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MODERN BIOMARKERS OF CORONARY HEART DISEASE

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Abstract
An ideal biomarker of acute myocardial infarction (AMI) should be sensitive and specific early in the course of the disease and have a high prognostic value, which could help clinicians to choose the most optimal treatment tactics. To date, there is no ideal biomarker of AMI, which would have all the characteristics at the same time.

Keywords: Intracellular enzyme, acute myocardial infarction, metabolism, glycogenolysis.

Introduction

Glycogen phosphorylase BB isoenzyme (GPBB) Glycogen phosphorylase (GP) is an intracellular enzyme that regulates carbohydrate metabolism by mobilizing glycogen. GP catalyzes the first step of glycogenolysis (glycogen cleavage), which results in the cleavage of the monosaccharide glucose-1-phosphate from this polysaccharide. There are three different GP isoenzymes: GPMM (present in skeletal muscle), GPLL (in liver), and GPBB (in brain and cardiac muscle tissue) [1,2,3]. During myocardial ischemia, the enzyme GPBB is activated and enhances glycogen cleavage. GPBB is released into the bloodstream 2-4 h after myocardial ischemic injury [42, 43]. Early release of GPBB into the blood is a common result of the combination of increased glycogenolysis and increased cell membrane permeability, which is typical for myocardial ischemia and necrosis [4,5,6,7,8]. In the study by N. Singh et al. [4,11,12] found that GPBB was the most sensitive and specific biomarker for the detection of AMI compared to myoglobin and CFK-MB in the first 3-4 h from the onset of chest pain. Thus, GPBB can be used as an additional biomarker for early diagnosis of AMI. Ischemia-modified albumin (IMA) During acute ischemia, the N-terminus of albumin is altered (modified), resulting in a decrease in its binding capacity: the resulting protein is called ischemia-modified albumin [4,5]. The advantage of this biomarker over

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cTn is that positive IMA levels are evident within minutes after ischemia and remain elevated for several hours, even before the development of myocardial necrosis [4,6].

Consequently, a negative IMA result at the initial assessment of the patient's condition indicates a low risk of adverse events in the patient, which provides significant cost savings [7]. In patients with suspected acute coronary syndrome, diagnostic accuracy on admission increased when IMA was used in combination with cTnT and electrocardiography data [8]. In fact, IMA combined with cTnT results is a more sensitive marker for predicting adverse cardiac events than cTnT alone, although the specificity and sensitivity of IMA are too low to be useful for clinical decision making when used as a stand-alone indicator.

Neuroendocrine biomarkers of acute myocardial infarction Natriuretic peptides (BNP / NT-proBNP) Brain natriuretic peptide, or Natriuretic peptide type B (BNP), is a hormone secreted by cardiomyocytes in the ventricles of the heart in response to cardiac stress and ventricular dysfunction. After its synthesis, the proBNP precursor is cleaved into the active hormone BNP, which has a number of functions in humans, and an inactive fragment NT-proBNP (N-terminal prohormone of brain natriuretic peptide). BNP functions include vasodilation, sodium and water excretion (natriuresis), and inhibition of the renin-angiotensin-aldosterone system [5,6]. Elevated BNP levels in patients with AMI have also been found to be associated with the size of the necrosis zone [5,8]. Although BNP/NT-proBNP levels are elevated in patients with acute coronary syndrome, they cannot be used as diagnostic markers because they are also elevated in other conditions that have similar symptoms to AMI, such as heart failure and pulmonary embolism. Because of partial clearance (removal) of BNP and NT-proBNP by renal excretion, high levels of BNP and NT-proBNP are also observed in patients with renal failure [9]. Several studies have demonstrated the high prognostic value of BNP and NTproBNP in patients with AMI. NT-proBNP in plasma measured 2-4 days after the development of AMI independently predicted left ventricular function and one-year survival of patients [6,10].

