, тогда как пациентам с острым инфарктом миокарда в целях минимизации рисков — отсроченно. Для диагностики *H. pylori* у пациентов с острым инфарктом миокарда применялся $^{13}$C-уреазный дыхательный тест, а у пациентов со стабильной ИБС — гистологическое исследование биоптатов, полученных во время ЭГДС. Контрольное обследование через 1 месяц включало ЭГДС для оценки состояния слизистой оболочки верхних отделов желудочно-кишечного тракта и $^{13}$C-уреазный дыхательный тест у пациентов с исходной инфекцией *H. pylori* для оценки эффективности эрадикационной терапии.

**Результаты.** Распространенность *H. pylori* составила 46,4 % в группе пациентов с острым инфарктом миокарда и 31,5 % в группе больных со стабильной ИБС. Плановая ЭГДС у пациентов со стабильной ИБС до лечения выявила эрозивно-язвенные поражения верхних отделов желудочно-кишечного тракта в 51,9 % случаев, причем 46,4 % этих поражений были ассоциированы с активной хеликобактерной инфекцией ($p$ = 0,012). Применение стратегии активного скрининга и превентивной терапии *H. pylori* у пациентов с острым инфарктом миокарда позволило достичь успешной эрадикации в 92,3 %. На контрольной ЭГДС через 1 месяц у 30,9 % пациентов с острым инфарктом миокарда сохранялись изменения слизистой оболочки, однако в большинстве случаев они были представлены минимальными и клинически малозначимыми находками (поверхностный гастрит, катаральный бульбит); собственно эрозивно-язвенные поражения выявлены лишь у 18,2 % больных, что подтверждает ульцерогенный эффект антитромбоцитарной терапии в их патогенезе. У пациентов со стабильной ИБС целенаправленная терапия (включавшая эрадикацию *H. pylori* и назначение ингибиторов протонной помпы) привела к достоверному снижению частоты эрозивно-язвенных поражений с 51,9 до 7,4 % ($p$ < 0,001). Многофакторный анализ подтвердил, что при стабильной ИБС активная инфекция *H. pylori* является независимым фактором риска поражений желудочно-кишечного тракта (скорректированное отношение шансов [ОШ] — 4,32; 95% ДИ: 1,22–18,20; $p$ = 0,023). Напротив, в группе пациентов с острым инфарктом миокарда после проведения превентивной эрадикации данная ассоциация полностью теряла статистическую значимость (ОШ = 0,84; 95% ДИ: 0,22–3,20; $p$ = 0,79).

**Выводы.** Эрозивно-язвенные поражения желудочно-кишечного тракта у пациентов с ИБС тесно связаны с инфекцией, вызванной *Helicobacter pylori*, и могут быть эффективно предотвращены путем ее ранней диагностики и эрадикации. У пациентов с острым инфарктом миокарда превентивная эрадикация *H. pylori* является обязательным, но недостаточным элементом профилактики и должна дополняться мерами снижения рисков, связанных с двойной антитромбоцитарной терапией. Таким образом, индивидуальный подход к гастропротекции в зависимости от клинической формы ИБС повышает безопасность и эффективность медикаментозного лечения данных групп пациентов.

**Ключевые слова:** острый инфаркт миокарда, ишемическая болезнь сердца, эрозивно-язвенные поражения, желудочно-кишечный тракт, *Helicobacter pylori*, факторы риска антитромбоцитарной терапии

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## Introduction

Antiplatelet therapy is widely used for secondary prevention of cardiovascular events in order to reduce the risk of recurrent ischemic episodes after percutaneous coronary intervention [1, 2]. According to clinical guidelines, the recommended duration of dual antiplatelet therapy in patients, who underwent percutaneous coronary intervention, is at least 12 months after an acute coronary syndrome and at least 6 months in stable coronary artery disease, to prevent thrombotic complications [3]. However, antiplatelet therapy is associated with an increased risk of gastrointestinal complications, in particular erosive-ulcerative lesions and bleeding [4, 5]. An important independent factor that potentiates the ulcerogenic effect of antiplatelet agents is *Helicobacter pylori* (*H. pylori*) infection [6, 7]. By inducing chronic inflammation of the gastric and duodenal mucosa, this microorganism substantially reduces mucosal resistance to the aggressive action of drugs, thereby creating a background for the development of both acute drug-induced injuries and chronic peptic ulcers [8]. Identification of risk factors for the progression of gastrointestinal complications in the setting of continuous antiplatelet therapy is a key task; its solution is necessary for the development of risk-stratification tools and personalized treatment approaches in patients with stable coronary artery disease and acute myocardial infarction.

**Aim:** to assess the frequency of erosive-ulcerative changes of the upper gastrointestinal tract (GIT) in patients with chronic coronary artery disease (CAD) and acute myocardial infarction (AMI) receiving antiplatelet therapy, as well as to determine the role of *H. pylori* infection in their development, in order to justify the advisability of routine screening and eradication as a strategy for primary and secondary prevention of gastrointestinal complications.

## Materials and methods

A single-center prospective cohort study was conducted at the M.P. Konchalovsky City Clinical Hospital (Zelenograd, Moscow) and included 110 patients older than 18 years. Depending on the clinical form of coronary artery disease, the patients were divided into two groups. Group 1 comprised 56 patients with acute myocardial infarction (AMI) at the time of hospitalization (mean age — 62.0 years), the majority (73.2 %; $n$ = 41) were men. Group 2 comprised 54 patients with stable CAD who had undergone elective coronary stenting within 6–12 months before enrolment and were under planned follow-up; their mean age was higher (67.5 years), with a similar proportion of men (72.6 %; $n$ = 37).

