CLINICAL FEATURES OF COMBINATION OF MYOCARDIAL INFARCTION AND ACUTE STROKE

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Cardiovascular and cerebrovascular diseases are the most pressing medical and social problems in the world. The increase in the prevalence of vascular diseases, which has been noted in recent years, has led to an increase in the incidence of acute myocardial infarction and cerebrovascular accident. Men suffer from ischemic heart disease much more often than women. At the age of 40-60, myocardial infarction is 3-5 times more common in men due to the earlier (10 years earlier than in women) development of atherosclerosis. After 55-60 years, the incidence among persons of both sexes is approximately the same. The mortality rate in acute myocardial infarction (AMI) is 30-35%. Statistically, 15-20% of sudden deaths are caused by AMI. It is known that the mortality rate in acute stroke (AS) in the early stages (30 days) is 32-42%, and during the year it increases to 48-63%. A high degree of correlation has been proven between an increase in the age of patients and an increase in the number of strokes. Up to 60 years of age, stroke occurs in a third of patients, two thirds of stroke occurs in the age group over 60 years old [34]. Approximately 80% of stroke patients have limited working capacity, and about 20% of them become disabled of group 1, ranking first among all causes of disability. Within 5 years, 25% of patients with cerebral infarction develop a relapse of stroke [10,13]. According to various authors, patients with ischemic brain damage more often die from cardiac causes than directly from stroke [35].

These two groups of diseases are united by many common risk factors, similarity in pathogenesis, as well as the fact that they continue to occupy a leading position in the structure of mortality. At present, the understanding of the close relationship between cardiac and cerebral pathology, which occurs against the background of various diseases of the cardiovascular system, has been significantly expanded and deepened. The combination of MI and AS is especially dangerous and prognostically unfavorable. According to the literature, the frequency of the combination of AMI and cerebral stroke ranges from 1.3 to 12.8%, more often it is observed in the first 2 weeks of the disease [29].

A common pathogenetic factor in the development of MI and ischemic stroke is multifocal atherosclerosis with a combined lesion of the coronary and carotid arteries. According to various authors, in patients with hemodynamically significant atherosclerosis of the coronary arteries, carotid lesions reach 30%, which significantly worsens the prognosis in patients with coronary heart disease in the general population (the survival rate within 5 years does not exceed 50%). In patients undergoing ischemic stroke, atherosclerosis of the coronary arteries is diagnosed in 30-60% of cases [20,22].

The possibility of combining these diseases is due to the pathogenetic relationship between trophic disorders in the myocardium and brain during hypoxia. One of the mechanisms for the development of cerebral ischemia is a decrease in myocardial contractility against the background of cardiac muscle necrosis in patients with subcompensated cerebral hemodynamics and low hemodynamic reserves. Consequently, the development of acute cerebral ischemia against the background of existing cardiac pathology triggers pathobiochemical cascade reactions that occur in all major parts of the central nervous system and cause changes in the neuronal pool, astrocytosis, microglial activation and associated dysfunction of the trophic supply of the brain [35]. The development of stroke is often associated with cardiogenic embolism, which occurs in rhythm and conduction disturbances, against the background of electrical instability of the myocardium. According to the literature, cardiac arrhythmias are found in 70-75% of patients with stroke [28]. Acute cerebral ischemia is usually caused by paroxysmal disturbances of central hemodynamics, which

is most often associated with insufficient myocardial contractility. Changes in the activity of the cardiovascular system, including the heart rate, is the most striking indicator of deviations that occur in the regulatory systems during ischemic stroke (IS), and they may be the earliest predictive signs of a patient's distress. After IS, almost all patients during sleep with episodes of apnea observe sinus arrhythmia with severe bradycardia, sometimes with short periods of asystole, which is abruptly replaced by tachycardia. Possibly severe atrial and ventricular premature beats, passing atrioventricular block of varying degrees, supraventricular and ventricular tachycardia. It is also known that cardiac arrhythmias can be an independent factor in the reduction of cerebral blood flow and lead to an additional deterioration of cerebral hemodynamics, as well as aggravate the course of AMI in patients with ischemic stroke. Frequent supraventricular extrasystole causes a reduction in cerebral blood flow by 7%, ventricular premature beats by 12%, and ventricular paroxysmal tachycardia by 40-75% [28,31]. When assessing the state of the cardiovascular system, it should be borne in mind that the work of the heart is influenced by intracardiac, peripheral reflexes, the arcs of which are closed not in the central nervous system, but in the intramural ganglia of the myocardium. Their defeat affects the autoregulation of cardiovascular functions. Disruption of intercellular interactions in the myocardium can lead to asynchronous excitation of myocardial cells and the appearance of cardiac arrhythmias.

