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SPECIFICITY OF BIOCHEMICAL BLOOD ANALYSIS IN MYOCARDIAL INFARCTION

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Abstract

This article analyzes the specificity of biochemical blood markers in the diagnosis and monitoring of myocardial infarction (MI). Special attention is given to the kinetics, diagnostic accuracy, and clinical relevance of cardiac-specific enzymes and proteins, including troponins, creatine kinase-MB, myoglobin, and emerging novel biomarkers. The discussion highlights the advantages and limitations of these markers in differentiating myocardial infarction from other cardiac and non-cardiac pathologies, and the role of multimodal biomarker strategies for improving diagnostic precision in acute coronary syndromes.

Keywords: Myocardial infarction, cardiac biomarkers, troponin, creatine kinase-MB, myoglobin, diagnostic specificity, acute coronary syndrome, biochemical analysis, laboratory diagnostics.

INTRODUCTION

Myocardial infarction (MI) remains one of the leading causes of morbidity and mortality worldwide, necessitating rapid and accurate diagnostic methods for effective clinical management. While electrocardiography and clinical symptoms provide initial suspicion, definitive diagnosis heavily relies on biochemical blood analysis. The identification of specific biomarkers released into the bloodstream due to myocardial cell injury is crucial in confirming myocardial infarction, stratifying risk, and monitoring therapeutic interventions. However, the specificity of different biomarkers varies, and their interpretation requires a nuanced understanding of biochemical kinetics and differential diagnoses. This article critically examines the biochemical markers used in MI diagnosis, emphasizing their specificity, dynamics, and clinical implications.

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MATERIALS AND METHODS

Biochemical analysis of blood in the setting of suspected myocardial infarction focuses primarily on detecting the release of intracellular proteins and enzymes from necrotic cardiac myocytes. Among these, cardiac troponins — specifically troponin I (cTnI) and troponin T (cTnT) — have emerged as the gold standard due to their superior specificity for myocardial tissue. Troponins begin to rise within 3 to 6 hours after myocardial injury, peak at 12 to 24 hours, and remain elevated for up to 10–14 days. Their high tissue specificity means that elevated troponin levels are strongly indicative of myocardial necrosis, although certain non-ischemic conditions such as myocarditis, renal failure, and sepsis can occasionally produce elevated levels, thereby challenging absolute specificity in some clinical contexts.

RESULTS AND DISCUSSION

Creatine kinase-MB isoenzyme (CK-MB) historically played a pivotal role before the advent of troponin assays. CK-MB levels rise within 4–6 hours post-infarction, peak at 12–24 hours, and return to baseline within 48–72 hours. Although CK-MB is more specific to cardiac muscle than total creatine kinase (CK), it is not exclusively cardiac-specific; elevated levels can occur with skeletal muscle injury or chronic muscle disease, reducing its specificity in certain patient populations. Nevertheless, CK-MB remains clinically valuable for detecting reinfarction due to its relatively short half-life compared to troponins.
Myoglobin, a small heme protein, is one of the earliest markers to rise — often detectable within 1–2 hours after myocardial injury. However, its lack of cardiac specificity severely limits its diagnostic utility; myoglobin elevations occur in a wide range of skeletal muscle injuries, burns, and even intense physical exercise. Despite this limitation, the high sensitivity of myoglobin in the very early phase of MI makes it useful in ruling out myocardial infarction when combined with more specific markers.

Newer biomarkers are currently under investigation to enhance specificity and improve early diagnosis. Heart-type fatty acid-binding protein (H-FABP) shows rapid release kinetics similar to myoglobin but with slightly greater cardiac specificity. Glycogen phosphorylase isoenzyme BB (GPBB) and copeptin have also attracted interest for their potential roles in early detection. However, none have yet supplanted troponins in terms of clinical acceptance [1].

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The specificity of biochemical markers is also influenced by the assay sensitivity and cutoff values used. High-sensitivity troponin (hs-Tn) assays allow detection of minimal myocardial injury, but at the cost of increased detection of clinically insignificant elevations. This phenomenon necessitates careful interpretation within clinical context, integrating timing of symptoms, electrocardiographic changes, and risk factors.

Moreover, multi-marker strategies that combine different biomarkers are gaining attention to improve diagnostic performance. For instance, combining hs-troponin with copeptin, a marker of stress response, provides higher negative predictive value for ruling out MI early in emergency settings. This approach compensates for the delayed rise of troponins by leveraging the immediate response of copeptin, enabling faster decision-making and patient triage.