In accordance with current guidelines, all patients received antiplatelet therapy. In the AMI group, 55 patients (98.2 %) were on dual antiplatelet therapy consisting of acetylsalicylic acid (ASA) plus a P2Y12 receptor inhibitor (ticagrelor or clopidogrel), while one patient (1.8 %) received ASA monotherapy. In the stable CAD group, 43 patients (79.6 %) continued dual antiplatelet therapy, whereas 11 patients (20.4 %) were receiving ASA monotherapy.

The following examinations were performed for all participants:

- collection of clinical and anamnestic data (cardiac history, life style factors, risk factors, concomitant medications);
- laboratory tests (complete blood count, biochemical panel, coagulation tests);
- instrumental and laboratory diagnostics of lesions of the upper gastrointestinal tract and *H. pylori* infection.

In the stable CAD group, all patients underwent planned EGD (FUJIFILM EG-760R, Japan) for visual assessment of the upper GIT mucosa. Simultaneously, invasive diagnosis of *H. pylori* was carried out by obtaining biopsy specimens with subsequent histological examination (hematoxylin and eosin and Romanowsky staining). After one month, the effectiveness of treatment was controlled by repeat EGD in all participants and a $^{13}$C-urea breath test in patients with baseline *H. pylori* infection.

The diagnostic approach in AMI patients was individualized. Patients with clinical signs of gastrointestinal bleeding underwent emergency EGD. In the remaining patients, EGD in the acute phase was considered undesirable because of the risk of hemodynamic complications; therefore, the non-invasive $^{13}$C-urea breath test was used for primary detection of *H. pylori*. Patients with confirmed infection (δ over baseline [DOB] > 4 ‰) were considered eligible for standard eradication therapy. However, in patients receiving ticagrelor, a risk of pharmacokinetic interaction between this drug and clarithromycin (a component of standard regimens) was identified. To avoid adverse effects, such patients received a modified first-line therapy in correspondence with the recommendations of the Maastricht VI/Florence Consensus and the safety principles of antiplatelet therapy. Patients on

ticagrelor were prescribed bismuth-based quadruple therapy (proton-pump inhibitor + bismuth tripotassium dicitrate + metronidazole + tetracycline) or a levofloxacin-containing regimen. Patients receiving clopidogrel were given standard triple therapy (PPI + amoxicillin + clarithromycin) for 14 days. The effectiveness of treatment was assessed after one month by repeat $^{13}$C-urea breath test and elective EGD to evaluate the mucosal condition.

Statistical analysis was performed using R software (v. 4.4.2) and the *gtsummary, tidyr, dplyr, readxl* packages. Descriptive statistics included analysis of data distribution: for continuous variables — median with quartiles (*Me* [*Q*1; *Q*3]); for categorical variables — absolute numbers and percentages. For comparison of independent samples, Fisher's exact test was used for categorical data, and the Mann — Whitney *U*-test for continuous variables. In unifactorial analysis, separate logistic regression models were built for each variable under study. In multivariate analysis, sex and age were additionally included in each model. Regression coefficients were exponentiated to obtain odds ratios (OR) with 95 % confidence intervals (CI). Statistical significance was set at $p < 0.05$ for all comparisons. Correction for multiple testing was performed using the Bonferroni method.

## Results

A total of 110 patients were included in the study and divided into two groups: the first group consisted of 56 patients with AMI, the second group — 54 patients with stable CAD. The groups did not differ with regard to sex, prevalence of smoking, or elevated body mass index. After Bonferroni correction for multiple comparisons, no significant difference in age was found either. At the same time, history of previous myocardial infarction was significantly more frequent in patients with stable CAD ($p < 0.001$). This group was also characterized by a predominance of ASA monotherapy ($p = 0.002$) and of the dual antiplatelet therapy combination "ASA + clopidogrel" ($p < 0.001$), whereas the combination "ASA + ticagrelor" was more frequently used in patients with AMI ($p < 0.001$). Prescription of proton pump inhibitors was also statistically significantly more common in the AMI group ($p < 0.001$) (Table 1).