Damage to a part of cardiomyocytes during myocardial ischemia triggers the processes of ischemic remodeling of the myocardium with the presence of zones of chronic ischemia (hibernation), zones of risk of ischemia and the formation of chronic heart failure, provokes processes of sclerosis, hypertrophy of the remaining myocytes with a change in their interposition, changes in the volume and mass of the myocardium. Thus, structural and functional changes in the left ventricle of the heart, prognostically unfavorable for the development of cerebral ischemia, are formed. The information provided shows the need for additional research in patients with acute myocardial infarction or with cerebrovascular accident.

In myocardial infarction, irritation of cardioreceptors occurs due to hypoxia, which triggers intracardial autoregulation mechanisms (intracardiac reflex). A decrease in blood pressure is accompanied by an acute decrease in coronary blood supply, which entails the development of reflex influences from the reflexogenic zones of the heart and large vessels on the brain, leading to spasms and stasis in the cerebral vessels, and in the heart - to aggravation of myocardial ischemia. The described pathogenetic mechanism of cerebral blood flow disturbance is more often observed in infarctions of the anterior wall of the left ventricle, since irritation in the region of the anterior descending coronary artery of the heart has a reflex effect on the vessels of the brain (mainly on the vessels of the brain stem). In elderly and senile people with severe atherosclerosis of cerebral vessels, neurological symptoms are persistent [24].

Myocardial damage in the post-stroke period can be associated with direct involvement of the central vegetative centers, and also occur due to the mass effect that develops in stroke in the hypothalamus and leads to the activation of efferent vegetative pathways and the hypothalamic-pituitary axis with the release of catecholamines and glucocorticosteroids, with the launch processes of alteration in the heart, blood vessels, lungs [2,4,25]. Alterative processes can be aggravated by the systemic inflammatory response syndrome (SIRS) developing in stroke. There is a significant activation of neuroinflammatory processes with the release of cytokines, adhesion molecules and other biologically active substances. This can lead to the development of SIRS and multiple organ damage, including the development of aseptic myocarditis pattern [5].

In the pathogenetic picture of AMI, catecholamine cytotoxicity should be distinguished first of all. Due to the violation of autonomic regulation in stroke, the release of adrenaline and norepinephrine from the presynaptic terminals of nerve fibers increases, and the action of "systemic" catecholamines is also important. However, based on the results of pathomorphological studies, we can assume that it plays a lesser role than their local influence. This can be confirmed by a peculiar morphological picture in the form of many small focal lesions of the myocardial tissue, most often of the type of coagulation necrosis around the axon terminal [9, 12].

Norepinephrine stimulates the formation of cyclic AMP (adenosine 3'5'-cyclophosphate) by activating adenylate cyclase of nerve endings, which causes the opening of calcium channels, the entry of calcium into the cell and the release of potassium. This leads to a change in the duration of the action

potential and may explain the high T-waves during electrocardiography (ECG). In other words, catecholamines cause the premature spread of the action potential, lengthening the depolarization phase and shortening the repolarization period, which can provoke cardiac arrhythmias.

With a long-term high level of norepinephrine, there is not only a malfunction of calcium channels, but also activation of lipid peroxidation, leading to damage to cellular structures [7, 15]. In response to a developing decrease in cardiac output, the sympathetic nervous system (SNS) is activated, accompanied by an increase in the heart rate (HR) and an increase in blood pressure (BP). An increase in coronary perfusion leads to uncompensated transmembrane calcium input and potassium output, disruption of the actin-myosin complex, ie, secondary reperfusion damage to cardiomyocytes [17-19]. The vasoconstrictor effect of catecholamines contributes to the development of arterial hypertension, which leads to an increase in the load on the myocardium and oxygen deficiency [3, 16, 26].

In view of the occurrence of instant vasoconstriction, an increase in oxygen demand does not lead to a compensatory increase in its delivery, which provokes the development of subendocardial ischemia, impaired cardiac output and leads to the risk of developing cardiogenic pulmonary edema. In clinical studies, almost all examined persons with acute cerebral ischemia were found to have an increase in blood plasma levels of noradrenaline by 2.4 times and adrenaline by 2 times. Especially high concentrations of catecholamines were observed during the first three days of the disease, with a gradual decrease by the 40th day. A correlation was obtained between a very high content of catecholamines in the blood and the severity of abnormalities on the ECG [1, 21].

