ISSN 2181-3187

GENERAL OVERVIEW OF CEREBRAL CARDIAC SYNDROME AND ITS RELEVANCE

Z.J.Narzilloeva

Annotation: Cerebrocardial syndrome is considered as a complex of cardiac disorders, which arise against the background of damage to the central nervous system and develop in acute cerebrovascular accident. A special role in its development is played by damage to the autonomic nervous system with the emergence of local and systemic catecholamine cytotoxicity , neuroinflammation and free radical damage. Clinically, this leads to the appearance of arrhythmias and other ECG phenomena, systolic and diastolic dysfunction, and an increased risk of sudden death, which requires identifying patients with similar risk factors and developing recommendations for their diagnosis and treatment.

Keywords: Cerebrocardial syndrome, acute cerebrovascular accident, subarachnoid hemorrhage, electrocardiography, cardiac arrhythmias.

For a Russian cardiologist brought up on the works of the national scientific school of the 20th century, who in recent years has been faced with semantic differences in international and domestic classifications of diseases, the emergence of “new” nosological forms of diseases in medicine is inevitably accompanied by the effect of “already seen.” Metabolic, non-coronarogenic and non-inflammatory myocardial diseases, which F. Wuhrmann (1956) called “the stepdaughters of the clinic and pathological anatomy,” have been studied for a long time, however, to this day this area of cardiology remains the least studied. According to H. Marriott (1960), there are more than 100 situations in which ECG changes can be perceived as ischemic. Historically, the clinical equivalents of these situations were myodegeneration and cardiac myasthenia, myocardia in France, myocardosis in Germany, cardiomyopathy (CM) in England and myocardial dystrophy (MCD) in Russia. The inclusion of the term “CMD” in the WHO glossary of diseases resolved many years of semantic controversy.

For the domestic clinician, the most acceptable classification of MKD by M.S. Kushakovsky (1977), according to which they distinguish alcoholic, disovarian (menopausal), diselectrolytic , neurogenic, endocrine-metabolic, disproteinemic , anemic, toxic, tonsillogenic , during physical stress (“athletic heart”) "), with physical effects on the heart (trauma, radiation), infiltrative, with systemic neuromuscular diseases and postpartum MCD (CMD) [2]. Of greatest interest for this topic are neurogenic, disovarial (menopausal) and physical stress-related ICM.

The concept of “ cerebrocardiac syndrome”

ISSN 2181-3187

As for neurogenic myocardial damage, they have been known for a long time, and we believe that it is more appropriate to consider them in the context of cerebrocardial syndrome (CCS), a term proposed in the mid-50s. last century to indicate certain changes on the ECG accompanying brain damage. Catecholamines are known to damage the myocardium. Histological analysis of the hearts of patients with ACN revealed disorders characteristic of excessive infusion of norepinephrine. In particular, the following were found: myocytolysis , local coagulative necrosis, subendocardial petechial hemorrhages, edema and interstitial mononuclear infiltration [27]. Catecholamines cause premature propagation of the action potential, prolonging the depolarization phase and shortening the repolarization period , which provokes rhythm disturbance as the initial manifestation of CCS. The response to the resulting decrease in effective cardiac output is increasing sympathetic stimulation in the form of an increase in heart rate and an increase in blood pressure. Compensatory increase in coronary perfusion leads to uncompensated transmembrane Ca2+ entry and K release, disruption of the actin- myosin complex, i.e. secondary reperfusion damage to cardiomyocytes . At the same time, long-term high levels of norepinephrine can cause not only disruption of the opening of calcium channels, but also activate peroxidation, which leads to cellular damage and leakage of cardiac enzymes from the cell [11, 12]. Clinical confirmation of the development of left ventricular dysfunction in this case will be an increase in the level of myocardial damage enzymes, characteristic ECG changes, and an increase in B-type natriuretic peptide [13, 14]. Unlike ischemic transient myocardial dysfunction, when the main trigger of myocardial damage is its reperfusion , for CCS the trigger of damage is dysregulation of the autonomic nervous system with excessive release of norepinephrine into the myocardium from sympathetic nerve endings [10]. Against the background of a persisting high concentration of catecholamines in the blood and synapses, a “ stunned ” state occurs myocard "("stunned or petrified myocardium"). In patients with premorbid coronary artery disease, this leads to the development of contractile necrosis, while in premorbidly healthy patients, cardiac dysfunction is potentially completely reversible within 3-42 days [17]. For CCS, the most changes in the final part of the ventricular complex are characteristic, in particular, a pronounced increase in the duration and amplitude of the T wave, its broadening (“dimensionless”, “crazy” T wave), inversion, enlargement of the U wave, fusion of the T and U waves (“T+U”) There is a prolongation of the QT and QTU intervals. Elevation or depression of the ST segment is possible, creating a picture of a “ pseudo-infarction ” curve. Similar ECG changes occur with Wellens. syndrome . Pronounced inverted T waves in patients with unstable angina were first described in 1982 and called “ Wellens” syndrome ”, named after one of the authors of the publication [3]. The ECG pattern has a high (84%) diagnostic value for critical (more than 70%) stenosis of the left coronary artery and prognostic

