AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 01, Issue 10, 2023 ISSN (E): 2993-2149

Chronic Cerebral Ischemia and Cardiac Arrhythmias

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Abstract: The article discusses the role of heart rhythm disturbances in the development of different types of ischemic stroke. In the pathogenesis of cardioembolic stroke, the paroxysmal form of atrial fibrillation is important and independent. In the development of hemodynamic stroke, one of the decisive factors is transient bradyarrhythmia due to atrioventricular block and sick sinus syndrome, as well as deterioration of left ventricular contractility due to transient myocardial ischemia. It has been shown that bradyarrhythmia and ventricular extrasystole, associated with a high risk of coronary complications and life-threatening for the patient, may not correlate with the severity of neurological deficit. In patients with cardiogenic ischemic stroke, in almost 40% of cases, pathogenetically significant cardiac arrhythmias are asymptomatic . The possibility of a hidden course of cardiac arrhythmias and episodes of myocardial ischemia, their detection in some cases only after the development of neurological symptoms and the impact on the course of the disease determine the importance of timely registration of these changes to optimize treatment tactics.

Keywords: ischemic stroke, cardiocerebral embolism, atrial fibrillation, bradyarrhythmia, cardiac output, Holter monitoring.

Introduction. Currently, the concept of heterogeneity of ischemic stroke remains relevant. The problem of cardioembolic stroke (CEI) is of particular social importance due to its development mainly in people of young working age. The relationship between cardiac and cerebrovascular pathology is due, on the one hand, to the commonality of a number of risk factors for the occurrence of coronary heart disease and ischemic stroke, and on the other hand, to the importance of heart pathology as one of the leading risk factors in the pathogenesis of cerebrovascular accident (CVA) [7, 14, 26, 27, 41]. Thus, it has been shown that the cause of ischemic stroke in 15-20% of cases is cardiogenic thromboembolism of cerebral vessels [2, 15, 24, 33, 41, 52]. In patients under 45 years of age, this percentage increases to 36 [13, 29].

One of the first reports of occlusion of cerebral arteries by a thrombus, which probably formed in the cavity of the heart, was made by Virchow back in 1847. He later called this phenomenon embolism (from the Greek “ plug ”). In the last few decades , advances in cardiac and neuroimaging have led to the real identification of potential and identification of new sources of embolism [16, 17, 33, 41, 52, 53]. Echocardiography in combination with transesophageal echocardiography is a priority diagnostic technique for identifying cardiogenic sources of embolism. According to various authors [16, 32, 38], from 25 to 46% of patients with stroke have at least one potential cardiac source of embolism, and this proportion increases in patients over 70 years of age.

It has been established that cardiocerebral embolism most often occurs in the following forms of heart pathology: atrial fibrillation, acute myocardial infarction, post-infarction cardiosclerosis with the formation of aneurysms and the presence of blood clots in the cavity of the left

ventricle, rheumatic heart disease, valve replacement. Less common causes include: non-ischemic cardiomyopathies , infective endocarditis, calcified aortic stenosis, congenital heart defects with septal defects, mitral valve prolapse, atrial myxoma , sick sinus syndrome, antiphospholipid syndrome, thrombophilic conditions [3, 13, 15, 41, 52].

According to a number of authors, the size and nature of the embolic fragment depend on the location of its formation, which, in turn, can affect the size of the cerebral ischemic focus. Yes, intracameral thrombus formation promotes the formation of larger emboli with the formation of a red (fibrin) thrombus. The pathogenetic factor in this case is circulatory stasis caused by focal or global akinesia, as well as in some cases additional endothelial damage. Valvular emboli are usually small and consist of a white (platelet-rich) thrombus or dystrophic calcifications [16, 33, 52].

The source of most cardiogenic embolisms are the chambers of the heart, and important importance is attached to disturbances in its rhythm. Cardiac arrhythmias create favorable hemodynamic conditions for thrombus formation , since turbulent blood flows promote hemostatic activation processes . The resulting platelet -fibrin clots, as well as fragments of blood clots, can penetrate from the left parts of the heart through the aorta into the main arteries of the head (MAG), leading to the development of cardiocerebral embolism. Thrombosis in cardiac arrhythmias mainly occurs in the left auricle and left atrium. A thrombus can become a source of repeated thromboembolism of both cerebral vessels and peripheral arteries. Multiple small emboli cause the clinical picture of repeated acute cerebrovascular accidents [1, 11, 41].

