MAIN MODERN LABORATORY BIOMARKERS OF ACUTE MYOCARDIAL INFARCTION

Sarang Meshram *- student of the 4th course international education faculty;*
Supervisor: Kudratova Z.E*.- PhD, associate professor of the department clinical laboratory diagnostics with the course of clinical laboratory diagnostics of the PGD;*
Samarkand state medical university
Samarkand, Uzbekistan

Abstract. The morbidity and mortality rates of acute myocardial infarction (AMI) have been increasing rapidly in recent years, causing significant socioeconomic damage. Cardiac-specific biomarkers play an important role in the diagnosis and prognosis of acute myocardial infarction. Current cardiac biomarkers of AMI can be divided into several groups: biomarkers of cardiomyocyte necrosis and ischemia, neuroendocrine biomarkers, inflammatory biomarkers, and a number of new biomarkers, the diagnostic value of which is still poorly understood.

Keywords: laboratory diagnosis, acute myocardial infarction, biomarkers, cardiac troponins, myoglobin, albumin;

To date, there are many cardiac status indicators known to have diagnostic and prognostic value in AMI, but none of them simultaneously meets the two key criteria of an ideal biomarker of AMI - absolute cardiac specificity and high sensitivity, confirming insignificant myocardial damage, which allows an accurate diagnosis and adequate treatment in the early stages of acute infarction. Conventionally, all known cardiac markers can be divided into several groups.

I. Biomarkers of cardiomyocyte necrosis and ischemia Cardiac troponins (cTnI, cTnT, cTnC) Troponin proteins are important for the interaction of actin and myosin, and regulation of muscle tissue contractile function in response to cytosolic calcium and troponin phosphorylation. The troponin complex is located together with tropomyosin on the actin filament. Cardiac-specific isoforms cTnI and cTnT exist in myocardial tissue, whereas cTnC is also expressed in skeletal muscle, which makes it unsuitable for use as a biomarker of AMI [11,12,13,14].

For the diagnosis of myocardial infarction, according to the third universal definition of myocardial infarction, the following laboratory criteria are used: an elevation greater than the 99th percentile (upper reference limit) and/or a pattern of decreasing serum cTn levels; in addition, cTn levels should be measured with a coefficient of variation ≤ 10% [14,15,16]. Advances in technology and the

development of highly sensitive assays (hs-cTn) have improved the identification of myocardial injury with the ability to detect elevated troponin levels within the first hours of symptomatic cardiac ischemia [1,2,3]. Although elevated serum cTn levels reflect myocardial injury, the mechanistic basis for this observation remains unclear. In addition to spontaneous AMI after acute coronary occlusion and rupture of atherosclerotic plaques, cardiac necrosis may be secondary to ischemia resulting from increased oxygen demand or decreased oxygen delivery due to coronary embolism, coronary artery spasm, arrhythmias, hypertension, severe anemia, and respiratory distress.

Consequently, cTn levels may be elevated not only in ischemic heart disease and myocardial infarction but also in other noncardiac disorders (Figure 1) [4,5,6]. One of the important factors contributing to the increase of cTn concentration in blood is the rate of their elimination, including renal filtration. Indeed, cTn levels are elevated in patients with renal failure without symptoms of acute coronary syndrome, although they are at increased risk of cardiac abnormalities. Highly sensitive detection methods are able to register minor and reversible myocardial damage occurring in some physiological, as well as in the initial stages of a number of pathological conditions [7,8,9], which significantly expands the possibilities of their use. High cTn level is useful for diagnostic purposes and is also an independent prognostic marker, as evidenced by several clinical trials and metaanalysis [10,11].

cTn levels may inform clinical decision-making in terms of whether to adopt a more aggressive or conservative course of treatment for acute coronary syndrome, as abnormal cTn levels may identify subgroups of patients who benefit most from early invasive therapy. With the development of highly sensitive methods for cTn determination, noninvasive diagnosis of cardiovascular disease has also become possible. Myoglobin (Mb) Myoglobin is a low molecular weight cytoplasmic heme protein that is the most sensitive common biomarker of AMI.

Due to its low molecular weight, myoglobin is much more rapidly released from cardiomycytes and can be detected in the blood 1 h after myocardial injury, peaks within 4-12 h, and then returns to baseline within a day [3,5,16]. However, myoglobin has lower specificity for cardiac necrosis than cTnI and cTnT, and myoglobin levels may be elevated in noncardiac disorders, such as skeletal muscle disease or injury, as well as in chronic kidney disease [3,6,7]. Despite the lack of cardiac specificity, the combination of myoglobin with cTnI or cTnT has significantly improved the ability to identify individuals at increased risk of AMI mortality compared with each of these biomarkers alone [3,8,9]. Myoglobin is excreted primarily through the kidneys, and renal impairment is considered a predictor of adverse outcome, including increased risk of mortality, in patients with AMI [4,10].

Thus, it is hypothesized that myoglobin predicts mortality by identifying patients

with renal failure.

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