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ISCHEMIC HEART DISEASE. MODERN METHODS FOR EARLY DETECTION OF CHANGES IN THE BLOOD CLOTTING SYSTEM IN TENSION STENOCARDIA IN YOUNG PEOPLE

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Annotation. Ischemic heart disease is one of the main causes of death all over the world. Clinically, causing myocardial infarction is fatal in adults mainly due to atherosclerotic changes in the coronary arteries and sudden vascular spasm caused by stress in young people, in many cases for failure to provide timely treatment measures. Research conducted by the most recent epidemiological taxing professors Khan M, Hashim M, Mustafa H on global disease from 2010 to 2020 shows that cardiac ischemic diseases affect about 126 million people worldwide (1,655 per 100,000 people), accounting for 1.72% of the world's population by country and region, causing 34.4 million and 17.4 million deaths respectively. Males are more likely to be observed compared to females at present at 1,655 per 100,000 population, increasing from 1,845 by 2030. Among the countries of Eastern Europe, the highest incidence is maintained.

Key words: coronary artery disease, acute coronary syndrome, Willebrand factor, hemostasis, anticoagulants.

Relevance. Cardiologists of the Republic note that ischemic heart diseases are common, the main causes of which are the following risks that cause this disease: that is, increased arterial blood pressure (Hypertension) in 25% of the population over 18 years old, increased blood cholesterol levels-30 percent, physically poor health-50 percent, excess body weight (obesity) -40 percent, tobacco smoking among men-54 percent. In Uzbekistan , the first place in the overall structure of the causes of death among the population between the ages of 30 and 69 in 2016-2019 is occupied by diseases of the circulatory system-71.7%, cardiovascular disease, arterial hypertension and their complications (myocardial infarction, blood flow to the brain and other diseases) are their main causes. The number of diseases of the circulatory system recorded with the first diagnosis per 100 thousand inhabitants was 3000. (Eds.Res. of the committee on statistics, 2020). According to a 2023 study published in the Journal of the American Association: due to Covid-19 infection, YuIK is causing an increase among young people. (Including 60,000 young people between the ages of 18 and 49)

Coronary heart disease (CHD) is an urgent socio—medical problem and the leading cause of death in many economically developed countries [3, 6, 9]. This progressive process includes atherosclerotic damage to the coronary vascular bed and disorders in the hemostasis system (Falk, 1985; M. Davies, 1995). Chronic coronary insufficiency has periods of exacerbation — the so-called episodes of instability [8]. Depending on the severity of the process of intravascular thrombosis and the reactivity of the cardiovascular system, such nosological units as unstable angina and acute myocardial infarction are

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clinically recorded. Taking into account the pathogenetic basis, acute coronary syndrome is differentiated with ST segment elevation (occlusion of the lumen of the coronary artery of the heart) and without its elevation (preservation of antegrade blood flow). Key events in the pathogenesis of acute coronary syndrome are represented by atherosclerotic lesions of the coronary arteries, vasoconstriction and intracoronary thrombosis [1, 11], including activation of vascular thrombocytic hemostasis, plasma procoagulants, fibrinolysis system and physiological anticoagulants. In this regard, acute coronary syndrome can be considered as part of the dynamic spectrum of thrombotic complications of coronary artery pathology, requiring specific correction of the parameters of the hemostasis system with antithrombotic drugs [1, 3, 4, 9]. Anticoagulation mechanisms play a leading role in maintaining the liquid state of the blood and limiting the processes of thrombosis [1,5]. The most important physiological anticoagulants blocking the main pathways of thrombosis are antithrombin III (AT III), proteins C and S [14]. However, the importance of physiological blood anticoagulants in the development of acute coronary syndrome and their relationship with the factors causing thrombosis remain insufficiently studied. Dynamic monitoring of the state of the hemostasis system [1, 10] is necessary to assess the effectiveness of antithrombotic therapy in the treatment of diseases in the pathogenesis of which thrombogenic risk factors are present (atherosclerosis, hyperlipidemia, hyperfibrinogenemia, etc.).

Research objective. I 20.8 CHD. Early detection of changes in the blood clotting system in tension stenocardia in young people (18-49).

Materials and methods. Studies on the problem of DIC syndrome in coronary heart disease (CHD) are few and are devoted mainly to its acute form in uncomplicated or complicated myocardial infarction [1, 2, 4]. Works concerning chronic DIC syndrome in chronic forms of coronary artery disease, in particular with angina pectoris, are rare.

The main group consisted of 54 patients with angina pectoris III functional class. aged 40 to 68 years (58.2 ± 0.9 years). Of these, 41 were men, 13 were women. The duration of IHD was 65.0 ± 6.4 months. Duration of angina pectoris – 58.8 ± 6.0 months. 51 patients were diagnosed with chronic heart failure (CHF). The duration of CHF was 29.0 ± 3.0 months.