Ischemic stroke in young patients has a number of features, and it is often a hereditary pathology; the family history of such patients is burdened by a high frequency of acute vascular episodes in relatives [23, 31, 35, 42, 50]. Epidemiological studies indicate an increase in the risk of stroke by 30-76% in the presence of a positive family history [31].

One of the first places in terms of frequency of development in young people is occupied by EI. The risk of its development is in patients with idiopathic mitral valve prolapse [17, 49], endocarditis, cardiomyopathies , cardiac myxoma , anatomical intracardiac anomalies, for example, paradoxical cardiac embolism from the venous system to the arterial system, as a result of existing defects of the interatrial or interventricular septum (patent foramen ovale) [10, 18, 25, 39, 41, 43, 46].

A separate group consists of the so-called monogenic strokes - conditions when the development of NMC is determined by the carriage of one or another mutant gene. Monogenic diseases associated with heart damage include a number of syndromes and diseases accompanied by malignant cardiac arrhythmias and a high risk of sudden cardiac death (SCD). The cause of hereditary heart rhythm disorders is considered to be anomalies of the following main classes of proteins: contractile and cytoskeletal proteins , ion channels and intercellular contacts, transmembrane transporters, as well as their modulators [22].

Familial arrhythmias are most often caused by channelopathies and are characterized by impaired generation of action potentials and excitability of cell membranes in the myocardium, skeletal muscles, and neurons. Genetically determined “cardiac” channelopathies , which manifest themselves as arrhythmias and can lead to CES, include hereditary familial forms of long QT interval syndrome. To date, 13 genotypes have been identified that determine the presence of different variants of QT syndrome and are designated as LQT, but the most common and clinically significant are 3 of them: LQTl (mutation of the potassium channel gene KCNQ1), LQT2 (potassium channel gene), LQT3 (sodium channel gene SCN5A) [34, 36, 39, 42]. The basis of life-threatening heart rhythm disturbances in this case is the asynchrony of repolarization of various parts of the ventricular myocardium and, as a consequence, an increase in its total duration. Myocardial depolarization is determined by the opening of fast sodium channels and inversion of the membrane charge of cardiomyocytes , and its repolarization and restoration of the original membrane charge occur due to the opening of potassium channels. Impaired

potassium or sodium channel function due to genetic mutations leads to slower myocardial repolarization and, consequently, to prolongation of the QT interval on the electrocardiogram (ECG). According to the International Registry, in approximately 85% of cases the disease is hereditary, while about 15% of cases are the result of new spontaneous mutations. Long QT syndrome is characterized by prolongation of the QT interval on the ECG and a predisposition to malignant ventricular arrhythmias and SCD. The QT interval is measured on a standard ECG in chest leads V2 and V5, the duration of the corrected QT interval (QTc) is calculated using the modified Bazett formula : QT with (8) = QT/VR-R (8) [20]. Prolongation of the QT interval from more than 450 ms in men and more than 470 ms in women is highly likely to indicate the presence of the syndrome. The main clinical manifestation of long QT syndrome is attacks of loss of consciousness caused by relapses of ventricular tachycardia of the “pirouette” type.

Short QT syndrome is a genetically heterogeneous disease with changes in potassium channels. Its ECG criterion is a decrease in QTc duration <300 ms with a high, symmetrical peak-shaped T wave [30]. One of the main stages in diagnosing the syndrome related to primary electrical heart diseases is the detection on a standard ECG of a sustained shortening compared to the norm in the duration of the QT interval. Population ECG screening programs, including medical examination of the population, are important in diagnosis [51].

Catecholamine-dependent ventricular tachycardia is a heterogeneous hereditary disease characterized by physical or emotional stress-induced bidirectional or polymorphic ventricular tachycardia, rapidly progressing to ventricular fibrillation, with a high risk of SCD, in which genetic defects of the calcium channel are found [21, 48].

