

ANTITHROMBOTIC THERAPY IN CARDIOLOGICAL PATIENTS

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**Abstract:** Prevention of thromboembolic complications, which are a key moment in the development of cardiovascular complications in coronary heart disease, involves long-term use of antithrombotic drugs. A retrospective analysis of ACS patients showed low adherence to treatment with antithrombotic drugs. Probably, the development of resistance to therapy also contributes to the appearance of myocardial infarctions.

**Key words:** myocardial infarction, thromboembolic complications, antithrombotic drugs, treatment adherence, treatment resistance.

### Introduction

Thrombosis plays a key role in the pathogenesis of coronary artery disease. Thromboembolic complications occupy an important place in the structure of the causes of cardiovascular mortality. Atherosclerotic vascular damage with violation of intimal integrity, slowing of blood flow, imbalance of the coagulation and anticoagulation system and violation of rheological parameters of blood lead to the formation of a blood clot and, as a consequence, partial or complete occlusion of the vessel lumen. For the prevention and treatment of coronary heart disease, antiplatelet agents are used – drugs that prevent thrombosis due to a decrease in the functional activity of platelets. Many medications have antiplatelet properties – aspirin, thienopyridine derivatives (clopidogrel, ticlopidine), curantil, etc. The basis of modern antiplatelet therapy is acetylsalicylic acid and thienopyridines. The most widely used acetylsalicylic acid, which has been used in medical practice for more than 100 years. Recognition of atherothrombosis as the basis of the pathogenesis of most cardiovascular diseases, advances in the study of molecular mechanisms of thrombosis influenced the development of antithrombotic therapy and contributed to the emergence of new drugs, in particular, tikogrelol. Modern antithrombotic drugs affect the process of blood clotting, suppressing the function of platelets and inhibiting the cascade of coagulation, and are also able to destroy formed blood clots and restore the patency of arteries. From the standpoint of medical evidence in patients with stable manifestations of atherothrombosis, the effectiveness of antiplatelet drugs has been

proven: aspirin, clopidogrel, ticlopidine, a combination of slowly the release form of dipyridamole and aspirin, as well as vitamin K antagonists.

The purpose of our study was to study the results of antithrombotic therapy in patients with acute coronary syndrome (ACS) who were admitted in Samarkand in the 1st half of 2021.

### **Materials and methods**

Under our supervision and treatment were 218 ACS patients aged 47 to 76 years old (average age  $63.2 \pm 1.8$ ), of which 59 are women and 159 men who were admitted in an emergency with pain syndrome to the GCC in 2014. All patients underwent ECG, EchoCG, chest X-ray, AS, general clinical and biochemical studies: lipid spectrum, glucose level, cardiomarkers (troponin, myoglobin, BNP, D-dimer).

### **Results and discussion**

All patients were concerned about pain in the heart, behind the sternum, shortness of breath, palpitations. On the ECG, all patients showed signs of ischemia, scarring was detected in 68 patients, atrial fibrillation in 88. A day later, interpreting the results of daily monitoring of ECG, cardiomarkers and EchoCG, unstable angina was diagnosed in 142 patients and in 76 – myocardial infarction. It was carried out as well as the levels of cardiomarkers were determined. In the group of patients with myocardial infarction, patients with a previous myocardial infarction with the development of postinfarction atherosclerosis, atrial fibrillation, and according to CAG, multivessel lesions of the arteries were more common. Antithrombotic therapy was previously recommended to all patients, but only 71.6% received it. Patients taking aspirin (72-33%) comprised group 1: combination therapy (aspirin and clopidogrel) (96-44%) – group 2 and ticlopidine (48-22%) – group 3. In a comparative study, the group of patients with myocardial infarction had the highest percentage of antithrombotic drugs (90.8%) than among patients with unstable angina (61.3%). At the same time, against the background of taking antithrombotic drugs, a myocardial infarction with a multivessel lesion of the coronary arteries develops. As we can see, most of the patients were on combination therapy with antithrombotic drugs. Since the 80s of the twentieth century, acetylsalicylic acid (ASA) has been actively included in clinical practice for the prevention of myocardial infarction in patients with acute coronary syndrome (ACS). It has been proven that ASA at doses of 75-325 mg/ day significantly reduces the frequency of development myocardial infarction and sudden cardiogenic death in ACS patients, continued administration of ASA after stabilization of the condition provides a long-term positive preventive effect. At the same time, a number of independent studies have noted that the use of ASA in small doses in combination with antianginal agents in patients with coronary artery disease increases the risk of a pain-free form of myocardial ischemia, and it is also not recommended to prescribe for vasospastic angina. In recent years, the issue of resistance to ASC therapy

