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Assessment of Efficacy and Optimization of Antiplatelet Therapy in Patients with Ischemic Heart Disease

Mirzayeva G. P., Jabbarov O. O., Umarova Z. F., Qodirova Sh. I., Tursunova L. D., Nadirova Yu. I., Rahmatov A. M.
Tashkent medical academy

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ABSTRACT

The review article presents the problems of inadequate disaggregant response to deaggregant therapy. So far no reliable information about the frequency and causes of these complications. Cardiovascular diseases stably hold the leading position among the causes of death in the world. Today, as we know, 50% of all deaths in developed countries are caused by coronary heart disease (CHD).

It requires astronomical sums to maintain social health population both in terms of improving the quality of life and its extension. It is the great social significance of the problem and its economic side is the main engines of progress in coronary surgery.

Cardiovascular diseases steadily hold the leading position among the causes of death in the world. Today, it is known that 50% of all deaths in developed countries are caused by coronary heart disease (CHD). More than 800,000 coronary artery bypass surgeries (ACBS) and 850,000 angioplasties are performed worldwide every year. This requires astronomical sums to maintain the social health of the population both in terms of improving the quality of life and prolonging it. It is the great social importance of the problem and its economic side that are the main drivers of coronary surgery progress [1]. According to estimations of the World Health Organization (WHO), over 17 mln. people die of cardiovascular diseases in the world annually; over 7 mln. of them - from CHD. In the Russian Federation in 2004 cardiovascular diseases were the cause of more than half of all deaths - 895.4 (56.1%) cases per 1596.0 cases per 100 000 population; moreover CHD mortality was 426.8 (26.7%) cases [Y.V. Belov 1987]. In Russia more than 1 million people die annually from cardiovascular diseases, constituting almost 60% in the structure of overall mortality, and the leading place here is occupied by coronary heart disease (CHD), proceeding with periods of stable course and exacerbations. Acute coronary syndrome (ACS) without ST-segment elevation is represented by the group of unstable angina (UA). Of all forms of ACS, unstable angina is the most promising in terms of preventing adverse outcomes in exacerbation of CHD [1].

One of the pathogenetically significant disorders in patients with unstable angina is a disorder of platelet-vascular hemostasis, including biologically active systems of regulation of platelet and vascular endothelial function (thromboxane, prostacyclin, endothelium, thrombomodulin, Willebrand factor, platelet factor 4 and others). The action of acetylsalicylic acid (ASA) as a

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therapeutic and prophylactic anti-aggregation agent is based on its ability to inhibit platelet cyclooxygenase, thus inhibiting their hyperaggregation, which is the initial link in the activation of the blood coagulation system [2]. This in turn disrupts the synthesis of active cyclooxygenase metabolites of arachidonic acid (AA), in particular thromboxane and prostacyclin, changes the state of endothelium and vascular tone.

On the one hand, the effectiveness of ASA in the treatment of CHD has been proved. On the other hand, the use of this drug is carried out empirically - without taking into account individual characteristics of platelet hemostasis regulation in a particular patient and in the absence of data on the drug concentration in blood and cells directly involved in hemostasis. Lack of information about individual rates of ASA pharmacokinetics in unstable angina combined with the lack of data on the status of active metabolites of AC and other regulators of endothelial-platelet function, mediating its effect on aggregation, may be one of the possible reasons for insufficient clinical effectiveness of ASA drugs, which is important in cases of coronary pathology.

In this connection, the study of regulators of endothelial-platelet function, including AC metabolites, and their influence on the state of platelet hemostasis can be very promising from the position of establishing predictors of the effectiveness of antiaggregation therapy in patients with unstable angina pectoris.

Currently, invasive methods of myocardial revascularization have firmly taken the leading position in the treatment of coronary heart disease. In comparison with conservative drug therapy, they allow to restore the patient's ability to work more effectively and to relieve him/her from the symptoms of angina pectoris. These advantages are due to the pathogenetic nature of invasive treatment, in which obstacles to normal blood flow are removed and adequate perfusion of the heart muscle is restored [3]. Surgical and endovascular techniques in cardiology clinic have been actively used during the last several decades.

As is known, in the 80s, interventional cardiologists proposed the treatment of single-vessel coronary artery lesions with catheter-based technology. But soon similar technique was extended to patients with multiple coronary artery lesions, unfavorable forms of coronary artery lesions and affected venous grafts. This immediately intensified trends in the treatment of CHD patients towards interventional cardiology. The unambiguous advantage of catheterization technique over surgical treatment is a greater “softness” for the patient and a lower incidence of complications in the postoperative period compared to ACS performed according to the standard technique. [4].

In spite of the fact that the catheterization technique is unable to provide complete myocardial revascularization, is accompanied by increased rate of return of symptoms, requires repeated interventions, as well as the existence of evidence of limitations of certain approaches, it is increasingly used in patients with CHD.

During the following years, there were numerous empirical attempts to use dextran and dipyridamole to reduce the risk of stent thrombosis, but all these attempts1 failed and did not lead to the desired result.

The next stage of work on this task is to conduct numerous studies with different groups of drugs, as well as with different representatives of one group: disaggregants (aspirin, ticlopidine, clopidogrel, absiximab eptifibatide, tirofiban); direct anticoagulants: heparin, low molecular weight heparin; direct thrombin inhibitors: bivalirudin and hirudin, indirect anticoagulants: warfarin. [5].

However, despite the data obtained, the results of these studies remain controversial - questions about the number and timing of loading doses, the number and duration of maintenance doses, as well as the number of antiplatelet agents used after PCI. Despite standard antiplatelet therapy, thrombotic complications such as strokes, infarcts and stent thrombosis are common.

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The problem of inadequate disaggregant response to antiplatelet therapy is poorly understood. To date, there is no reliable information on the frequency and causes of these complications.

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