In patients with stable angina pectoris, the concentration of the Willebrand factor in blood plasma exceeded the control values, in blood plasma in patients with acute coronary syndrome, the concentration was 64.3% higher than in the control and 23.1% higher than in the first group ($p < 0.05$). An increase in the content of Willebrand factor in patients with coronary heart disease indicated damage to the vascular endothelium, more pronounced in acute coronary syndrome [7]. A retrospective analysis of the data obtained revealed that in patients with acute myocardial infarction without a Q wave, the level of Willebrand factor had maximum values before the start of heparin therapy: 174.3±3.21% ($p < 0.05$). The concentration of RFMC in blood plasma in patients of the first and second groups exceeded the control data by 25.1 and 67.2%, respectively ($p < 0.05$). A comparative analysis of the results showed that in patients with acute coronary syndrome, the content of RFMC was significantly higher (by 33.8%; $p < 0.05$) than in patients with stable angina pectoris, and this did not contradict the literature data [10], further confirming the fact of thrombinemia in exacerbation of coronary heart disease. When analyzing the content of physiological anticoagulants (AT III, proteins C and S) in the first and control groups, a significant

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decrease was found in patients with coronary heart disease. When comparing these indicators with the control, the degrees of their decrease were 7.4, 9.6 and 3.5%, respectively, which indicated an increased consumption of physiological anticoagulants in chronic coronary heart disease, indicating the possibility of chronic intravascular coagulation [10, 13]. When comparing the average values of AT III, proteins C and S obtained in the second group with those in the first and control groups, a significant increase was revealed (see Table 1). Thus, the amount of AT III in blood plasma in patients with acute coronary syndrome before the start of heparin therapy exceeded the values of AT III in patients with stable angina pectoris and healthy faces. Analysis of the content of protein C in blood plasma in the second group revealed its growth. The concentration of protein S in blood plasma was higher than that in the first and control groups, that is, there was a compensatory increase in these indicators during the period of exacerbation of coronary heart disease in response to the triggering of hypercoagulation mechanisms [9]. Correlation analysis in patients with acute coronary syndrome revealed a relationship between coagulation and anticoagulation levels of hemostasis. In the first group, there was a significant negative correlation between an increase in APTT and a decrease in AT III indicators ($r=-0.42$; $p=0.01$), in the second group a direct relationship between these changes was obtained ($r=0.47$; $p=0.01$). The study revealed a decrease in APTT to 30.3 ± 0.22 s and an increase in the amount of AT III to $111.3 \pm 5.61\%$. The values of the Willebrand factor in the first group correlated with the level of AT III ($r=0.64$; $p=0.01$), protein C ($r=0.71$; $p=0.01$) and protein S ($r=-0.82$; $p=0.01$).

In the second group, when comparing the concentration of Willebrand factor with the content of physiological anticoagulants in blood plasma, a negative correlation was obtained with AT III ($r=-0.49$; $p=0.01$), protein C ($r=-0.54$; $p=0.01$) and protein S ($r=-0.44$; $p=0.01$). Analyzing the changes in the studied parameters in the second group before and after heparin therapy (Table. 2), we have identified an increase in APTT and INR. After heparin therapy, the content of fibrinogen, Willebrand factor and RFMC significantly decreased. After heparin therapy, there was also a decrease in the amount of AT III, protein C and protein S, which indicated an increased consumption of physiological anticoagulants during heparin therapy [3]. At the beginning of the study, 12 admitted patients with acute coronary syndrome had a troponin test positive. A retrospective analysis revealed a significant initial increase in the concentration of- fibrinogen concentrations in blood plasma and Willebrand factor compared with those in other patients (5.8 ± 1.17 g/l and $174.3\pm3.21\%$, respectively), and after heparin therapy, the degree of decrease in these indicators was much lower than in patients with unstable angina (3.2 ± 1.89 g/l and $150.7\pm3.52\%$, respectively; $p<0.05$). After anticoagulant therapy in troponin-positive patients, the level of consumption of physiological anticoagulants (decrease in the amount of AT III, proteins C and S) was higher than in other examined patients of this group ($p<0.05$). The content of physiological anticoagulants in their blood plasma was 90.7 ± 3.09, 104.1 ± 2.82 and $89.8\pm3.14\%$, respectively. Thus, in acute myocardial infarction without a Q wave, more pronounced disorders of the hemostasis system developed. In patients with coronary heart disease, there was a thrombogenic shift in the parameters of the hemostasis system towards hypercoagulation, most pronounced in patients with acute coronary syndrome, which is confirmed by literature data on the presence of chronic intravascular coagulation in patients with coronary heart disease [12, 13, 15], which acquires signs of acute course in acute coronary syndrome [6, 9].

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Conclusions.

1. In patients with coronary heart disease, changes in the platelet-vascular hemostasis system are characterized by a significant increase in the concentration of Willebrand factor, RFMC, and fibrinogen in blood plasma, most pronounced in acute coronary syndrome, which can serve as a noninvasive indicator reflecting the degree of progression of coronary heart disease.

2. In patients with acute myocardial infarction without a Q wave, the amount of Willebrand factor, RFMC and fibrinogen in blood plasma was initially significantly higher than in other patients, which indicated more pronounced disorders of the hemostasis system.

3. In patients with coronary heart disease, stable low concentrations of AT III, proteins C and S were noted in the functioning of anticoagulation and fibrinolysis systems, which, combined with increased indicators of the blood coagulation system, allows us to state the presence of hypercoagulation in the vascular bed. In acute coronary syndrome, a compensatory increase in the level of physiological anticoagulants was noted in response to an increase in the concentration of parameters of the coagulation link of hemostasis.

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