has been actively discussed, which is understood as the inability of ASA in some patients to adequately suppress platelet function, reduce the synthesis of thromboxane A<sub>2</sub> and / or prolong the bleeding time. The prevalence of resistance to ASA therapy, according to various researchers, ranges from 10 to 45%, which contradicts the positive clinical results of the use of ASA. One of the most likely causes of resistance is insufficient adherence of patients to treatment. The reasons for such a low adherence to aspirin treatment may be related to the risk of complications when taking them. As is known, the main clinical problem with the use of ASA is the risk of bleeding from the gastrointestinal tract, and therefore the issues of prevention of damage to the gastrointestinal mucosa when prescribing aspirin are relevant. The use of intestinal-soluble forms of ASA does not solve this problem. Aspirin is rapidly absorbed in the stomach and small intestine, its half-life in the bloodstream is only 15-20 minutes. The concentration of aspirin in plasma reaches a peak in 30-40 minutes, and the suppression of platelet function an hour after administration. Y widely the absorption of the intestinal-soluble forms of aspirin used in recent years is slowed down and the peak plasma concentration occurs only after 3-4 hours. The use of aspirin can reduce the frequency of cardiovascular death by 10-15% and the frequency of fatal cardiovascular episodes by at least 20% in patients with a high risk of atherothrombotic complications. Ticlopidine and clopidogrel are thienopyridine derivatives whose mechanism of action is associated with selective inhibition of ADP-induced platelet aggregation. Both drugs turn into liver into active metabolites. Therefore, the onset of action of thienopyridines is delayed. The need for preliminary metabolism of ticlopidine derivatives leads to a delay in the manifestation of their antiplatelet action. For a faster onset of action, the first dose should be higher. Currently, it is recommended to take 300-600 mg of clopidogrel or 500 mg of ticlopidine at the same time. Ticlopidine is an effective antithrombotic agent, but it has undesirable side effects, including the possibility of hypercholesterolemia and neutropenia, thrombocytopenia, anemia and thrombotic thrombocytopenic purpura. Clopidogrel has a better safety profile and has practically replaced ticlopidine in most clinical situations. It is rapidly absorbed and converted into an active metabolite with a plasma elimination period of 8 hours. The onset of action of clopidogrel is faster than that of ticlopidine. After the reception food bioavailability of ticlopidine increases by about 20%, when used after antacids it decreases by 20%. The absorption of clopidogrel is not affected by food intake and antacids. Suppression of ADP-induced platelet aggregation depends on the dose of clopidogrel, after taking the first dose, platelet aggregation is suppressed by 40%. Against the background of regular administration of the drug, the suppression of platelet aggregation increases, and after 7 days after the drug is discontinued, it completely disappears.

## Conclusion

The value of the load test of clopidogrel has not been definitively established and is 300-600 mg, supporting the dose in most cases is 75 mg (sometimes 150mg). Thus, a retrospective analysis of the results of the use of antithrombotic drugs in patients CHD shows low adherence to therapy. The development of myocardial infarction, especially repeated, against the background of multivessel damage to the coronary arteries, may be associated with the development of resistance to antithrombotic therapy.

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