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The Recent Advances in the Serological Detection of Acute Myocardial Infarction

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Abstract

Acute Myocardial Infarction (AMI) is one of the leading causes of morbidity and mortality worldwide. The greatest risk of fatality occurs within the first hours of initiation of AMI. Thus, the early diagnosis of cardiac ischemia is fundamental for the effective management of AMI patients. Inadequate diagnosis of patients with chest pain often leads to inadequate admission of patients without AMI and vice versa. In addition to the clinical history, physical examination, accurate electrocardiogram findings, and evaluation of cardiac biomarkers play an important role in the early diagnosis of acute ischemia. The present review discusses in detail the various cardiac biomarkers released during the event of an AMI.

Keywords: Acute myocardial infarction; Electrocardiogram; Atherosclerosis; Biomarkers

Introduction

Acute Myocardial Infarction (AMI), commonly known as a heart attack is the result of a reversible or irreversible ischemia caused by interruption of the supply of oxygen-laden blood to certain areas of the heart. This interruption leads to apoptosis of cardiac cells, which release into the extracellular environment a variety of intracellular constituents such as organelles, cytosolic enzymes and biologically active structural proteins that are subsequently considered biomarkers for diagnosis and / or prognosis of injury [1]. In the majority of cases, AMI leads to severe heart failure and is therefore considered one of the leading causes of morbidity and mortality worldwide. A large part of the population is susceptible due to a range of risk factors such as advanced age, *Diabetes mellitus*, hypertension, renal insufficiency, hemodynamic instability, compromised left ventricular systolic function and the presence of atheroma plaques in large arteries that can consequently lead to atherosclerosis [1,2].

Initially, cardiac tissue damage, ranging from mild to severe, leading to heart failure, was identified primarily by electrocardiogram (ECG) and serological markers with late identification windows that presented low specificity, representing not only the heart as also skeletal muscle and other organs such as lung, kidney and liver [3]. Several researches report a multitude of biomarkers subdivided into six categories: Markers of inflammation; Markers of renovation and remodeling of matrices; Biochemical markers; Neuro-hormonal markers; Nutrition and metabolism markers; and finally, cardiac muscle cell markers.

The strategies of detection of acute and chronic myocardial infarction with the advent of the discovery of new markers are usually composed of electrocardiographic examination, cTnI and cTnT cardiac troponins, as well as other markers such as myoglobin, creatine kinase (CK), MB Creatine kinase (CK-MB), and more recently ischemia-modified albumin (IMA), which is a prognostic marker of cardiac lesions [1,4,5].

This article aims to review the application of cTnI and cTnT cardiac troponins as a serological and prognostic marker in order to clarify some aspects related to its wide use.

Literature Review

Markers of myocardial lesions

During the last decades, a multitude of biomarkers were being discovered and characterized, however, they were sometimes not sensitive and specific enough to be applied solely and exclusively as cardiac markers. With the advent of new discoveries, it was necessary to classify these markers so that they reached three essential criteria for their application in clinical diagnosis. These criteria consist of: 1) Reliability, speed and low cost; 2) Provide additional information that physicians do not obtain through physical examination; 3) Ability of the biomarker to influence the decision making of the responsible physician [6]. Over the years, despite the established criteria, many markers were discovered and characterized, some were considered almost exclusively for the diagnosis of cardiac tissue damage.

Inflammatory markers

The measurement of inflammatory markers is an important component of prognostic and diagnostic strategies for AMI. Inflammatory cytokines are believed to act actively in the progression of myocardial events [7]. A number of molecules have been studied, including: C-reactive protein (CRP), myeloperoxidase, TNF, IL-6, selectin, VCAM-1, VEGF, PIGF, EGF, among others.

C-reactive protein

CRP is an important inflammatory marker directly involved in the immune response. It acts as a mediator and amateur for inflammatory processes. Since 1990, this molecule has been associated with a variety of cardiovascular diseases, and its correlation with atherosclerosis, Coronary Artery Disease (CAD), Acute Coronary Syndrome (ACS) and AMI has now been studied, since its role in the prognosis of cardiac events has already been proven [8].