***Table 1.*** Main characteristics of patient groups

| Parameter | AMI group (*n* = 56) | Stable CAD group (*n* = 54) | *p* |
|---|---|---|---|
| ***Demographic indices*** | | | |
| Male sex, *n* (%) | 41 (73.2 %) | 41 (76.0 %) | 0.828 |
| Age, years, *Me* [*Q*1; *Q*3] | 62.0 [53.8; 68.0] | 67.5 [60.3; 74.0] | 0.009 |
| ***Clinical risk factors*** | | | |
| Body mass index, kg/m$^2$, *Me* [*Q*1; *Q*3] | 29.6 [27.1; 32.2] | 29.0 [25.9; 32.3] | 0.528 |
| Smoking, *n* (%) | 24 (42.9 %) | 21 (38.9 %) | 0.702 |
| Type 2 diabetes mellitus, *n* (%) | 11 (19.7 %) | 20 (37.0 %) | 0.056 |
| Chronic kidney disease, stage ≥ 3a, *n* (%) | 10 (17.9 %) | 11 (20.4 %) | 0.738 |
| Chronic obstructive pulmonary disease, *n* (%) | 3 (5.4 %) | 14 (26.0 %) | 0.003* |
| History of stroke, *n* (%) | 3 (5.4 %) | 5 (9.3 %) | 0.481 |
| Heart failure, *n* (%) | 47 (84.0 %) | 51 (94.5 %) | 0.130 |
| Prior myocardial infarction, *n* (%) | 11 (19.6 %) | 31 (57.4 %) | <0.001* |
| ***History of erosive-ulcerative gastrointestinal lesions*** | | | |
| History of gastric ulcer, *n* (%) | 5 (8.9 %) | 6 (11.1 %) | 0.760 |
| History duodenal ulcer, *n* (%) | 2 (3.6 %) | 3 (5.6 %) | |
| ***Pharmacotherapy at the time of EGD*** | | | |
| Acetylsalicylic acid monotherapy, *n* (%) | 1 (1.8 %) | 11 (20.4 %) | 0.002* |
| Acetylsalicylic acid + clopidogrel, *n* (%) | 2 (3.6 %) | 27 (50.0 %) | <0.001* |
| Acetylsalicylic acid + ticagrelor, *n* (%) | 53 (94.7 %) | 16 (29.6 %) | <0.001* |
| Proton-pump inhibitor, *n* (%) | 52 (92.9 %) | 29 (53.7 %) | <0.001* |

***Note:*** *AMI — acute myocardial infarction; CAD — coronary artery disease; * the threshold of statistical significance after Bonferroni correction is p < 0.0033.*

In the stable CAD group, one case of active gastrointestinal bleeding was registered, which required emergency endoscopic hemostasis. The clinical presentation, diagnosis, and management of this patient are described in detail in a previously published case report [9]. This patient was included in the present prospective study, which allowed us to analyze his case in the context of a systematic assessment of risk factors. EGD revealed a chronic gastric ulcer with bleeding (Forrest Ib) against the background of active *H. pylori* infection (Fig. 1).

In the AMI group, three cases of clinically significant gastrointestinal bleeding were recorded within the first 48 hours of hospitalization (5.4 %); one of these cases had a fatal outcome within the following two month. In all three cases, emergency EGD was performed and revealed ulcerative defects of the stomach and duodenum with endoscopic stigmata of active or recent bleeding (Forrest Ib–IIc). Active *H. pylori* infection was confirmed by subsequent histological examination of biopsy specimens in all three patients. All patients received *H. pylori* eradication therapy in accordance with clinical guidelines.

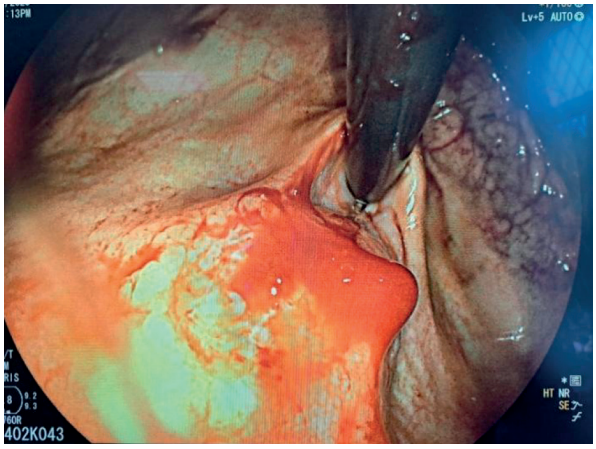

***Figure 1.*** Endoscopic picture of bleeding from a subcardiac gastric ulcer (Forrest Ib) in a patient with stable coronary artery disease receiving dual antiplatelet therapy

The results of the primary planned EGD in patients with stable CAD are presented in Figure 2.