Acute stroke can lead to the syndrome of "stunned" myocardium, which is a reversible condition accompanied by arrhythmias, ECG changes, left ventricular dysfunction and increased concentration of ischemic markers. This condition is directly related to the release of catecholamines from the sympathetic nerve terminals, rather than to the release of systemic catecholamines. Prolonged opening of beta-1 adrenergic calcium channels leads to a rapid depletion of adenosine triphosphate, leading to mitochondrial dysfunction and cell death. Stress release of corticosteroids makes an additional "contribution" to myocardial damage. Hans Seli was the first to prove that stress can lead to cardiac damage such as myofibril degeneration [14]. Probably, corticosteroids can potentiate the action of catecholamines, but do not carry out cardiotoxic actions by themselves.

ECG is the main method for diagnosing AMI. However, in 68.5% of patients in the acute period of ischemic stroke, changes in the ECG are detected, taking place on days 1-2. According to the literature, 6 typical variants of changes in the end part of the ventricular ECG complex were established, which showed that, unlike AMI, there are no changes in the QRS complexes and the dome-shaped elevation of the ST segment in stroke, while changes in the ST-T complexes are very labile, with a favorable course stroke, by 3-5-7 days, the normal shape and polarity of the teeth are restored. [23].

The most frequent clinical manifestations occurring in the structure of AMI are arrhythmias and blockages: sinus bradycardia or tachycardia; sinus block; AV blockade; atrial and ventricular premature beats; atrial fibrillation; atrial fibrillation; slow atrioventricular rhythm; violation of intraventricular conduction; blockade of the legs of the bundle of His; ventricular tachyarrhythmias.

In this case, the most frequent arrhythmia in the acute period of cerebrovascular accident is atrial fibrillation, which is associated with a high incidence of cardioembolism and significantly increases the incidence of recurrent strokes [8, 25, 30]. In addition, atrial fibrillation, ventricular extrasystoles and bundle branch blockade are significantly more common in cardioembolic stroke compared with atherothrombotic and hemodynamic stroke [27, 30, 32]. This type of stroke is characterized by a more severe course and slow recovery of neurological deficit. A relationship was established between the localization of the lesion associated with a specific vascular pool and changes according to ECG data: atrial fibrillation and tachycardia are more common when the focus is located in the basin of the left middle cerebral artery, and bradycardia and atrial conduction disorders - when the focus is located in the vertebral-basilar basin. [32]. In other studies, supraventricular and ventricular ectopic activity increases in patients with acute stroke without a history of cardiac pathology, which is likely due to cerebrogenic effects. In the acute phase of IS, an increase in supraventricular and ventricular ectopic activity and changes in heart rate were recorded, which depended on the size and localization of the ischemic focus. At the end of the acute period of stroke, AMI

regressed in most patients. Regression of neurological deficit in the acute phase of IS was inversely related to the frequency of episodes of bradyarrhythmias [27, 32].

Special attention should be paid to the increase in the QT interval in patients with stroke, since this is an unfavorable factor provoking arrhythmogenic complications. A QT interval greater than 450 ms is considered an important predictor of such complications and is associated with a threefold risk of sudden death from cardiovascular causes.

Clinical observations have shown that cardiac damage can develop immediately or within a few hours after an acute cerebral catastrophe. With hemorrhagic transformation of IS, complicating the course of stroke in the acute period, there was a significantly larger number of patients with cardiac arrhythmias, and patients with atrial fibrillation were significantly more frequent [33].

Some patients may have an asymptomatic course of AMI with a slight increase in cardiac enzymes of myocardial damage, while others develop a clinic of cardiogenic shock, acute heart failure and pulmonary edema. Pulmonary edema has been reported in approximately 10% of patients with subarachnoid hemorrhage [27].

A frequent manifestation of AMI is the development of systolic dysfunction with hypokinetic changes in the walls of the heart according to echocardiography. An equally important role is played by the development of diastolic dysfunction with an increase in end-diastolic pressure in the left ventricle and activation of the sympathetic nervous system, which leads to endothelial dysfunction and the development of hypercoagulation [11].

The existing close causal relationship between myocardial infarction and acute cerebrovascular accident determines the complex clinical course of the disease, leading to tactical errors in the management of this category of patients. However, given the high mortality of patients with a combination of two vascular catastrophes, it is necessary to further study the etiology and pathogenesis of myocardial infarction and acute cerebrovascular accident with the development of algorithms for individual preclinical prediction.
Thus, the presence of a complex symptom complex when examining patients requires the doctor to be alert and conduct a targeted search for a combination of myocardial infarction and acute cerebrovascular accident. Timely recognition of cases of a combination of two groups of diseases and an individual approach is extremely important, since late diagnosis leads to errors in the tactics of specialized hospitalization and lengthens the time for the provision of specialized medical care and optimization of treatment measures.

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