Myeloperoxidase

Peroxidases are important molecules that act in the detoxification process neutralizing some Reactive Oxygen Species (ROS) like hydrogen

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peroxide. This class of molecules are important antioxidant agents, and when they are in less quantity than ROS give rise to oxidative stress, which negatively affects endothelial functions and the cardiovascular system. Reactive oxygen species are extremely difficult to measure, and because of this, it was necessary to identify their reducing agents, such as myeloperoxidase, the most studied class on this group of markers [9].

TNF

As well as CRP, tumor necrosis factor, or TNF, has been studied since the year 1990. Since then, it is known that it is elevated in AMI events; however, its role is still little known in prognosis and evolution framework. It is known that TNF molecules and their sTNF-RI and sTNF-RII receptors are elevated in chronic cardiac events, in addition to having a mild inotropic effect on the cardiac muscle; however, there are studies that suggest that these same molecules become cardio-depressive after some time [10].

IL-6

Interleukin (IL)-6 is a pleiotropic cytokine with a broad range of humoral and cellular immune effects relating to inflammation, host defense, and tissue injury [11,12]. Produced in response to several factors, including infection, IL-1, interferon-γ, and tumor necrosis factor [13-15] IL-6 is a central mediator of the acute-phase response and a primary determinant of hepatic production of C-reactive protein [16,17]. Although elevated levels of IL-6 have been reported in some chronic inflammatory conditions [12], epidemiological data evaluating the potential role of IL-6 in early atherogenesis are sparse. However, experimental studies indicate that vascular endothelial and smooth muscle cells from normal and aneurysmal arteries produce IL-6, [18-20] that IL-6 gene transcripts are expressed in human atherosclerotic lesions [21,22], and that IL-6 may have procoagulant effects [23-25]. Furthermore, prospective studies of apparently healthy as well as high-risk individuals indicate that elevated levels of C-reactive protein, a potential surrogate for IL-6 activity, are associated with first coronary and cerebrovascular events. Finally, elevated levels of IL-6 and other acute-phase proteins have been reported among patients with acute coronary syndromes [26], even among those without overt plaque rupture or acute tissue trauma [27,28]. Interluekin-6 (IL-6) is released from skeletal muscle cells and induced by exercise, heat, catecholamine, glucose, lipopolysaccharide, reactive oxygen species, and inflammation and heat increases IL-6 in skeletal muscle cells through the TRPV1, PKC, and CREB signal transduction pathway [29].

Selectin

Leucocyte adhesion and subsequent migration across the endothelium is pivotal to the development of coronary atherosclerosis [30]. Their initial attachment to endothelial cells is mediated by cell adhesion molecules, including the selectin family. E-selectin is synthesized by endothelial cells in response to stimulation by interleukin-1 and tumour necrosis factor-alpha [31]. It binds to a ligand on the cell surface of leucocytes, causing the leucocytes to 'roll' across the endothelium [32]. Inhibition of neutrophil adhesion has been shown to limit myocardial infarct size and reduce myocardial reperfusion injury in animal models [33]. Soluble E-selectin is potentially useful as a biochemical marker as it can be easily measured by ELISA techniques and is stable under laboratory conditions [34]. Several authors have reported raised E-selectin levels in AMI [35-40]. P-selectin, a cellular adhesion molecule of platelets and endothelial cells stored in both the α-granules of platelets and in the Weibel-Palade bodies of endothelial cells [41,42], is rapidly expressed on the surface of activated platelets and endothelial cells [43,44]. It is involved in mediating platelets and the rolling of leukocytes on activated endothelial cells [45,46] as well as in interactions of activated platelets with leukocytes. Other studies also demonstrated that the size of the aggregated platelets and the immobilization of platelets on the clots depend on P-selectin [47].

Matrix remodeling markers

After reversible or irreversible ischemia, there is a need in the cardiac tissue to remodel injured areas in order to reconstruct the cardiac pump structurally and functionally. During this process, it is possible to identify several biomarkers that actively act, such as procollagen, laminin, tenascin, and metalloproteinases, among others.

Pro-collagen

Collagen plays an important role in the remodeling of cardiac tissue after an AMI event; however, the way in which such remodeling takes place, replacing areas damaged by fibrosis, has an important consequence, heart failure, due to loss of function of the remodeled areas. Since its presence is certain after an AMI event, collagen has been studied as an important marker for determining the extent of cardiac fibrosis. In the procollagen class, the most studied is type III (PIIINP), where it has been found to be elevated in patients with AMI events when compared to healthy patients [48].