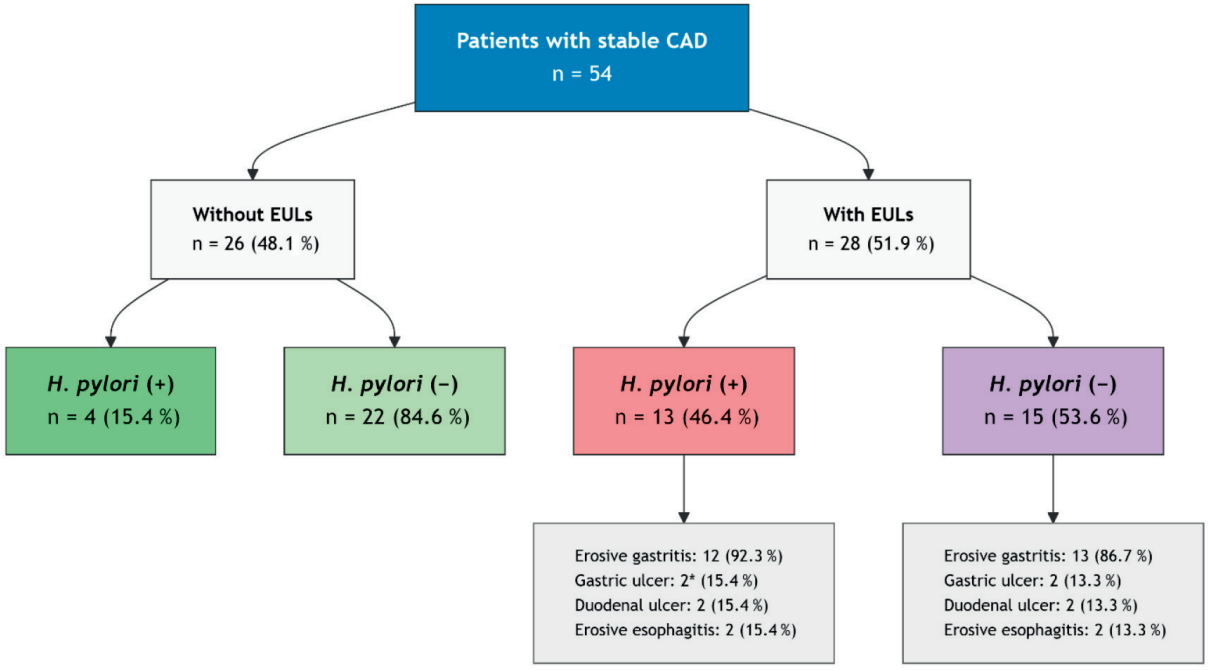


***Figure 2.*** Results of the assessment of the upper gastrointestinal mucosa condition and the frequency of *H. pylori* infection in the stable coronary artery disease group (EULs — erosive ulcerative lesions; CAD — coronary artery disease)

Erosive-ulcerative lesions were detected in every second patient (51.9 %), and 46.4 % of these cases were associated with active *H. pylori* infection ($p = 0.012$).

All patients with stable CAD in whom EGD revealed erosive-ulcerative changes ($n = 28$) and/or active *H. pylori* infection ($n = 17$) were prescribed appropriate *H. pylori* eradication therapy and proton-pump inhibitors.

Of the 56 patients with AMI (excluding the 3 cases with gastrointestinal bleeding), 53 patients underwent a strategy of preventive eradication on the basis of the results of the $^{13}$C-urea breath test.

The follow-up examination after 1 month allowed evaluation of the effectiveness of the applied treatment methods. In the stable CAD group, after eradication therapy, the frequency of erosive-ulcerative lesions statistically significantly decreased from 51.9 % ($n = 28$) to 7.4 % ($n = 4$; $p < 0.001$). This corresponds to a relative risk reduction of 85.7 % (Fig. 3).

In the AMI group, where all patients with an initially positive $^{13}$C-urea breath test received preventive eradication, 55 patients underwent follow-up EGD after 1 month (one patient had died from complications of myocardial infarction). The protocol at this stage included not only erosive-ulcerative lesions but also minimal, superficial mucosal changes.

No pathological findings were detected in 38 patients (69.1 %). Minimal and clinically insignificant changes (superficial gastritis, catarrhal bulbitis) were recorded in 7 patients (12.7 %). Erosive and ulcerative lesions (erosive gastritis, erosive bulbitis, gastric and duodenal ulcers) were noted in 10 patients (18.2 %).

Given the high efficacy of eradication therapy (92.3 %), it can be concluded that the detected mucosal changes are largely unrelated to *H. pylori* and are primarily driven by the ulcerogenic effect of antiplatelet therapy, which was mandatory in this patient cohort (Table 2).

The distribution of erosive-ulcerative lesion types differed between the groups. In all stable CAD patients, lesions were combined, whereas in the AMI group all erosive-ulcerative lesions were isolated: erosive gastritis predominated (7 cases, 70.0 %), with one case (10.0 %) each of erosive bulbitis, gastric ulcer, and duodenal ulcer. Due to the small number of cases in the stable CAD group, detailed comparison of individual lesion types was not performed.

To identify independent predictors of the development of erosive-ulcerative lesions of the upper gastrointestinal tract (including cases complicated by bleeding), multivariate logistic regression analysis was performed separately for the stable CAD and AMI groups.

