Pro Index | Journal of Innovation in Educational and Social Research
Volume: 3 Issue: 10 Year: 2025
ISSN: 2992-894X | http://journals.proindex.uz

# In Cases of Ischemic Heart Disease, Mucosal Alterations in the Upper Gastrointestinal Tract

**Raupov Abdurahmon Ortiq o'g'li**
Bukhara State Medical Institute named after Abu Ali ibn Sina, Uzbekistan, Bukhara, st., A. Navoi., raupov.abdurahmon@bsmi.uz

***Resume:*** This article examines the intricate connection between upper gastrointestinal tract (UGIT) abnormalities and ischemic heart disease (IHD), especially in individuals undergoing antiplatelet therapy or with atrial fibrillation. In the co-development of cardiovascular and gastrointestinal disorders, it highlights the impact of lifestyle variables, psycho-emotional stress, and Helicobacter pylori infection. For individuals with IHD and concomitant UGIT disorders, the study emphasizes the value of early diagnosis, endoscopic examination, and customized preventative measures.

***Keywords:*** ischemic heart disease, gastrointestinal lesions, helicobacter pylori , antiplatelet therapy, psycho-emotional stress

Ischemic heart disease (IHD) is a cardiac pathology primarily caused by atherosclerosis of the coronary arteries, which under certain conditions leads to a mismatch between the myocardium's demand for blood supply and the possible volume of blood flow through the affected arteries. Atherosclerosis is characterized by excessive lipid deposition in the vessel walls. This pathological process affects not only the coronary arteries but also the arteries supplying other organs. Lesions of the superior mesenteric artery result in atrophic processes in the stomach, typical of elderly and senile individuals, and the formation of trophic gastric ulcers. Intestinal ischemia due to atherosclerosis of the mesenteric arteries most often manifests as hypokinesia and hypotonia of the intestinal loops, potentially progressing to dynamic intestinal obstruction in severe cases. Prolonged and pronounced insufficiency of intestinal blood supply causes metabolic disturbances in the intestinal wall, dysbiosis, development of hypovitaminosis, and wasting. It is known that the atherosclerotic process begins as early as 20–30 years of age and subsequently progresses at a rate depending on various factors, including risk factors. Pathological-anatomical data indicate that 20% of individuals aged 26–30 years already have coronary artery atherosclerosis [1]. IHD is one of the most common diseases, posing significant medical, social, and economic challenges. Simultaneously, IHD is a leading cause of both temporary and permanent disability, as well as mortality in developed countries worldwide. According to Rosstat data, the number of fatal cases due to IHD in 2014 was 134.1 per 100,000 men and 18.0 per 100,000 women, and in 2015 it was 125.2 per 100,000 men and 16.3 per 100,000 women [2].

In patients with ischemic heart disease (IHD), there is an observed increase in the incidence of acute erosive and ulcerative lesions of the upper gastrointestinal tract (UGIT), which is caused not only by trophic disturbances of the tissues but also by the extensive use of various medications for the treatment of IHD. These drugs contribute to an increase in the acid-peptic factor and a decrease in mucus secretion, which protects the mucosal lining from these effects. In particular, the modern therapeutic strategy of antiplatelet therapy (APT), based on extensive experience with the use of acetylsalicylic acid (ASA), oral indirect anticoagulants, thienopyridines, and their combinations, plays a significant role. The use of standard doses of ASA (75–150 mg) or vitamin K antagonists increases the risk of bleeding by 1.8 times, while the use of clopidogrel increases this risk by 1.1

times. Dual APT is associated with a higher relative risk of bleeding: the combination of ASA and dipyridamole increases the risk by 2.3 times; ASA and indirect anticoagulants — by 5.3 times; ASA and clopidogrel — by 7.4 times [3].

A high incidence of upper gastrointestinal tract (UGIT) lesions is noted in patients with atrial fibrillation (AF), particularly when combined with ischemic heart disease (IHD). This combination is observed in 65.2% of cases [4]. AF is a type of supraventricular tachycardia characterized by chaotic atrial activity, with atrial contractions occurring at a rate of 350–700 beats per minute. Arrhythmias develop due to structural changes in the cardiac conduction system in the context of heart diseases and/or under the influence of autonomic, endocrine, electrolyte, and other metabolic disorders, intoxications, and certain medications [5]. The problem of cardiac rhythm disturbances remains as relevant as that of IHD. Rhythm disturbances are frequently encountered in everyday medical practice, and the spectrum of cardiac arrhythmias is extremely broad in terms of etiology, clinical manifestation, diagnosis, and prognosis. At the same time, rhythm disturbances are among the most frequent and significant complications of a wide variety of diseases.