Brugada syndrome is a primary electrical heart disease with a high risk of SCD. A molecular genetic analysis of Brugada syndrome revealed that the disease is linked to the SCN5A gene, which encodes the α-subunit of the sodium channel [21]. Currently, more than 100 different mutations that are important for the development of the syndrome have been identified in this gene. In addition to genetically determined dysfunction of sodium channels, mutations have been identified in genes that modulate the function of sodium channels and encode L-type cardiac calcium channels. However, the genetic nature of the pathology in most patients with the Brugada syndrome phenotype still remains unclear.

Lenegra's disease (isolated cardiac conduction disorder) is a primary degenerative disease primarily affecting the cardiac conduction system with progressive cardiac conduction impairment. The first description of a mutation in the SCN5A gene was made in 1999. The consequence of this mutation is a decrease in the function of the sodium channel. In several families with idiopathic ventricular fibrillation, mutations in the gene encoding the sodium channel SCN5A were identified, leading to a decrease in the number of functioning sodium channels with a decrease in their activity [48, 56].

Idiopathic ventricular fibrillation is diagnosed based on the identification of life-threatening ventricular arrhythmias and the exclusion of other diseases with a risk of sudden death.

Primary sick sinus syndrome, characterized by sinus bradycardia and periods of sinus node arrest, is a primary arrhythmogenic disease and is genetically heterogeneous.

In the case of the genetically determined variant of familial valvular myxoma , patients often have a number of characteristic additional features, including multiple pigmented skin lesions (lentiginosis), cutaneous myxoma, myxoid fibroadenoma of the mammary gland, and pigmented nodular adenoma of the adrenal gland. In more than 1/2 of cases, similar lesions are present in one of the close relatives [17, 39].

The characteristic signs of clinical manifestations of EI in the overwhelming majority of cases are the sudden development of a stroke with the maximum severity of neurological symptoms at the onset of the disease. In approximately 15% of patients, symptoms develop stepwise , which

is associated with subsequent hemorrhagic transformation in the ischemic zone, as well as with distal migration of embolic fragments [12, 14, 44].

Patients with CES are also characterized by the formation of large cerebral infarcts, which is associated with more frequent pathology of the heart chambers. Most often (85% of cases) the vascularization of the left middle cerebral artery is involved in the lesion process. Only 15% of infarctions are localized in the vertebrobasilar system, mainly in the territory of the left posterior cerebral artery. According to many authors, cardiogenic emboli reach the arteries of the vertebrobasilar system in almost 20% of cases, which is much more common than previously thought [5, 6, 8, 9, 14, 37].

In addition, more than 1/2 of patients with CES have silent and repeated NMCs located in the contralateral blood supply, which worsens the prognosis of the disease. It should be emphasized that a history of systemic embolism and the presence of silent foci of ischemia are considered characteristic signs of CES [6, 9, 12, 32, 44].

Thus, in the absence of pathognomonic clinical symptoms, there are a number of signs characteristic of CES, each of them individually is not specific, and only their combination has diagnostic value. Almost all patients with CES have cardiac pathology with a high embologenic risk, and in 1/4 of cases changes are detected for the first time.

Holter ECG monitoring

It is believed that one of the most common causes of cardiogenic embolism is heart rhythm disturbances [11, 17, 19, 32, 41, 48, 52]. In contrast to the permanent form of atrial fibrillation, paroxysmal arrhythmia is often clinically asymptomatic with the absence of anamnestic data on its presence before the stroke, not registered with a standard ECG, and can immediately result in cardiocerebral embolism with the formation of cerebral infarction. In addition, such arrhythmias as paroxysmal supraventricular tachycardia, frequent supraventricular extrasystole, high-grade ventricular extrasystole, bradyarrhythmias due to impaired cardiac conduction and sick sinus syndrome are also clinically asymptomatic in almost 1/2 of patients and are detected only using a Holter ECG - monitoring .