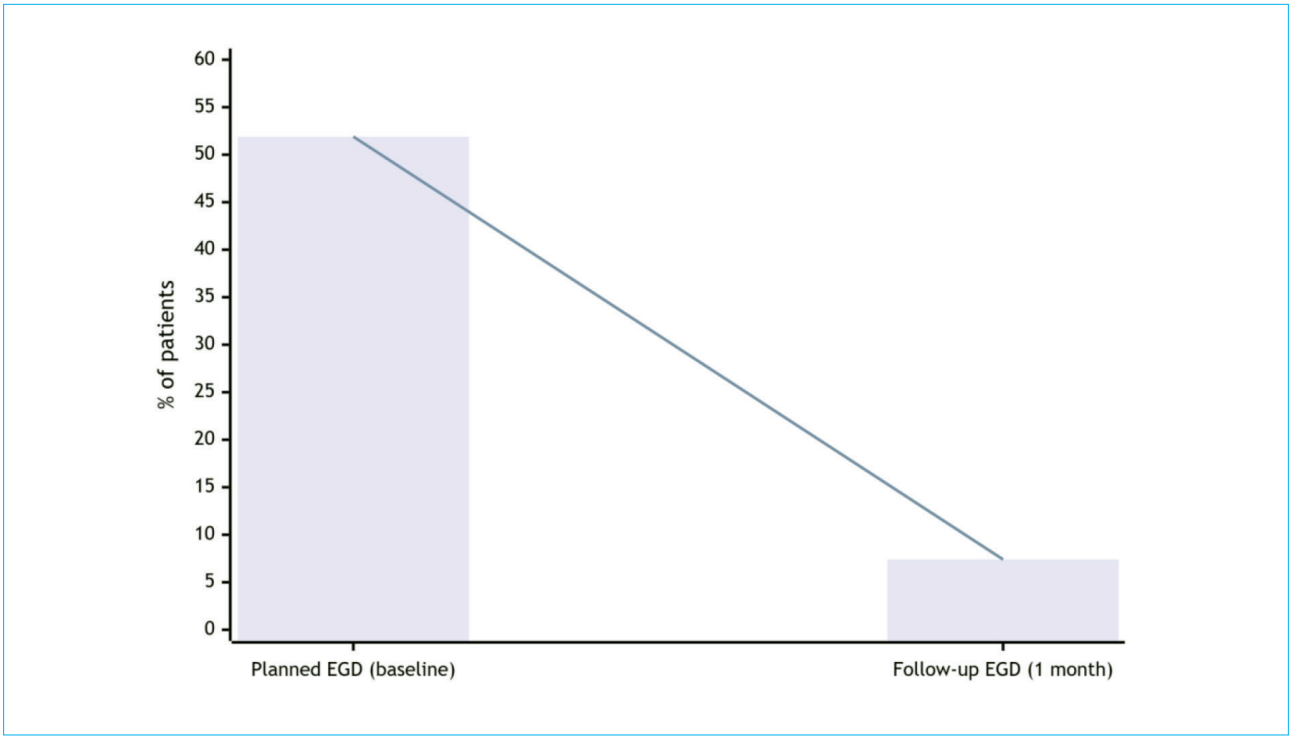


***Figure 3.*** Dynamics of the frequency of erosive-ulcerative lesions of the gastrointestinal tract in patients with stable coronary artery disease during treatment (EGD — esophagogastroduodenoscopy; EULs — erosive-ulcerative lesions)

***Table 2.*** Efficacy of *H. pylori* eradication and frequency of erosive-ulcerative gastrointestinal lesions in patients after acute myocardial infarction and with stable coronary artery disease

| Parameter | AMI group ($n$ = 56) | Stable CAD group ($n$ = 54) | $p$ |
|---|---|---|---|
| ***Baseline H. pylori infection*** | | | |
| Positive $^{13}$C-urea breath test, $n$ (%) | 26 (46.4 %) | 17 (31.5 %) | 0.122 |
| ***Eradication efficacy*** | | | |
| Successful eradication, $n/N$ (%) | 24/26 (92.3 %) | 16/17 (94.1 %) | >0.999 |
| ***Follow-up EGD results*** | | | |
| Patients who underwent follow-up EGD, $n$ (%) | 55 (98.2 %) | 54 (100 %) | 1.000 |
| Patients with any mucosal abnormality, $n$ (%) | 17 (30.9 %) | – | – |
| Patients with EULs, $n$ (%) | 10 (18.2 %) | 4 (7.4 %)* | <0.001 |
| ***Lesion characteristics (among patients with EULs)*** | | | |
| Erosive gastritis, $n$ (%) | 7 (70.0 %) | – | – |
| Erosive bulbitis, $n$ (%) | 1 (10.0 %) | – | |
| Gastric ulcer, $n$ (%) | 1 (10.0 %) | – | |
| Duodenal ulcer, $n$ (%) | 1 (10.0 %) | – | |

***Note:*** *AMI — acute myocardial infarction; CAD — coronary artery disease; EGD — esophagogastroduodenoscopy; n — number of patients with the specified symptom; N — total number of patients in a group or subgroup; * — all 4 patients with stable CAD had combined lesions (due to the small number of observations, detailing is not appropriate).*

In the group of patients with stable CAD, in whom the assessment was performed before any intervention, active *H. pylori* infection proved to be a significant and independent predictor of erosive-ulcerative lesions development. In the logistic regression model adjusted for sex and age, the presence of infection was associated with a more than 4-fold increase in risk (adjusted OR = 4.32; 95% CI: 1.22–18.2; $p$ = 0.023). In contrast, in the AMI group, where the majority of patients with detected infection received preventive *H. pylori* eradication, this factor completely lost its prognostic significance. In the multivariate model, prior and treated *H. pylori*

