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Commentary

Cardiac Biomarkers and their Relationship with Myocardial Metabolism

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DESCRIPTION

Cardiac biomarkers are measurable substances released into the bloodstream that reflect physiological or pathological processes occurring within the heart. Traditionally, these biomarkers have been used to diagnose myocardial infarction, assess cardiac injury, and stratify patient risk. However, beyond their diagnostic utility, cardiac biomarkers provide valuable insight into the metabolic state of the myocardium. Because the heart is a metabolically demanding organ, changes in myocardial metabolism are often mirrored by alterations in circulating biomarkers. Understanding this relationship enhances interpretation of biomarker elevations and deepens knowledge of cardiac pathophysiology.

The myocardium relies on continuous energy production to sustain contractile function. Under normal conditions, cardiac cells generate adenosine triphosphate primarily through oxidative phosphorylation, with fatty acids supplying most of the energy substrate. Glucose, lactate, ketone bodies, and amino acids contribute variably depending on physiological demands. This metabolic flexibility allows the heart to adapt to changes in workload and substrate availability. Any disruption in this finely balanced system results in metabolic stress, which may manifest as the release of specific cardiac biomarkers.

One of the most widely used cardiac biomarkers, cardiac troponin, illustrates the link between metabolism and cellular integrity. Troponins are structural proteins involved in calcium-mediated muscle contraction. Under metabolic stress, such as ischemia or hypoxia, reduced Adenosine Triphosphate (ATP) availability impairs calcium handling and contractile regulation. This leads to structural destabilization of the sarcomere and increased membrane permeability, allowing troponin molecules to leak into the circulation. Even minor metabolic disturbances that do not cause irreversible necrosis may result in low-level troponin release, highlighting its sensitivity to metabolic injury rather than cell death alone. Myoglobin is another early biomarker of myocardial injury with metabolic relevance. As an oxygen-binding protein within muscle cells, myoglobin facilitates intracellular oxygen transport and storage. During ischemia, oxygen deprivation alters myoglobin function and promotes its release from damaged cells. Although not cardiac-specific, elevated myoglobin levels reflect acute disturbances in myocardial oxygen metabolism and often precede the rise of more specific biomarkers.

Metabolic stress in the myocardium also influences the release of natriuretic peptides, particularly B-type natriuretic peptide and its inactive fragment NT-proBNP. These biomarkers are synthesized in response to increased wall tension and myocardial stretch, conditions often linked to altered energy utilization. In heart failure, inefficient myocardial metabolism leads to reduced contractile performance, increased ventricular filling pressures, and subsequent natriuretic peptide release. Thus, elevated natriuretic peptides serve as markers of both hemodynamic burden and underlying metabolic inefficiency. Emerging cardiac biomarkers provide even more direct connections to myocardial metabolism. Heart-type fatty acid-binding protein is involved in intracellular fatty acid transport and oxidation. During ischemia or metabolic overload, disruption of fatty acid metabolism leads to the release of this protein into circulation. Elevated levels reflect early metabolic injury and impaired substrate utilization within cardiomyocytes, making it a sensitive indicator of ischemic metabolic stress.

Lactate and related metabolic byproducts, although not traditional cardiac biomarkers, offer insight into myocardial metabolic shifts. Increased lactate production occurs when the heart transitions from aerobic to anaerobic metabolism due to insufficient oxygen supply. Elevated systemic or coronary sinus lactate levels indicate impaired oxidative phosphorylation and increased reliance on glycolysis. When interpreted alongside cardiac-specific biomarkers, lactate levels help distinguish metabolic stress from structural damage. Oxidative stress biomarkers also bridge the gap between myocardial metabolism and injury. Reactive oxygen species are generated as byproducts of mitochondrial respiration, particularly during ischemia and reperfusion. Biomarkers such as malondialdehyde and oxidized lipoproteins reflect lipid peroxidation and mitochondrial dysfunction. While not exclusive to the heart, their elevation in cardiac conditions underscores the metabolic origins of myocardial injury.

Inflammatory biomarkers further reflect metabolic disturbances. C-reactive protein and interleukin-6 increase in response to

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myocardial injury but are also influenced by metabolic dysfunction and oxidative stress. Inflammation alters substrate utilization, impairs insulin signaling, and exacerbates energy inefficiency in cardiomyocytes. Thus, inflammatory biomarkers indirectly represent metabolic derangements accompanying cardiac disease. The temporal patterns of cardiac biomarkers provide additional metabolic information. Early biomarker release often reflects reversible metabolic stress and membrane permeability changes, whereas sustained elevations suggest prolonged metabolic failure and structural damage. High-sensitivity assays have revealed that even transient metabolic disturbances can produce measurable biomarker fluctuations, challenging the traditional binary interpretation of normal versus pathological values.

CONCLUSION

Cardiac biomarkers are not merely indicators of myocardial injury but reflections of underlying metabolic processes within the heart. Alterations in energy production, substrate utilization, oxygen handling, and mitochondrial function influence the release and kinetics of these biomarkers. Appreciating their metabolic context enhances diagnostic precision and provides deeper insight into the pathophysiology of cardiac disease. As research advances, integrating metabolic biomarkers with traditional cardiac markers may lead to more personalized and effective approaches to cardiovascular care.