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Coagulation System Difficulties in Patients with Atrial Fibrillation

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Abstract In terms of prevalence in the population after extrasystole, atrial fibrillation (AF) ranks 2nd. Research by Mahmood S.S. and Wyndham C.R. demonstrates a large increase in patients: at the age of 65, AF is diagnosed in 3-5%, and over 80 years it is detected in 5-15% [1,2]. According to Kurbanov R.D., 2007 and co-authors, in 56% of Uzbekistan, cardiovascular diseases are a common cause of mortality, and disability - 25%, which harms the health of the nation and the state budget. Based on the results of population studies, among the population of the Republic about 11% over the age of 40 suffer from coronary heart disease [3]. It was established that 10.8% of those examined, suffering from coronary heart disease, in 1.5% of cases suffered myocardial infarction, angina pectoris - 4.4%, 4.9% are painless forms, and 2/3 of patients did not even know about the presence of pathology [4].

Keywords Persistent atrial fibrillation, Blood fluidity, Arrhythmia, Heart disease

1. Introduction

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

Currently, the prevalence of AF (2%) has doubled against the background of intellectual indicators. The prevalence of AF changes with age, impairment and in the presence of structural heart disease [5,6,7].

Over the past 2 decades, coronary heart disease with atrial fibrillation remains one of the pressing 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 early stages [8].

Purpose of the study: study of the problem of hemostasis in patients with persistent atrial fibrillation.

The formation of platelet clots occurs 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 of the coronary arteries. Subsequently, platelets activate each other, forming platelet thrombi [9,10].

Thus, scientists Falk (1985) and M. Davies (1995) 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 manifested in a variety of ways. According to the research of Horstkotte D., Petersen P. and co-authors, in patients with coronary heart disease atrial fibrillation in the form of changes in hemostasis in the atria, blood stasis is noted, its fluidity decreases, which can be observed during ultrasound examination, which is a predictor of thrombus formation [12,13].

According to the analysis of Khairy M., Yoshinoa Satoshi and co-authors, the changes associated with the hemostasis system in patients suffering from atrial fibrillation and their contribution to the risk of thromboembolic complications (TEC) indicate the need for a detailed study of all links of hemostasis [14,15].

The results of fundamental studies by Kamphuisen P.W., et al. (2008), Schulz B., et al. (2008), Siller-Matula J.M., et al. (2010) describe the onset of pathogenesis leading to the development of hemostasis disorders, and disorders in this system lead to the development of thrombosis [16,17,28].

Serebryanaya N.B. and co-authors (2018) emphasize that platelets, being metabolically active cells, play a significant role in a number of physiological and pathological processes, i.e. in angiogenesis, implementation of inflammatory and reparative processes. A defect in the above functions entails an increased risk of bleeding, and vice versa, thrombus formation [19]. Since the issue of TEO prevention remains open, antithrombotic prophylaxis in atrial fibrillation is the most important topic for study and implementation in screening

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monitoring [20].

Thus, Ogurkova O.N. and co-authors, 2021, selected 2 groups of patients with paroxysmal and persistent forms of AF, taking β-blockers (β-AB) and not taking β-AB. As a result of their study, it was noted that in the group with persistent atrial fibrillation not taking β-AB, the most pronounced increase in spontaneous platelet aggregation was observed. Changes in plasma composition, with an increase in the level of biologically active substances in the blood and metabolic disorders lead to an increased ability of platelet aggregation [21].

During the study, the scientists noted that platelet dysfunction in atrial fibrillation is a sought-after task for developing an individual approach to the choice of antithrombotic therapy in the future [21].

According to Kamath S. and Nathan P., 2002 and 2016, patients with AF have an excess of catecholamines in the blood, activation of the sympathoadrenal system and cell aggregation activity can lead to cell aggregation and adhesion [22,23].

Japanese scientists noted in their study that a reduction 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, with an adrenaline concentration in the reaction medium of 2.5 μg/ml, there is pronounced hypoadrenoreactivity of platelets, both with and without taking β-AB, without affecting platelet aggregation. Так, при длительном нарушении ритма сердца нарушается внутрисердечная гемодинамика и активация системы свертывания крови [25,26]. Russian and foreign scientists have come to the same conclusion that patients with atrial fibrillation are subject to increased aggregation of formed elements, leading to changes in the functional activity of platelets and disruption of the functional activity of cells [27,28]. Adrenaline binds to adrenergic receptors on the cell surface, resulting in the release of ADP from dense granules, which through the purinergic receptors P2Y1 and P2Y12 on the platelet surface, with the participation of the coupling of α2-adrenergic receptors and P2Y12 purine receptors with Gi-protein signaling pathways, leads to an increase in the functional activity of platelets [29,30], which leads to hyperadrenoreactivity of platelets under the influence of high concentrations of adrenaline in vitro.

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 the activation of the blood coagulation system and a decrease in its fibrinolytic activity, which is associated with disorders in the hemostasis system [31,32]. Numerous studies of SPAF-III have shown an increase in the level of fibrinogen, D-dimer, the thrombin-antithrombin III complex, and elevated values of tissue plasminogen activator antigen and its inhibitor type 1 [33,34,35,36,37,38].

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

2. Conclusions

Impaired platelet function in atrial fibrillation is a sought-after task for the development of an individual approach to the choice of antithrombotic therapy in the future [21], just as the role of systemic rheological changes remains virtually unexplored.

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