ISSN 2181-3187

significance (38% risk of adverse cardiac events expected in the next 16 months, including acute myocardial infarction) [4]. Unlike Wellens syndrome , T waves in CCS are asymmetrical, high-amplitude, wide and “too large for myocardial infarction.” The most common rhythm disturbances are: sinus bradycardia (less commonly tachycardia), ventricular or atrial extrasystole, atrial fibrillation, slow atrioventricular rhythm. Sometimes there is a violation of intraventricular conduction in the form of transient blockades of one of the His bundle branches [5-7]. CCS is characterized by rapid reverse dynamics that do not reflect the direction of the cerebral process [8]. Pathophysiology CCS is caused by the following pathological conditions: acute cerebrovascular accident (ACVA), traumatic brain injury (TBI), encephalitis, subarachnoid and intracranial hemorrhages, infarctions and brain tumors, embolism, thrombosis, aneurysms and cerebral vascular malformations , neuroinfections , intoxication , comas of various origins, neurosurgical operations, epilepsy. CCS, as a special case of cerebro -visceral pathology, indicates the inextricable connection and interdependence of cerebral and systemic hemodynamics [9]. It is known that the development of CCS is based on complex metabolic destructive disorders that occur in the myocardium following cerebral pathology. Cardiac function is regulated by centers in the brain stem through sympathetic and parasympathetic innervation. Damage to certain brain structures is accompanied by disruption of cardiovascular autonomic regulation [10] with excessive sympathetic activation [11]. Centrogenic excitation of the sympathetic-adrenal system and dysfunction of the autonomic nervous system lead to increased release of catecholamines into the blood plasma, causing shifts in the functioning of humoral systems and profound disturbances in tissue metabolism in the myocardium, both at the level of substrates and at the level of enzymatic reactions. Excessive toxic effects of catecholamines lead to a decrease in the density and affinity of β- adrenergic receptors (AR) of cardiomyocytes , an increase in the amount of catecholamines in the myocardium and a decrease in the contractile force of cardiomyocytes (“metabolic damage of the myocardium of the hyperadrenergic type”) [2]. “Histotoxic myocardial damage” due to hypercatecholaminemia [12] causes secondary morphofunctional changes in cardiomyocytes such as myocytolysis and focal myocardial micronecrosis (the so-called “adrenaline myocarditis”) [13]. With a sufficient duration of the “sympathetic storm” [14], the reserves of norepinephrine in the myocardial tissue are depleted. Catecholamines change the density and duration of the slow Ca - Na -current in the 2nd phase of the action potential, increase the density and shorten the activation time of the K-current Ix 1, and also stimulate the Ca -current entering the cell . The changes occurring can be schematically represented as follows: ß-ARs of the myocardium cause an increase and acceleration of the energy entering the cardiomyocytes Ca current, which leads to an increase in the intracellular concentration of Ca ions and an increase in the permeability of the cell membrane for