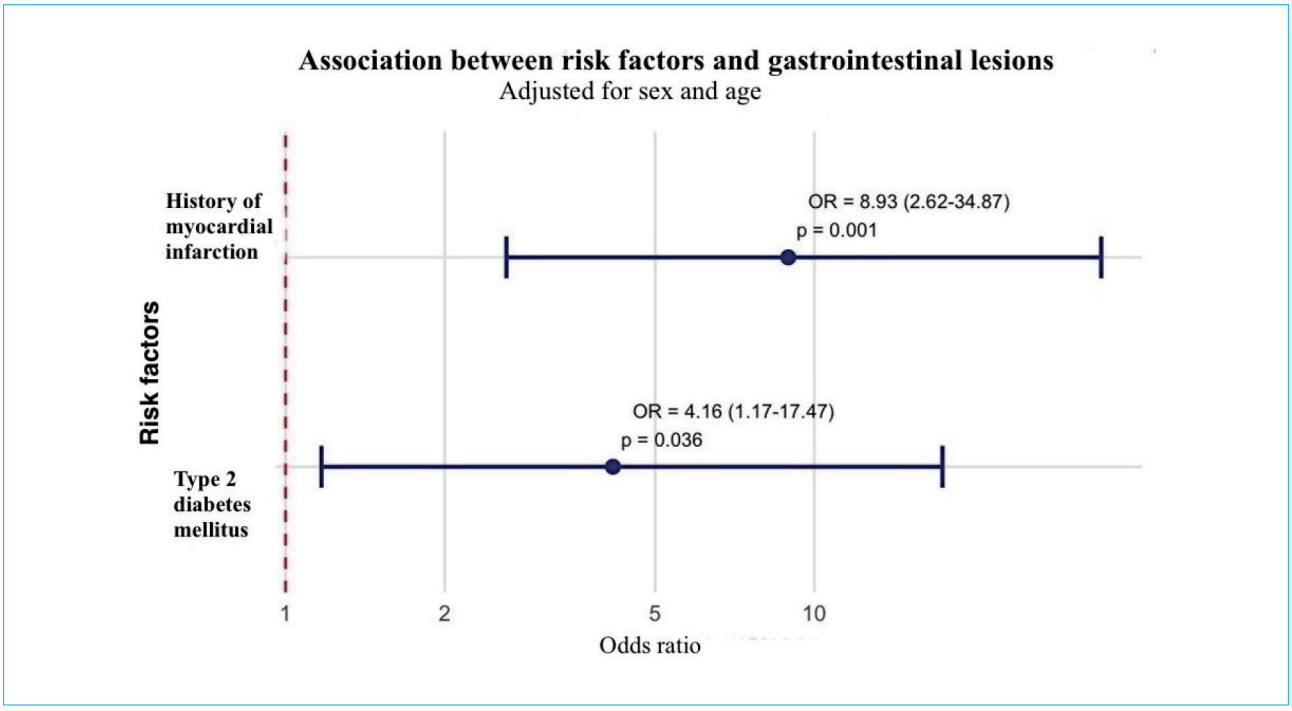


***Figure 4.*** Factors associated with the risk of erosive-ulcerative lesions and (or) gastrointestinal bleeding in patients with stable coronary artery disease (results of multivariate logistic regression analysis)

infection was not associated with an increased risk of erosive-ulcerative lesions (adjusted OR = 0.84; 95% CI: 0.22–3.20; $p = 0.79$).

In addition to *H. pylori*, multivariate analysis in the stable CAD group identified two further independent clinical predictors of increased risk of erosive-ulcerative lesions: type 2 diabetes mellitus and a history of myocardial infarction (Fig. 4).

## Discussion

The present prospective study clearly demonstrates the association between active *H. pylori* infection and the development of erosive-ulcerative lesions, including life-threatening gastrointestinal bleeding, in cardiac patients. All cases of clinically significant bleeding detected in the study occurred in patients with undiagnosed and, therefore, untreated *H. pylori* infection at the time of the event.

Data from a large randomized controlled trial HEAT show that eradication of *H. pylori* in patients taking long-term ASA leads to a statistically significant reduction in the risk of ulcer bleeding by 65 % over the first 2.5 years of follow-up (hazard ratio 0.35; 95% CI: 0.14–0.89; $p = 0.028$) [10, 11]. This is consistent with the pronounced protective effect of eradication therapy observed in the present study in patients with stable CAD (reduction in the frequency of erosive-ulcerative lesions by 85.7 %).

Apart from *H. pylori* infection, type 2 diabetes mellitus and a history of myocardial infarction were independent predictors of gastrointestinal lesions in our analysis. The combination of these three factors defines the clinical profile of a patient at high gastroenterological risk.

Type 2 diabetes is traditionally associated with an increased risk of cardiovascular complications [12]. Its role in the development of gastrointestinal lesions, identified in our analysis, is supported by data from a large meta-analysis by J. Chen et al. (2023) which demonstrated that a genetic predisposition to type 2 diabetes is an independent risk factor for a number of gastroenterological diseases, including gastric ulcer (OR = 1.12; 95% CI: 1.07–1.17; $p < 0.001$) and acute gastritis (OR = 1.11; 95% CI: 1.03–1.20; $p = 0.005$) [13]. Thus, the identified genetic link confirms that type 2 diabetes increases the risk of structural damage to gastric mucosa. Against the background of systemic inflammation and microangiopathy, the mucosa becomes more vulnerable to the action of other aggressive factors, such as *H. pylori* or antiplatelet drugs.

History of myocardial infarction is associated with a set of factors that increase the vulnerability of the gastrointestinal mucosa, including chronic intestinal ischemia due to widespread atherosclerosis (involvement of mesenteric arteries) and long-term use of aggressive antiplatelet therapy with a systemic and local ulcerogenic effect. The obtained data are consistent with the results of other studies in which coronary artery disease was confirmed as an independent risk factor for gastrointestinal bleeding, which is attributed to systemic inflammation and the obligatory antithrombotic therapy in this category of patients [14, 15].

The most notable result of the present study was that in the AMI group, despite the high efficacy of preventive eradication therapy (92.3 %), 30.9 % of patients still showed some mucosal changes in the upper gastrointestinal tract at the 1-month follow-up. At the same time, true erosive-ulcerative lesions were detected in only 18.2 % of patients, and *H. pylori* infection completely lost statistical significance as a predictor (OR = 0.84; $p = 0.79$). In these patients, non-infectious, mainly drug-related and stress-related factors come to the forefront. The most important of these is aggressive antiplatelet therapy: the majority of patients (94.6 %) received the combination of ASA with ticagrelor, which has the most pronounced ulcerogenic effect among dual antiplatelet therapy regimens. Systematic reviews and meta-analyses of randomized controlled trials demonstrate that ticagrelor, compared with clopidogrel, is associated with a statistically significant increase in the risk of gastrointestinal bleeding [16, 17]. Moreover, in the study by M.Y. Tian et al. (2023), BARC types 2, 3, and 5 bleeding occurred significantly more frequently in patients in the combination therapy group (ticagrelor + aspirin) compared with ticagrelor monotherapy (27.0 % vs. 8.6 %; $p < 0.001$) [18].