Laminin

Laminins, the first extracellular matrix (ECM) glycoproteins detectable in the embryo, are found in basement membrane. Laminins consist of three peptide chains: α, β, and γ. Laminin proteins is detected in the infarct area day 3 post-MI, peaks in concentration at days 7-11, and then returns to baseline levels [49]. The wide existence of laminins throughout the infarct area suggests that they may directly regulate left ventricle repair post-MI. In patients with AMI, serum laminin level is higher than in patients with stable coronary artery disease and those without coronary artery disease [50]. This report suggests the possibility that serum laminin could be potential prognostic marker for MI patients.

Tenascin-C

Tenascin-C is an oligometric glycoprotein exclusively expressed in the chordae tendineae and base of valve leaflets in the normal heart. In animal models of MI, tenascin-C expression can be detected in the infarct border zone, and is thought to loosen the strong adhesion of surviving cardiomyocytes to connective tissue [51]. Accordingly, tenascin-C aggravates left ventricle remodeling and dysfunction after MI in mice; its deletion attenuates adverse left ventricle fibrosis and dysfunction, without affecting infarct sizes or survival rates [52]. *In vitro*, tenascin-C fosters fibroblast migration and differentiation, and collagen gel contraction [53]. In patients with MI, serum concentration of tenascin-C positively correlates with the incidence of adverse cardiac remodeling and worse clinical outcomes [54-56].

Metaloproteins

Metalloproteinases are a group of endopeptidases involved directly in the degradation of extracellular matrix proteins and in the decomposition of molecules that are involved in chemoactivation. Tissue inhibitors of metalloproteinases inhibit these molecules and it has been suggested that an imbalance between PMMs and TIMps plays a role in ventricular remodeling [57].

Biochemical markers

Natriuretic peptides: Natriuretic peptides are a group of neurohormones that affect body fluid homeostasis via natriuresis

and diuresis. Natriuretic peptides also decrease vasoconstriction by decreasing the synthesis of angiotensin II and norepinephrine [58]. There are two main types of natriuretic peptides: BNP and natriuretic peptide type A (ANP). The most widely studied of these peptides is BNP. BNP is synthesized as a pre-prohormone and is released in response to volume overload and wall stress, which made BNP an attractive target to aid in the diagnosis of heart failure [59].

Growth differentiation factor 15: Growth differentiation factor 15 (GDF-15) is a member of the TGF-β superfamily. In healthy individuals, GDF-15 is only expressed in the central nervous system and placenta [60]. However, many tissues can express GDF-15, including the heart, in response to lesions, hypoxia or exposure to cytokines. At heart, GDF-15 was found to have anti-hypertrophic effects [61,62]. *In vitro* studies have shown that GDF-15 is highly expressed after exposure to low-density oxidized lipoprotein and GDF-15 expression has been discovered as co-localized in human arteriosclerotic blood vessels [63], suggesting that GDF-15 is highly expressed in atherosclerosis and probably in CAD.

Neurohormonal markers: The neurohormones and mediators activated in heart failure can be grouped into two major groups that have opposite activity [64]. First, those which increase contractility and heart rate produce peripheral vasoconstriction, promote liquid retention, and, in the tissues, induce proliferative responses. This activity is mediated by the increase in sympathetic activity and activation of the renin-angiotensin-aldosterone system, vasopressin and endothelin. Second, other mediators, such as natriuretic peptides, adrenomedullin and cytokines, induce opposite responses and cause vasodilation and diuretic effects, reduce cellular proliferation and induce apoptosis. High plasma concentrations of some of these mediators, such as norepinephrine and angiotensin, directly contribute to increasing mortality in heart failure, whereas it is believed that other hormones are only indirect markers of greater severity [65,66].

Although in clinical practice determining neurohormone values in serum has been of limited use, knowledge concerning them has made it possible to improve and develop new treatments for heart failure.

Myocardial markers

Troponins: Troponins are a protein complex that make up the regulatory system of myosin-dependent calcium interaction with actin in the contraction of the cardiac and skeletal muscle [3]. They are subdivided into three different isoforms, known as I, C and T, where the subtype cTnC is not applied as a diagnosis and prognosis of AMI because they are encoded by the same gene sequences in both skeletal muscle and cardiac muscle. However, unlike cTnC, the most commonly used markers in most cases where suspected AMI are cTnI (cardiac troponin I) and cTnT (cardiac troponin T), which are specific for heart muscle, as they are encoded by different genes and therefore give rise to immunologically distinct proteins [2,67]. Such knowledge about the cTnI and cTnT isoforms allowed the development of extremely low cross-reactivity monoclonal antibodies specific for cardiac isoforms facilitating the diagnosis of AMI [3].