ISSN 2181-3187

K ions, resulting in an increase in the density of the outgoing K current (current Ix 1) , and therefore a more rapid completion of the repolarization of cell membranes and a shortening of the action potential. Neurogenic (adrenergic) CMP manifests itself in the clinic in 2 variants: in the form of acute effects effects of catecholamines on the myocardium and in the form of a norepinephrine-deficient state of the myocardium. S.P. Astrakhantseva and M.S. Kostomarova (1966) found in almost all examined individuals with acute cerebral ischemia an increase in the content of norepinephrine in the blood plasma by 2.4 times and adrenaline by 2 times. Particularly high concentrations of catecholamines occurred during the first three days of the disease with a gradual decrease by the 40th day. The authors obtained a correlation between very high levels of catecholamines in the blood and the severity of these abnormalities on the ECG [15]. It is known that subarachnoid hemorrhage (SAH) usually involves the ventricles of the brain, which are located in close proximity to the hypothalamus, and, apparently, it is this circumstance that leads to the release of excess catecholamines into the blood [16]. At the cellular level, this causes hypermetabolism and electrolyte imbalance in mitochondria [17] and toxic damage to cardiomyocytes [18]. G.NeilDwyer et al (1990) found a direct correlation between the concentration of catecholamines in the blood plasma and ECG changes in SAH [19]. Later, between 4-6 days from the acute onset of SAH, sympathicotonia is replaced by hyperactivity of the parasympathetic nervous system [20]. Activation of the sympathetic nervous system may also occur through the insula Reili) [21], and an asymmetry of cardiovascular effects is revealed: the sympathetic ones are caused by the right-sided, and the parasympathetic ones by the left-sided dominant [22]. Experiments on rats simulating cerebral infarction confirmed the hypothesis about the difference between right- and left-sided cerebrocardial effects, including the type of arrhythmias. RDLane et al . (1992) found a relationship between right hemisphere stroke and supraventricular arrhythmia, as well as between left hemisphere stroke and ventricular arrhythmias. Patients with cerebral infarction localized in the right hemisphere show a more pronounced increase in blood pressure values and variability than with left-sided infarcts. In this group, arrhythmias and prolongation of the QT interval are more often observed, as well as an increase in the concentration of norepinephrine in the blood plasma. Right-sided autonomic stimuli have a predominant effect on the sinoatrial node, and stimulation or suppression of the right part of the medulla oblongata and hypothalamus has a greater effect on ectopic cardiac activity [23]. Heart rhythm disturbances in conditions of impaired autoregulation of cerebral blood flow negatively affect reparative processes in the zone of cerebral ischemia. Even a moderate transient cardiogenic drop in blood pressure further impairs the blood supply to the peri-infarction area. Frequent supraventricular extrasystole can cause a decrease in cerebral blood flow by 7%, ventricular extrasystole by 12%, and ventricular paroxysmal

ISSN 2181-3187

tachycardia by 40-75%. Prolonged supraventricular paroxysmal tachycardias lead to a significant decrease in left ventricular stroke volume with subsequent deterioration of cerebral hemodynamics [24]. According to another theory, hypomagnesemia plays an important role in the pathogenesis of CKS [25]. CCS with suprabulbar localization of the process (tumors, traumatic brain injuries - TBI, encephalitis, intracerebral hematomas, cerebral infarctions) can be caused by mechanical factors: impaired cerebrospinal fluid dynamics , increased intracranial pressure, cerebral edema, compression of the brain stem, in general, the degree of suddenness and speed of development of cerebral pathology. Systematizing clinical and experimental materials, I.I. Isakov (1971) identifies 7 electrocardiographic variants of CCS: vagotonic , tachycardial , β- pansympathicotonic (circulatory), hyperamphotonic , dystrophic, arrhythmic and acute cerebral (emergency) [26]. The clinical significance of neurogenic changes in the heart lies in the similarity of the ECG abnormalities that develop with myocardial lesions. Cardiac damage may develop immediately or within a few hours after ACV. Some patients may have an asymptomatic course with a slight increase in cardiac enzymes of myocardial damage, while others develop a clinical picture of cardiogenic shock, acute heart failure and pulmonary edema. Pulmonary edema has been reported in approximately 10% of patients with SAH [3, 4].

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ISSN 2181-3187

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