The estimated prevalence of AF is 3.2 per 1,000 population. It can occur at any age but is most common in individuals aged 70–79. The total number of AF cases in Russia is approximately 2.5 million. Arrhythmias often determine the prognosis for both work capacity and life. Annual hospitalizations may reach 1.227 million, with an average hospital stay of 6.9 bed-days per AF case, an estimated mortality rate of 1%, and the total number of days of incapacity for work may reach 3.386 million per year [5]. Isolated rhythm disturbances, even when they do not significantly impair hemodynamics, may be poorly tolerated by patients and lead to significant lifestyle changes. The issue of assessing UGIT mucosal lesions remains insufficiently studied, especially in patients with rhythm disturbances on the background of IHD. Addressing this issue is an urgent task for the preventive treatment of UGIT lesions both in isolated IHD and in IHD combined with AF.

To study the features of mucosal lesions of the upper gastrointestinal tract (UGIT) in patients with chronic ischemic heart disease (CIHD) without rhythm disturbances and those complicated by atrial fibrillation (AF), the most informative diagnostic method is endoscopy, particularly esophagogastroduodenoscopy (EGD). EGD is a probing method that, using a flexible endoscope, allows for visualization of the esophagus, stomach, and duodenum. It enables the identification of anatomical changes in these organs, assessment of their functional status, and collection of biopsy samples to determine the morphological characteristics of the pathology and, often, its etiology. In patients with CIHD both without rhythm disturbances and those with AF, various mucosal changes of the UGIT are observed, including inflammatory, hyperplastic, metaplastic, erosive, and ulcerative processes [6].

Such changes may be caused by various factors, one of which is antiplatelet therapy (APT). Nonsteroidal anti-inflammatory drugs (NSAIDs), including acetylsalicylic acid (ASA), are known to contribute to the development of erosions and ulcers in the stomach and duodenum in patients. These lesions may be complicated by gastrointestinal bleeding (GIB), which poses an additional risk to the prognosis and survival of patients. According to various authors, NSAID therapy more than doubles the risk of GIB in any population group in Europe. In 20–25% of individuals on long-term NSAID therapy, acute ulcers develop, and in more than 50% of cases, erosive gastroduodenitis is observed. Additionally, 53.5% of all patients admitted with a diagnosis of acute GIB had been taking NSAIDs [4,10]. The likelihood of bleeding is reportedly highest during the first month of NSAID use, gradually decreases by the end of the first year, and increases again in old age.However, this is not the only factor contributing to UGIT lesions in IHD. The prognosis in patients with IHD complicated by bleeding of varying severity is worse due to a reduction in coronary reserve. This is further aggravated by anemia, disturbances in hemostasis regulation, impaired homeokinesis, and central hemodynamics, along with disrupted renal nitrogen-excreting function and elevated serum creatinine levels.The issue of comorbidity between IHD and gastrointestinal diseases has attracted researchers' attention since the late 20th century. In Northern Ireland and the UK, it was noted that Helicobacter pylori infection was associated with a higher

prevalence of IHD. Helicobacter pylori, an established etiological factor in gastritis, peptic ulcer disease, and gastric cancer, may also participate in the pathogenesis of chronic vascular diseases. This is due to the shared pathogenic mechanisms of atherosclerosis in IHD and inflammation in H. pylori infection. Over the past 20 years, numerous studies have investigated a possible causal relationship between Helicobacter pylori and IHD, although no definitive conclusion has been reached. Various pathogenic mechanisms have been explored through which H. pylori infection could increase cardiovascular risk. It has been established that chronic H. pylori infection, acquired in youth, increases the lifetime risk of developing IHD. Furthermore, the risk of IHD complications in the presence of Helicobacter pylori infection is significantly elevated, independent of other trigger factors. However, current evidence is still insufficient to recognize the infection as a definitive risk factor for IHD. Should its etiological role be confirmed, new opportunities may emerge for the prevention of IHD risk factors [6,7].

A comparative study of the morphology of antral gastric mucosa biopsy specimens from patients with stable exertional angina and those without ischemic heart disease (IHD) revealed significant differences only in the degree of mononuclear cell infiltration, which was significantly more frequent in the control group. It should be noted that patients with stable exertional angina were more likely to have atrophy of the antral gastric mucosa compared to those without IHD. Helicobacter pylori was detected slightly more frequently in IHD patients by both cytological (70% vs. 57.3%) and histological (84% vs. 73.3%) methods. Other morphological parameters of the antral gastric mucosa were nearly identical between the two groups [8].

The coexistence of these pathologies is also facilitated by the widespread prevalence of smoking in the population, living conditions, and age. One of the most significant risk factors for IHD and its complications is an elevated level of psycho-emotional stress, commonly associated with the so-called stress-coronary personality profile (Type A). This profile includes personality traits such as anger, depression, constant anxiety, aggressiveness, excessive ambition, frequent psychological stress, and a lack of family support and understanding. Psycho-emotional stress and these character traits, inherent to the stress-coronary profile, are accompanied by a high release of catecholamines into the bloodstream, which increases myocardial oxygen demand, heart rate, and blood pressure. This contributes to the development of myocardial ischemia, increased coagulation activity, and the deposition of cholesterol plaques in the blood vessels, ultimately leading to the development of IHD [9].

