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Copeptin in Early Diagnosis of Myocardial Infarction in patients with acute Coronary Syndrome.

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Emergency care for patients with suspected myocardial infarction is critically important in the fight for the patient's life, in our study we want to present a new opportunity for early diagnosis of myocardial infarction, which will allow faster verification of the diagnosis. Among patients with acute coronary syndrome, it is important to identify groups of patients with myocardial necrosis who have an increased risk of complications and death. In this group of patients, the most aggressive treatment tactics are indicated, including the use of percutaneous coronary intervention or coronary artery bypass grafting. Currently, the "gold standard" in verifying myocardial necrosis and, consequently, infarction is the determination of an increase in the level of troponin T or I. This is recognized as a prerequisite for the diagnosis of acute myocardial infarction (AMI), along with one of the following factors: chest pain ischemic character, the presence of ECG signs of ischemia, a decrease in the mass of the contractile myocardium according to imaging methods. However, despite the widespread use and high efficiency of troponin use in the diagnosis of myocardial necrosis in patients admitted with suspected acute coronary syndrome, significant shortcomings in measuring troponin levels remain relatively late increases in blood levels after the onset of myocardial infarction (3-6 hours after the onset of necrosis), as well as an increase in its level not associated with myocardial ischemia, for example, in sepsis, septic shock, pulmonary embolism, subarachnoid hemorrhages, severe heart and renal failure, etc., in connection with which there is a need for longer observation and sequential testing for troponin in dynamics in patients with intermediate or high clinical index of suspicion of ACS, which can lead to a delayed diagnosis and treatment initiation, increased complications and potential mortality. In this regard, the search continues for additional diagnostic criteria for the early manifestations of myocardial infarction in order to optimize treatment tactics and reduce lethal and non-lethal complications of myocardial infarction. One of these markers, which have recently attracted the attention of researchers, is copeptin. Diagnosis of myocardial infarction in the early stages in practice can be difficult due to the fact that myocardial infarction is often atypical, the indicators of cardiospecific markers of necrosis do not exceed the threshold values of the norm, and electrocardiogram (ECG) data in the first hours of the disease are not always diagnostically significant, or the assessment of indicators The ECG is difficult due to changes that were present initially (aneurysm of the left ventricle, blockade of the bundle of His, etc.). Therefore, the need arose for additional methods that would make it possible to diagnose myocardial infarction already in the first hour from the beginning of the index event. Copeptin levels can be used as a diagnostic marker in patients with suspected MI in combination with other biomarkers, but, to date, the potential significance of copeptin in the early diagnosis of MI remains insufficiently understood, which necessitates further research. Copeptin, a glycopeptide acid consisting of 39 amino acids, is the C-terminal part of proasopressin and is excreted together with AVP in equimolar concentrations, reflecting the level of endogenous stress in the human body. Direct determination of vasopressin content is difficult today, since the hormone in the blood is unstable, has a short half-life, and 90% of the circulating hormone is associated with platelets [8]. Therefore, an accurate, reproducible and reliable method for assessing the level of vasopressin in the blood is currently lacking. The clinic uses a surrogate vasopressin marker - copeptin. The functions of copeptin in the body remain poorly understood. Copeptin is secreted in an amount equimolar to vasopressin.

In contrast to vasopressin, the level of copeptin remains stable in the blood for several days and therefore is more readily available for determination [9]. However, being a marker of neurohormonal stress, copeptin is not specific for the pathology of the cardiovascular system. Although copeptin does not act as a cardio-specific marker, it is nevertheless possible to use this biomarker as a diagnostic method for MI, since its concentration in the blood increases in the first hours of the disease [9]. Early studies have shown that copeptin increases significantly in the blood as early as the first hour of MI and then decreases over several hours. For example, in a study by Gu YL, et al. (2011) [10] in 145 patients with MI, blood levels of copeptin, CK-MB, and Tn T were determined in dynamics. It was found that the peak concentration of copeptin was recorded immediately after the onset of MI symptoms and was equal to 249 pmol / l. Peak values of CC-MB and Tn T were recorded much later and were equal to 275 U / L and 5.75 µg / L. Copeptin levels decreased by the 10th hour from the onset of myocardial infarction, and the levels of CK-MB and Tn T - much later.

The degree of increase in copeptin was significantly greater in patients with ST-segment elevation MI on ECG than in patients with non-ST-segment elevation MI on ECG. In the works of Ananth V, Beig J (2016) [11], a relationship was found between the presence of copeptin in the blood and the size of myocardial infarction when the area of myocardial lesion was determined using magnetic resonance imaging. Evaluation of the effectiveness of the combined use of 2 biomarkers - Tn and copeptin in the diagnosis of MI were reviewed in the studies of Afzali D, et al. (2013) [12], Folli Ch, et al. (2013) [13], Sayed ZH, et al. (2014) [14]. Afzali D, et al. (2013) [12] included 230 patients admitted to the hospital with a diagnosis of ACS in the study. Tn I and copeptin were determined on admission to the hospital and additionally after 3-6 hours. MI was diagnosed in 107 patients (MI with ST-segment elevation on the ECG in 24, without ST-segment elevation on the ECG - in 83). It was revealed that the level of copeptin was significantly higher in patients with MI compared with patients without MI (median 20.8 versus 12.2 pmol / L, $P < 0.0001$). Tn I levels less than 0.04 ng / ml and copeptin levels less than 14 pmol / L had a high negative predictive value (97.3%) for the diagnosis of MI. Similar results were obtained in the extensive multicenter study COPED-MIRRO (2014) [15]. The study included patients admitted to the admission departments of hospitals with a diagnosis of ACS in the first 12 hours from the onset of symptoms of the disease with an initial ECG unchanged and negative Tn values. The second blood sampling for Tn was 6 hours later. The number of patients was 1018. The negative predictive value of copeptin in the diagnosis of MI was 94.2% and was higher in patients over 70 years old (95.1% and 92.6%, $P < 0.05$), and with a disease duration of less than 6 hours (97.8% vs. 93.9%, $P < 0.01$). In conclusion, the authors of the studies summarize the conclusion that the negative predictive value of both biomarkers makes it possible to reliably exclude MI. Conclusion: The key point of all studies is that the specific period of time during which it is necessary to determine copeptin in patients with ACS remains insufficiently open and understandable. Also, the work carried out to this day has not yet provided any information about the possibility of using copeptin for the treatment of patients with MI in order to improve the prognosis. The actual question remains open and requires further research.