The body of data obtained calls for more in-depth investigation of pathogenesis, careful risk stratification of gastroenterological complications, and the implementation of a multidisciplinary approach (cardiologist + gastroenterologist) in the management of cardiac patients.

***Study limitations***

The results should be interpreted with due consideration of the relatively small sample size, the single-center design, and the lack of long-term data on the effect of eradication on distant cardiovascular outcomes.

## Conclusion

The present study substantiates a differentiated approach to the prevention of gastrointestinal complications in cardiac patients. In stable CAD,

active diagnosis and eradication of *H. pylori* — the main modifiable risk factor — should be the key strategy, allowing a marked reduction in erosive-ulcerative lesions. In AMI, the strategy of preventive *H. pylori* eradication is a mandatory but insufficient element of prophylaxis. The main attention must be focused on the risk factors related to aggressive antiplatelet therapy and acute stress induced injury of the gastrointestinal mucosa.

## References / Литература


1. *He C., Li Y., Jiang X., Jiang M.N., Zhao X.X., Ma S.R., et al.* Progression of gastrointestinal injury during antiplatelet therapy after percutaneous coronary intervention: A secondary analysis of the OPT PEACE randomized clinical trial. *JAMA Netw Open.* 2023;6(11):e2343219. DOI: 10.1001/jamanetworkopen.2023.43219
2. *Деева Е.С., Исайкина М.А., Трушина О.Ю., Фомин В.В., Погонин А.В., Тамкаева М.К.* Осложнения двойной антитромбоцитарной терапии со стороны желудочно-кишечного тракта у пациентов с ишемической болезнью сердца: стратификация риска, современная тактика ведения. *Терапевтический архив.* 2026;98(2):125–30. [*Deeva E.S., Isaikina M.A., Trushina O.I., Fomin V.V., Pogonin A.V., Tamkaeva M.K.* Gastrointestinal complications of dual antiplatelet therapy in patients with ischemic heart disease: Risk stratification, current management tactics. A review. *Terapevticheskii arkhiv.* 2026;98(2):125–30. (In Russ.)]. DOI: 10.26442/00403660.2026.02.203532
3. *Аверков О.В., Арутюнян Г.К., Дупляков Д.В., Константинова Е.В., Никулина Н.Н., Шахнович Р.М. и др.* Острый инфаркт миокарда с подъемом сегмента ST электрокардиограммы. Клинические рекомендации 2024. *Российский кардиологический журнал.* 2025;30(3):6306. [*Averkov O.V., Arutyunyan G.K., Duplyakov D.V., Konstantinova E.V., Nikulina N.N., Shakhnovich R.M., et al.* Acute ST-segment elevation myocardial infarction. Clinical Guidelines 2024. *Russian Journal of Cardiology.* 2025;30(3):6306. (In Russ.)]. DOI: 10.15829/1560-4071-2025-6306
4. *Parekh P.J., Oldfield E.C.* 4th, Johnson D.A. Current strategies to reduce gastrointestinal bleeding risk associated with antiplatelet agents. *Drugs.* 2015;75(14):1613–25. DOI: 10.1007/s40265-015-0455-1
5. *Ивашкин В.Т., Маев И.В., Трухманов А.С., Шептулин А.А., Симаненков В.И., Лапина Т.Л. и др.* Депрескрайбинг ингибиторов протонной помпы и выбор оптимального препарата данной группы (по результатам научного форума, состоявшегося в рамках XXVI Объединенной Российской гастроэнтерологической недели). *Российский журнал гастроэнтерологии, гепатологии, колопроктологии.* 2020;30(6):7–18. [*Ivashkin V.T., Maev I.V., Trukhmanov A.S., Sheptulin A.A., Simanenkov V.I., Lapina T.L., et al.* Deprescribing of proton pump inhibitors and choice of optimal drug of this group (based on the results of the scientific forum held at the 26th United Russian Gastroenterology Week). *Russian Journal of Gastroenterology, Hepatology, Coloproctology.* 2020;30(6):7–18. (In Russ.)]. DOI: 10.22416/1382-4376-2020-30-6-7-18
6. *Wärme J., Sundqvist M., Mars K., Aladellie L., Pawelzik S.C., Erlinge D., et al.* Helicobacter pylori screening in clinical routine during hospitalization for acute myocardial infarction. *Am Heart J.* 2021;231:105–9. DOI: 10.1016/j.ahj.2020.10.072
7. *Ивашкин В.Т., Лапина Т.Л., Маев И.В., Драпкина О.М., Козлов Р.С., Шептулин А.А. и др.* Клинические рекомендации Российской гастроэнтерологической ассоциации, Научного сообщества по содействию клиническому изучению микробиома человека, Российского общества профилактики неинфекционных заболеваний, Межрегиональной ассоциации по клинической микробиологии и антимикробной химиотерапии по диагностике и лечению *H. pylori* у взрослых. *Российский журнал гастроэнтерологии, гепатологии, колопроктологии.* 2022;32(6):72–93. [*Ivashkin V.T., Lapina T.L., Maev I.V., Drapkina O.M., Kozlov R.S., Sheptulin A.A., et al.* Clinical guidelines of the Russian Gastroenterological Association, Scientific Community for the Clinical Study of Human Microbiome, Russian Society for Prevention of Non communicable Diseases, Interregional Association for Clinical Microbiology and Antimicrobial Chemotherapy for diagnosis and treatment of *H. pylori* in adults. *Russian Journal of Gastroenterology, Hepatology, Coloproctology.* 2022;32(6):72–93. (In Russ.)]. DOI: 10.22416/1382-4376-2022-32-6-72-93
8. *Ивашкин В.Т., Маев И.В., Лапина Т.Л., Кучерявый Ю.А., Абдулхаков С.Р., Алексеева О.П. и др.* *H. pylori*-ассоциированный, постэрадикационный и нехеликобактерный гастриты: алгоритм диагностики и лечения (обзор литературы и резолюция Совета экспертов Российской гастроэнтерологической ассоциации). *Российский журнал гастроэнтерологии, гепатологии, колопроктологии.* 2024;34(3):7–23. [*Ivashkin V.T., Maev I.V., Lapina T.L., Kucheryavy Yu.A., Abdulkhakov S.R., Alekseeva O.P., et al. H. pylori*-associated, post-eradication and non-helicobacter gastritis: Algorithm of diagnosis and treatment (a literature review and resolution of the Expert Council of the Russian Gastroenterological Association). *Russian Journal of Gastroenterology, Hepatology, Coloproctology.* 2024;34(3):7–23. (In Russ.)]. DOI: 10.22416/1382-4376-2024-34-3-7-23
9. *Деева Е.С., Исайкина М.А., Трушина О.Ю., Фомин В.В., Погонин А.В., Тамкаева М.К.* Клинический случай желудочно-кишечного кровотечения на фоне приема двойной антиагрегантной терапии у пациента с ишемической болезнью сердца. *Евразийский кардиологический журнал.* 2024;4:98–103. [*Deeva E.S., Isaykina M.A., Trushina O.Yu., Fomin V.V., Pogonin A.V., Tamkaeva M.K.* Clinical case of gastrointestinal bleeding during dual antiplatelet therapy in a patient with coronary artery disease. *Eurasian Heart Journal.* 2024;4:98–103. (In Russ.)]. DOI: 10.38109/2225-1685-2024-4-98-103
10. *Hawkey C., Avery A., Coupland C.A.C., Crooks C., Dumbleton J., Hobbs F.D.R., et al.; HEAT Trialists.* Helicobacter pylori eradication for primary prevention of peptic ulcer bleeding in older patients prescribed aspirin in primary care (HEAT): A randomised, double-blind, placebo-controlled trial. *Lancet.* 2022;400(10363):1597–606. DOI: 10.1016/S0140-6736(22)01843-8
11. *Hawkey C.J., Avery A.J., Coupland C.A., Crooks C.J., Dumbleton J.S., Hobbs F.R., et al.; HEAT Trialists.* Eradication of Helicobacter pylori for prevention of aspirin associated peptic ulcer bleeding in adults over 65 years: The HEAT RCT. *Health Technol Assess.* 2025;29(42):1–62. DOI: 10.3310/LLKF7871
12. *Sattar N., Rawshani A., Franzén S., Rawshani A., Svensson A.M., Rosengren A., et al.* Age at diagnosis of type 2 diabetes mellitus and associations with cardiovascular and mortality risks. *Circulation.* 2019;139(19):2228–37. DOI: 10.1161/CIRCULATIONAHA.118.037885
13. *Chen J., Yuan S., Fu T., Ruan X., Qiao J., Wang X., et al.* Gastrointestinal consequences of type 2 diabetes mellitus and impaired glycemic homeostasis: A Mendelian randomization study. *Diabetes Care.* 2023;46(4):828–35. DOI: 10.2337/dc22-1385
14. *Patel P., Nigam N., Sengupta N.* Lower gastrointestinal bleeding in patients with coronary artery disease on antithrombotics and subsequent mortality risk. *J Gastroenterol Hepatol.* 2018;33(6):1185–91. DOI: 10.1111/jgh.14048