**Conclusion.** The coexistence of ischemic heart disease (IHD) and upper gastrointestinal tract (UGIT) lesions presents a significant clinical challenge, especially in the context of antiplatelet therapy and atrial fibrillation. Endoscopic evaluation reveals a high prevalence of erosive and ulcerative changes, often exacerbated by NSAIDs, Helicobacter pylori infection, and psycho-emotional stress. Understanding the multifactorial nature of this comorbidity is essential for developing effective strategies for prevention and management.

## REFERENCES

1. Nikulina NN. Modern features post-mortem diagnostic and statistical registration of coronary artery disease as the cause of death. Perm medical Journal 2011; 28 (2): 133-40. (in Russ.) Никулина Н. Н. Современные особенности посмертной диагностики и статистической регистрации ишемической болезни в качестве причины смерти. Пермский медицинский журнал 2011; 28 (2): 133-40.
2. Clinical practice guidelines for the treatment of stable ischemic heart disease 2013. Russ J Cardiol 2014; 7: 50-3. (in Russ.) Клинические рекомендации по лечению стабильной ишемической болезни сердца 2013. Российский кардиологический журнал 2014; 7: 50-3.
3. Sumarokov AB, Burakovsky LI, Teacher IA. Bleeding in patients with coronary heart disease during therapy with antiplatelet agents. CardioSomatica 2011; 3: 29-35. (in Russ.) Сумароков А. Б., Бурячковская Л. И., Учитель И. А. Кровотечения у больных с ишемической

болезнью сердца при проведении терапии антиагрегантами. CardioСоматика 2011; 3: 29-35.

4. Vasiliev YuV. Of Acid-related gastrointestinal diseases and coronary heart disease. Lechaschi Vrach 2006; 1: 50-5. (in Russ.) Васильев Ю. В. Кислотозависимые заболевания ЖКТ и ишемическая болезнь сердца. Лечащий врач 2006; 1: 50-5.
5. Vaneeva OS, Sidorenko BA. The Modern treatment strategies and the identification of predictors of recurrence of atrial fibrillation. Medical journal "Difficult patient" 2012; 10 (4): 17-23. (in Russ.) Ваниева О. С., Сидоренко Б. А. Современные стратегии лечения и выявление предикторов рецидива мерцательной аритмии. Медицинский журнал "Трудный пациент" 2012; 10 (4): 17-23.
6. Simonova ZhG. Monograph "Comorbid conditions: Ischemic heart disease and gastroduodenal pathology". Kirov: Publishing house "Raduga-PRESS" 2015. 130 p. (in Russ.) Симонова Ж. Г. Монография "Коморбидные состояния: Ишемическая болезнь сердца и гастродуоденальная патология". Киров: Изд-во "РадугаПРЕСС" 2015. 130 с.
7. Zubareva AA, Chicherina EN. Acute coronary syndrome and pathology of the upper gastrointestinal tract. Lechaschi Vrach 2016; 12: 77-81. (in Russ.) Зубарева А. А., Чичерина Е. Н. Острый коронарный синдром и патология верхних отделов желудочно-кишечного тракта. Лечащий врач 2016; 12: 77-81.
8. Osadchy VA, Sergeev AN, Rasskazov YV, et al. Clinical and morphological features of gastroduodenal erosions and ulcers associated with unstable angina, and the value of physiological disorders in their Genesis. Russian Journal of Cardiology 2012; 5: 34-9. (in Russ.) Осадчий В. А., Сергеев А. Н., Рассказова Ю. В. и др. Клиникоморфологические особенности гастродуоденальных эрозий и язв, ассоциированных с нестабильной стенокардией, и значение физиологических нарушений в их генезе. Российский кардиологический журнал 2012; 5: 34-9.
9. Health care in Russia. 2015. Stat.sat./Rosstat. M., W-46, 2015. p. 29. (in Russ.) Здравоохранение в России. 2015. Стат.сб./Росстат. М., З-46, 2015. с. 29.
10. Vertkin AL, Zairatyants OV, Vovk EI, Kolobov SV. Treatment and prevention of gastrointestinal bleeding during exacerbation of ischemic heart disease. Pharmateca 2007; 15: 54-60. (in Russ.) Верткин А. Л., Зайратьянц О. В., Вовк Е. И., Колобов С. В. Лечение и профилактика желудочно-кишечных кровотечений при обострении ишемической болезни сердца. Фарматека 2007; 15: 54-60.