Holter ECG monitoring is one of the most informative methods for diagnosing arrhythmias. With its help, it became possible to determine the true frequency of arrhythmias, clarify their nature, identify asymptomatic rhythm disturbances, accurately determine how a particular disorder affects the patient's condition and well-being, and adequately assess the effectiveness antiarrhythmic therapy [4, 47, 48, 54]. This makes it possible to significantly increase the level of diagnosis of heart rhythm disorders, especially those of a paroxysmal unstable nature. In addition, the method has no contraindications, is as physiological as possible and is applicable in all age groups. According to the literature, in patients with SCI, using Holter ECG monitoring , a significantly higher frequency of cardiac arrhythmias was found compared to patients who had not suffered a stroke [20, 40, 48, 53].

the Holter ultrasound is of undeniable diagnostic value in such cases. ECG monitoring . Thus, only an in-depth cardiac examination using this method makes it possible to establish the cause of ischemic stroke in these patients and transfer them from the stroke group of unknown etiology to the CES group.

Cardiac rhythm disturbances can lead to cerebral ischemia both through cardiocerebral embolism and due to a decrease in cardiac output (MCV) with changes in general hemodynamics, especially in the presence of stenotic lesions of intracranial vessels and MAG.

In addition to the embologenic danger, cardiac arrhythmias lead to the development of cerebral ischemia due to a decrease in MOS in combination with MAG pathology [20, 53]. In this regard, of interest is a group of patients with the so-called hemodynamic stroke, in whom cerebrovascular accident developed through the mechanism of cerebrovascular insufficiency as a

result of a combination of pathology of cerebral vessels and disturbances in general hemodynamics.

Chronic cardiac pathology, leading to a sharp decrease in MVR against the background of the existing depletion of cerebrovascular reserve, includes transient conduction disturbances leading to severe bradyarrhythmia (usually caused by sick sinus syndrome or high-degree AV block), acute myocardial infarction or episodes of silent myocardial ischemia in patients with an initial decrease in left ventricular contraction (ejection fraction less than 40%) against the background of post-infarction cardiosclerosis [14, 19, 53].

Thus, cardiogenic cerebral ischemia can develop not only as a result of cardiocerebral embolism, but also against the background of cerebrovascular insufficiency, which may be based on cardiac pathology, leading to sudden changes in central hemodynamics (a drop in MOS and a decrease in blood pressure).

It is important to note that cardiac changes, which lead to deterioration of cerebral hemodynamics, are also a factor aggravating the course of stroke. In addition to the additional adverse effect on cerebral hemodynamics in patients with stroke, cardiac pathology may have an independent prognostic value both in terms of the causes of repeated cerebrovascular accidents and in terms of coronary complications, such as myocardial infarction and sudden death [21].

Long-term prospective epidemiological studies have established that the main cause of death in patients with stroke is changes in cardiac activity. According to the literature, the main causes of cardiac complications are considered to be: ventricular arrhythmia, myocardial ischemia with depression of the 8T segment and primary bradyarrhythmia . At the same time, the development of bradyarrhythmia in 80% of cases is caused by SA blockade and 20% by AV blockade [14, 19, 45].

There are many studies that show that high-grade ventricular extrasystole has its own prognostic significance regarding the risk of sudden death, especially in patients with organic heart diseases [11, 48].

In addition to heart rhythm disturbances, silent myocardial ischemia is of significant importance in the prognosis of diseases, which, according to some data, increases the risk of coronary death by 3-4 times compared with the control group, reaching a 10-fold increase in patients with organic heart diseases [14, 15 , 28, 55].

Conclusions. Thus, cardiac rhythm and conduction disturbances in patients with Acute cerebrovascular accidents can have different meanings: they can cause the development of stroke, aggravate the course of the disease, and influence the prognosis and outcome of stroke.

It should be emphasized once again that a feature of arrhythmias is often an asymptomatic course and the presence in patients who have no complaints or anamnestic information. The possibility of a hidden course of heart rhythm disturbances and episodes of myocardial ischemia, their detection in some cases only after the development of neurological symptoms and the impact on the course of the disease determine the importance of timely registration of these changes using Holter ECG monitoring, which allows to significantly expand the diagnosis of the leading pathogenetic mechanism of ischemic stroke development and optimize treatment tactics.

This determines the need for coordinated tactics for the management of patients with vascular pathology of the brain by cardiologists and neurologists.

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