15. *Kou Y., Ye S., Tian Y., Yang K., Qin L., Huang Z., et al.* Risk factors for gastrointestinal bleeding in patients with acute myocardial infarction: Multicenter retrospective cohort study. *J Med Internet Res*. 2025;27:e67346. DOI: 10.2196/67346
16. *Alamzaib S.M., Maniya M.T., Hazaveh S., Cheema A.A.A., Sultan W., Qatanani A., et al.* The risk of gastrointestinal bleeding in patients taking third-generation P2Y12 inhibitors compared with clopidogrel: Systematic review and meta-analysis. *Ann Med Surg (Lond)*. 2025;87(10):6694–701. DOI: 10.1097/MS9.0000000000003821
17. *Guo C.G., Chen L., Chan E.W., Cheung K.S., Isshiki T., Wong I.C.K., et al.* Systematic review with meta-analysis: The risk of gastrointestinal bleeding in patients taking third-generation P2Y12 inhibitors compared with clopidogrel. *Aliment Pharmacol Ther*. 2019;49(1):7–19. DOI: 10.1111/apt.15059
18. *Tian M.Y., Wang X.Y., Chen F., Guo Y.F.* Comparison of ticagrelor monotherapy and ticagrelor plus aspirin among patients with acute coronary syndrome combined with high-risk of gastrointestinal bleeding after percutaneous coronary intervention: A retrospective cohort study. *J Cardiovasc Pharmacol*. 2023;82(4):327–32. DOI: 10.1097/FJC.0000000000001461

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