In differential diagnosis, it is crucial to recognize conditions that mimic MI biochemically. Elevated troponins are observed in pulmonary embolism, heart failure, stroke, and critical illnesses. Renal dysfunction, in particular, poses significant challenges, as reduced clearance of troponin fragments can result in chronically elevated baseline levels. Therefore, interpreting biochemical results requires integration with clinical findings and imaging studies to avoid misdiagnosis and unnecessary invasive procedures [2].

Additionally, the timing of blood sampling critically affects the sensitivity and specificity of biomarker interpretation. Serial sampling protocols — typically obtaining blood at admission, 3 hours, and 6 hours — increase diagnostic accuracy by identifying dynamic changes consistent with acute injury rather than relying on a single measurement.

Overall, while troponins remain the cornerstone of biochemical diagnosis in myocardial infarction due to their unparalleled specificity for myocardial tissue, their interpretation is not straightforward. A comprehensive approach that considers kinetics, patient history, comorbidities, and assay limitations is essential for maximizing diagnostic specificity and improving patient outcomes.

Beyond conventional cardiac markers, the specificity of biochemical blood analysis in myocardial infarction must be understood in the broader context of myocardial pathophysiology and evolving diagnostic technologies. The process of myocardial infarction initiates a complex cascade of cellular events, beginning with acute ischemia and leading to irreversible cell membrane disruption, cytoplasmic leakage, and inflammatory activation. As a result, the release profile of biomarkers is intrinsically linked to the molecular characteristics of myocardial cells and the dynamics of cellular death and repair. This biological complexity explains why no single biomarker can completely define the presence or severity of infarction in isolation [3].

High-sensitivity troponin (hs-Tn) assays represent a major advancement in the early detection of myocardial injury; however, their increased sensitivity introduces diagnostic dilemmas regarding specificity. Small troponin elevations detected by hs-Tn assays often occur in chronic conditions such as heart failure, chronic kidney disease, or hypertensive left ventricular hypertrophy, where ongoing low-level myocardial stress or subclinical injury exists without frank infarction. This phenomenon, termed "troponin-positive non-MI," demands that clinicians interpret elevated results within the complete clinical context, using serial testing and clinical correlation to distinguish acute coronary syndromes from chronic myocardial injury. In fact, dynamic changes in troponin concentrations over time — particularly a significant rise and/or fall pattern — have emerged as critical criteria for differentiating acute MI from other causes of troponin release.

Furthermore, the search for non-classical biomarkers that enhance specificity and provide additional pathophysiological insights is an active area of cardiovascular research. Among such markers, copeptin — a surrogate for vasopressin release under stress conditions — demonstrates promise when combined with troponins to accelerate rule-out protocols. Similarly, mid-regional pro-adrenomedullin (MR-proADM) and soluble ST2 (sST2) are under investigation for their roles in identifying myocardial stress, ventricular remodeling, and inflammation, offering supplementary information about the extent of myocardial injury and the risk of adverse outcomes [4].

Another important frontier is the exploration of microRNAs (miRNAs) as highly specific, early, and dynamic biomarkers of myocardial infarction. Circulating miRNAs such as miR-1, miR-133a, and miR-208a have shown substantial promise in preliminary studies due to their cardiac-specific expression profiles and rapid release following myocardial injury. However, challenges remain regarding assay standardization, inter-individual variability, and clinical implementation at scale. Nevertheless, miRNAs may represent a future paradigm shift by enabling ultra-early diagnosis even before structural damage becomes evident on imaging.

Emerging imaging-biomarker hybrid approaches further enhance diagnostic specificity. Cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement can correlate directly with biochemical markers of myocardial necrosis, validating borderline biochemical findings and providing spatial visualization of infarct size and location. Integration of imaging findings with serial biomarker profiles helps differentiate true infarction from conditions like takotsubo cardiomyopathy or myocarditis, which may also exhibit elevated troponins but with distinct structural patterns [5].

CONCLUSION

Biochemical blood analysis has revolutionized the diagnosis of myocardial infarction, with cardiac-specific biomarkers serving as critical tools in modern cardiology. Troponins, despite their occasional elevation in non-infarct conditions, provide the highest specificity for myocardial injury and remain the primary diagnostic markers. Ancillary markers like CK-MB and myoglobin contribute valuable temporal information, particularly in the early and reinfarction phases. The integration of high-sensitivity assays, multi-marker panels, and judicious clinical interpretation continues to advance the precision of myocardial infarction diagnosis. Ongoing research into novel biomarkers and technological innovations promises further refinement in the specificity and speed of biochemical diagnostics for acute coronary syndromes.

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