Elevated BNP levels at initial admission in patients with ST-segment elevation myocardial infarction were associated with impaired reperfusion after fibrinolysis and higher 30-day mortality. After adjustment for cTnI, BNP remained independently associated with patient mortality, and the probability of heart failure and risk of patient death increased with higher baseline BNP concentrations [6,11]. Adrenomedullin (ADM) Adrenomedullin is a regulatory peptide (hormone) whose levels are elevated in the serum of patients with cardiovascular disease. It attenuates the development of infarction during acute myocardial injury and can potentially influence the pathological process both in the acute phase of AMI and as a result of remodeling. Elevated adrenomedullin levels are indicative of cardiac remodeling and may improve risk stratification in patients with heart failure and AMI [6].

Catestatin (CST) is an important peptide that regulates cardiovascular function. Catestatin is an antagonist of catecholamines (dopamine and norepinephrine), showing antihypertensive, antiapoptotic, cardioprotective and hypoglycemic properties [6,7,8,9,10]. According to studies, patients with AMI have higher levels of catestatin than controls. Catestatin concentrations are positively correlated with ventricular remodeling [6,8] and catecholamine levels and, according to the authors, exhibit protective properties in the postinfarction period [6,9].

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Copeptin Copeptin, a small glycopeptide of 39 amino acids, is the C-terminal fragment of preprovasopressin, which is secreted in equimolar amounts with antidiuretic hormone (ADH, vasopressin) into the bloodstream after cleavage in the neurohypophysis. Copeptin is thus a surrogate biomarker of ADH, and its serum levels reflect ADH production by the neurohypophysis [9,70,11,12]. The formation and secretion of ADH and copeptin is analogous to the formation of insulin and C-peptide by pancreatic endocrinocytes. In the bloodstream, ADH is unstable and mainly bound to platelets, while copeptin, on the contrary, is a stable biomarker, which makes it suitable for diagnostic purposes [5,6,7,8,9]. In a recent study by K. Kim et al. showed that the multimarker strategy using copeptin and hs-cTnI was not inferior to serial hs-cTnI measurements in the diagnosis of AMI. Both sensitivity and negative predictive value of the multimarker strategy were equal to 100%.

However, the diagnostic performance of copeptin alone in AMI was limited. The specificity and positive predictive value of the multimarker strategy were lower than that of serial hs-cTnI measurement. According to J. Jeong et al., the diagnostic efficiency of copeptin in the early diagnosis of AMI is higher than that of cTnI. The combination of copeptin and cTnI also has better diagnostic efficiency than the combination of CFK-MB and cTnI in the early diagnosis of AMI [7,8]. M. Budnik et al. [4,5,6] concluded that the copeptin/NT-proBNP ratio can be used in the differential diagnosis of AMI and takotsubo syndrome - a pathological condition similar in clinical symptoms to AMI, which is almost always accompanied by an increase in serum cTn concentration. Thus, the study of copeptin in patients with suspected AMI has some advantages over cTn. Further studies are needed to clarify these possibilities.

Components of the renin-angiotensin-aldosterone system (RAAS) The renin-angiotensin-aldosterone system (RAAS) is a hormonal system that regulates blood pressure and fluid balance in the human body. The main components of the RAAS used as biomarkers of cardiovascular disease are renin, angiotensin II, and aldosterone [1,2,3].

RAAS is activated after AMI, leading to increased blood volume and vasoconstriction. Aldosterone contributes to a wide range of harmful cardiovascular effects during AMI, including acute endothelial dysfunction, increased oxidative stress, cardiac myocyte necrosis, and increased myocardial hypertrophy and fibrosis [7,8]. Except for BNP, the aforementioned neuroendocrine markers are not yet used in clinical practice for diagnosis or prognosis. Nevertheless, studies have shown that treatment of AMI patients with neuroendocrine inhibitors, particularly RAAS inhibitors, reduced morbidity and mortality. For example, mortality and heart failure rates in patients with acute coronary syndrome were reduced when angiotensin-converting enzyme inhibitor/angiotensin receptor blocker and aldosterone inhibitors were administered [7,8,9,10,13]. Given these data, higher levels of these compounds in AMI may indirectly indicate an unfavorable prognosis.

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