CLINICAL SIGNIFICANCE OF CARDIAC TROPONINS IN SEPSIS

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КЛИНИЧЕСКОЕ ЗНАЧЕНИЕ СЕРДЕЧНЫХ ТРОПОНИНОВ ПРИ СЕПСИСЕ

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Annotation. The article is devoted to a review of the literature, which examines the diagnostic value of cardiac troponins in patients with sepsis. The most studied mechanisms of increasing the concentration of cardiac troponins in septic patients in the absence of myocardial infarction and severe coronary heart disease are considered. Much attention is paid here to the possibility of using troponins for risk stratification and prognosis of patients.

Key words: cardiac troponins, myocardial infarction, sepsis, clinical significance.

Аннотация. Статья посвящена обзору литературы, где рассматривается диагностическое значение сердечных тропонинов у пациентов сепсисом. Рассматриваются наиболее изученные механизмы повышения концентрации кардиальных тропонинов у септических пациентов в отсутствии инфаркта миокарда и выраженной ишемической болезни сердца. Большое внимание здесь

уделено возможности применения тропонинов для стратификации риска и прогноза пациентов.

Ключевые слова: сердечные тропонины, инфаркт миокарда, сепсис, клиническое значение.

Introduction. Troponin proteins are the most important regulators of myocardial contractile function, among them the cardiac isoforms of troponins T and I are considered the most specific biomarkers of myocardial infarction among all known to date. However, since the invention of the very first troponin immunoassays in the 90s. There have been numerous reports of the detection of elevated levels of cardiac troponins in a number of other pathological conditions not associated with myocardial infarction [1, 4, 15].

According to many researchers who have studied troponin levels in patients with sepsis and septic shock, the percentage increase in cardiac troponin levels averages 40-80%. At the same time, there were no signs of pronounced changes in the coronary vessels according to the results of coronary angiography, which indicates a predominantly non-ischemic etiology of cTnT and cTn I elevation [2, 5, 16].

A close relationship was established between the degree of endotoxinemia and a decrease in ejection fraction according to echocardiography, which was accompanied by directly proportional increased values of c TnT and I. It was shown that in most patients, high concentrations of troponins were closely correlated with an unfavorable prognosis, and convalescence was accompanied by normalization of ejection fraction and levels troponins [2, 7, 11].

Fenton K.E. and colleagues studied the kinetics of cTnI concentrations in 23 children with sepsis, conducting serial measurements from the moment of admission and during 72 hours of hospital stay. At the same time, cTnI values were elevated in more than half of the patients; by 12 hours after admission they reached peak values and were approximately 10 times higher than the upper reference limit. An increase in troponins was associated with a decrease in systolic cardiac function, the severity of sepsis, and an unfavorable outcome. In children with the most favorable prognosis, there was then a gradual decrease in cTnI concentration, and by day 3, some of them completely returned to normal [2, 8, 12].

Sheyin O. et al. confirmed the important prognostic value of biomarkers of myocardial damage in their meta-analysis, which included 1857 patients included in 17 studies [2, 14, 16].

However, there are studies in which researchers did not find a relationship between troponin concentrations and patient prognosis.

The invention and improvement of highly sensitive methods for determining troponins is associated with the acceleration of the diagnosis of MI.

At the same time, despite the important advantages of modern highly and ultrasensitive immunoassays for troponins, there are also disadvantages. An increase in the sensitivity of the methods has led to a slight decrease in specificity, which is expressed in the form of a larger percentage of cases of elevated levels of cTnT and cTnI outside the established diagnosis of myocardial infarction and, accordingly, leads to difficulty in differential diagnosis. Thus, in a study by Rosjo H. Et al. 2011, compared moderate- and high-sensitivity troponin T in 207 patients with established sepsis without CAD. The hs-TnT level was elevated in 80% of patients, while the normal cTnT level was elevated in only 42%. At the same time, elevation of the hs-TnT value was recorded more often and reached greater heights in patients with severe sepsis (septic shock) and was an independent predictor of mortality [1, 13].

The causes and mechanisms of increased cTnT and cTnI remain the subject of debate, on the basis of which several hypotheses and theories have been put forward. Some researchers believe that direct damage to cardiomyocytes is caused by circulating inflammatory factors (tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), interleukin-6 (IL-6) and a number of others), the concentration of which is in serum levels during sepsis increase tens and hundreds of times. Kumar A. and colleagues conducted an experimental study: they injected plasma obtained from patients with sepsis, in which inflammatory cytokines were elevated, into healthy rats and noted myocardial depression, expressed as a decrease in the frequency and amplitude of cardiomyocyte contractions.

It is worth noting that septic shock is often accompanied by the occurrence of renal failure, in which glomerular filtration is impaired, which is considered an additional mechanism for increasing cardiac troponins, as demonstrated in their study by Wilhelm J. et al. [3, 9, 15]. The authors compared groups of patients and found that patients with uncomplicated sepsis had lower prevalence and troponin concentrations than patients with severe sepsis complicated by renal failure ($p<0.001$). The involvement of the kidneys in the elimination of troponins was confirmed in the large-scale CRIC cohort study, which included 2464 patients suffering from chronic renal failure (CRF) and without signs of coronary artery disease. An increase in hs-TnT was recorded in 81% of patients and depended on the glomerular filtration rate (GFR). In patients with end-stage chronic renal failure with a significantly reduced GFR, hs-TnT concentrations were higher than in patients with early stage chronic renal failure [6].

Considering the above, it is worth assuming the simultaneous participation of several pathogenetic mechanisms; the predominance of a specific one depends on the cause that caused sepsis, the degree of its severity, as well as on the presence of concomitant pathologies and complications.

Conclusions. The importance of monitoring myocardial function in patients using troponins is supported by their prognostic value and should be performed in all

patients with sepsis. The very wide range of elevated troponin levels in sepsis is due to several reasons: the severity of sepsis, the cause, the presence of concomitant diseases and complications, as well as the different sensitivity of troponin diagnostics. The differential diagnosis of myocardial infarction and sepsis should be more carefully carried out. Inflammatory cytokines, as well as various bacterial exo- and endotoxins, have variable cardiotoxicity. Identifying the specific causes and mechanisms of increased troponins and myocardial damage in sepsis seems to be an urgent task for subsequent research, since it is necessary for the development of auxiliary targeted therapy to attenuate or prevent the effect of damaging factors on the myocardium.

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