Problems of hemostasis in patients with persistent atrial fibrillation on the background of coronary heart disease (review article)

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ABSTRACT

Ischemic heart disease (CHD) is still the most common disease worldwide, including Uzbekistan [1,2]. In terms of prevalence in the population after extrasystole, atrial fibrillation (AF) takes 2nd place. According to the data of domestic scientists of our country, among the population of Uzbekistan, cardiovascular diseases are the most common cause of death at 56%, and disability - 25%, which causes significant harm to the health of the nation and the state budget. Based on the results of population studies, about 11% of the population of the Republic over the age of 40 suffer from coronary artery disease [3]. The purpose of this study was to study new scientific literature and modern views on changes in the hemostasis system in coronary artery disease and ventricular fibrillation.

Keywords: coronary heart disease, hemostasis, coagulogram, atrial fibrillation, arhythmia.

In clinical practice, atrial fibrillation (AF) is considered the most common arrhythmia and in the adult population in the general population is 3% [**4**].

It is believed that AF is an independent risk factor for cardiovascular disease, because. cardiac arrhythmia leads to a deterioration in the quality of life of patients, the possibility of thromboembolic complications, leading to sudden cardiac death.

The prevalence of AF (2%) has now doubled compared to previous decades. The prevalence of AF varies with age, gender, and in the presence of structural heart disease [5,6,7].

Over the past 2 decades, coronary artery disease with atrial fibrillation has remained one of the topical health issues and serious causes in economically developed countries. The epidemiology of AF is growing due to the increased ability of our society to treat chronic cardiac and non-cardiac diseases, to diagnose AF at an early stage [8].

Purpose of the study: to study the problem of hemostasis in patients with persistent atrial fibrillation against the background of coronary heart disease.

Introduction. The origin of the formation of platelet aggregates is as follows. When a vessel is damaged, collagen is exposed on its wall, which is both a substrate and an activator of platelet aggregation. Platelets adhere to the damaged endothelium of the vascular wall using specific collagen receptors, which is one of the triggers in the development of a parietal thrombus in the coronary arteries. Subsequently, platelets activate each other, forming platelet thrombi [9,10].

So, scientists Falk and M. Davies noted in their works that the progressive process includes

not only atherosclerotic lesions of the coronary vascular bed, but also disturbances in the hemostasis system [11].

Changes in blood viscosity in the development of thrombosis and embolism are quite diverse. According to studies by Horstkotte D., Petersen P. and co-authors, in patients with coronary heart disease, atrial fibrillation in the form of a change in hemostasis in the atria, blood stasis is noted, its fluidity decreases, which can be observed with ultrasound, which is a predictor of thrombosis [12,13].

According to the analyzes of Khairy M., Yoshinoa Satoshi et al., associated with changes in the hemostasis system in patients with atrial fibrillation and their contribution to the risk of thromboembolic complications (TEC), indicates the need for a detailed study of all links of hemostasis [14,15].

In the results of fundamental research Kamphuisen P.W., et al. (2008), Schulz B., et al. (2008), Siller-Matula J.M., et al. (2010) describes the beginning of pathogenesis leading to the development of hemostasis disorders, and disturbances in this system lead to the development of thrombosis [16,17,18].

Serebryanaya N.B. et al. (2018) emphasize that platelets, being metabolically active cells, play a significant role in a number of physiological and pathological processes, i.e. in angiogenesis, the implementation of inflammatory and reparative processes. A defect in the above functions entails an increased risk of bleeding, and vice versa, thrombosis [19]. So, Ogurkova O.N. et al., 2021, selected 2 groups of patients with paroxysmal and persistent forms of AF, taking β-blockers (β-blockers) and not taking β-blockers. As a result of their study, it was noted that in the group with persistent atrial fibrillation not taking β-blockers, the most pronounced increase in spontaneous platelet aggregation was observed. A change in the composition of the plasma, with an increase in the level of biologically active substances in the blood and metabolic disorders, leads to an increased ability of platelet aggregation [20].

During the study, scientists noted that platelet dysfunction in atrial fibrillation is a demanded task for developing an individual approach to the choice of antithrombotic therapy in the future [20].

According to Kamath S. and Nathan P., 2002 and 2016, patients with AF have an excessive amount of catecholamines in the blood, activation of the sympathoadrenal system and cell aggregation activity can lead to cell aggregation and adhesion [21,22].

Japanese scientists noted in their study that a decrease in catecholamine damage to the endothelium contributes to the normalization of endothelial function, an increase in NO content with the restoration of angioaggregant properties [23,24]. On the contrary, in 1997 Li-Saw-Hee F.L. and his team noted that in patients with paroxysmal and persistent forms of AF, at a concentration of adrenaline in the reaction medium of 2.5 μg / ml, there is a pronounced hypoadrenergic reactivity of platelets, with or without β-blockers, without affecting platelet aggregation. So, with prolonged heart rhythm disturbance, intracardiac hemodynamics and activation of the blood coagulation system are disturbed [25,26]. Russian and foreign scientists came to the same opinion that patients with atrial fibrillation are subject to increased aggregation of formed elements, leading to a change in the functional activity of platelets and a violation of the functional activity of cells [27,28]. Adrenaline binds to adrenoreceptors on the cell surface, releasing ADP from dense granules, which through the purinergic receptors P2Y1 and P2Y12 on the platelet surface, with the participation of the conjugation of α2-adrenergic receptors and P2Y12 purine receptors with the Gi protein of signaling pathways, leads to an increase in the functional activity of platelets [29,30], which leads to platelet hyperadrenoreactivity under the action of high concentrations of adrenaline in vitro.

Even Mondillo S. and a number of other authors, in the early 2000s, mentioned in scientific publications about endothelial dysfunction an increase in biomarkers - von Willebrand factor, atrial natriuretic peptide, manifested by activation of the blood coagulation system and a decrease in its fibrinolytic activity, which is interconnected with disorders in the hemostasis system

[31,32]. Numerous SPAF-III studies have shown an increase in the level of fibrinogen, D-dimer, the thrombin-antithrombin III complex, elevated values of the antigen of tissue plasminogen activator and its type 1 inhibitor [33,34,35,36,37,38].

In turn, D-dimer is a product of fibrin destruction, indicating the activation of blood coagulation, but was not included in the number of independent predictors of thrombosis [39].

Conclusion. Thus, the issue of prevention of thromboembolic complications remains open, antithrombotic prophylaxis in atrial fibrillation is the most important topic for study and implementation in screening monitoring. Platelet dysfunction in atrial fibrillation is a demanded task for developing an individual approach to the choice of antithrombotic therapy in the future.

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