Commonly aimed at the detection of AMI, cTnI and cTnT isoforms are present at extremely high levels when there is an episode of myocardial injury. After an event, cTnI troponin usually peaks within about 1 day, and troponin cTnT tends to reach it around 3-4 days after the initiation of AMI, both of which remain elevated by 4 to 5 days [68].

Troponin I: Cardiac troponin I (cTnI) is uniquely different from troponin I present in skeletal muscle fibers. Cardiac isoform I is not expressed in human skeletal muscle during fetal development, after skeletal muscle trauma or during regeneration of this type of muscle, since it is not encoded by the same gene [69]. Because of this specificity, most authors point to cardiac isoform I as the marker that is closest to the ideal for evaluation of specific cardiac lesions in children and adults [70].

In acute heart failure, small troponin elevations have occurred in approximately 20-50% of patients, a consequence of myocardial stress due to inflammation, oxidative stress, and neurohormonal activation [71]. Other possible mechanisms of elevation of troponin would be coronary hypo perfusion secondary to low cardiac output and elevation of intracavitary pressures, with consequent reduction of coronary perfusion pressure. Troponin I is also elevated in a large percentage of patients with acute heart failure without acute coronary obstructions and in some studies, has been shown to be an independent predictor of mortality [72]. Another application of troponin I is in the diagnosis of trauma and suspicion of traumatic cardiac contusion without the false positives presented when using CK-MB for this purpose. Its use in suspected cases of recent cocaine use is also important, since the specificity of myoglobin and CK-MB are strongly affected and of troponin does not suffer any type of interference [73].

Troponin I is increasingly gaining acceptance as a serum marker of choice in the risk stratification of acute coronary syndromes. It can be detected in the peripheral blood even when there has not been an elevation of CK-MB levels. It is added the fact that there is a direct quantitative relationship between troponin level and adverse outcome. Some patients may present normal CK-MB, but troponin is already detectable, denoting minor myocardial injury, which leads these patients to be considered as high risk [74]. It is worth remembering that troponin, regardless of whether T or I, is usually positive only about four to six hours after the onset of symptoms and remains so for about two weeks, which makes it difficult to diagnose re-infarction by this marker [75].

Troponin T: T isoform is expressed to a lesser degree in skeletal muscle and some data indicate that there are at least some patients with skeletal muscle lesions who have proteins that are detected by the antibodies in the cTnT and HS-cTnT (ultra-sensitive) assay. This implies that skeletal muscle may in some patients be the source in case of elevation of cTnT detected in the blood (REF.). However, this problem had only been reported about the T isoform, continuing the cTnI protein, being widely applied as a specifically cardiac marker [68]. Therefore, in most clinical situations, its specificity should be comparable to that of cTnI.

Troponin test: According to the American College of Cardiology (ACC) and the European Society of Cardiology (ESC), AMI should be diagnosed if cTnI or cTnT levels are greater than 99%, with a coefficient of variation of 10% or less, detected within 24 h after the event clinical index [76]. Values in the intermediate zone suggest reduced myocardial damage [77]. Large infarctions are considered when cardiac troponin levels are greater than 99% and when the CK-MB fraction is elevated in the presence of ischemic symptoms. Microinfarcts are considered when the cardiac troponins level is greater than the 99% with a normal CK-MB fraction [3].

Nanoparticles: In a recent study, Kim et al. developed a nanosensor coated gold sun particles. Such a system was able to detect extremely low troponin levels, the lowest value being found so far, and still about 8 times lower than the recommended value for detecting AMI [78].

Conclusion

The analysis of cardiac biomarkers has become the first line of diagnostic tools for AMI, and has greatly enabled physicians in the

rapid diagnosis and immediate treatment planning, thus reducing the mortality rate to a large extent. However, the future of cardiac biomarkers will follow the analysis of a panel of markers for the diagnosis and prognosis of myocardial infarction.

Competing Interests

None of the authors have any competing interests.

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