

Role of Inflammatory Biomarkers in Predicting Acute Coronary Syndromes

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Introduction

Acute Coronary Syndromes (ACS), encompassing conditions such as unstable angina, Non-ST-Segment Elevation Myocardial Infarction (NSTEMI) and ST-Segment Elevation Myocardial Infarction (STEMI), represent critical manifestations of Coronary Artery Disease (CAD) that often result from the rupture or erosion of atherosclerotic plaques followed by thrombus formation. While traditional risk factors such as hypertension, hyperlipidemia, smoking and diabetes play pivotal roles in the pathogenesis of ACS, it has become increasingly evident that inflammation is a central underlying mechanism in both the initiation and progression of atherosclerosis and the acute events that characterize ACS. Inflammatory processes contribute to plaque instability, endothelial dysfunction and thrombogenesis. Consequently, inflammatory biomarkers have emerged as valuable tools for predicting the risk of ACS, aiding in early diagnosis, risk stratification and guiding therapeutic strategies. High-sensitivity C-Reactive Protein (hs-CRP), Inter Leukin-6 (IL-6), Tumor Necrosis Factor-Alpha (TNF- α) and other novel biomarkers reflect different aspects of the inflammatory cascade and have shown promise in forecasting cardiovascular events, sometimes even in the absence of traditional symptoms [1].

Description

The role of inflammatory biomarkers in ACS prediction is grounded in the pathophysiological understanding that atherosclerosis is not merely a lipid storage disease but also a chronic inflammatory condition. As plaques become unstable and prone to rupture, immune cells such as macrophages and T-cells infiltrate the arterial wall, releasing pro-inflammatory cytokines and enzymes that degrade the fibrous cap. This pro-inflammatory microenvironment significantly raises the probability of thrombus formation and subsequent coronary artery occlusion. Among the most extensively studied biomarkers, hs-CRP is a well-validated and easily measurable protein synthesized by the liver in response to IL-6. Elevated levels of hs-CRP have consistently been associated with increased risk of future cardiovascular events in apparently healthy individuals and those with existing cardiovascular disease. For instance, the JUPITER trial demonstrated that individuals with elevated hs-CRP but normal LDL cholesterol levels benefited significantly from statin therapy, suggesting that inflammation independently contributes to cardiovascular risk. Interleukin-6, a cytokine upstream of CRP production, is another critical biomarker that reflects ongoing inflammatory activity within atherosclerotic plaques. IL-6 levels rise rapidly during acute events and have been correlated with both short-term and long-term cardiovascular mortality. Unlike CRP, which is a more general marker of systemic inflammation, IL-6 provides a closer insight into cytokine-mediated signaling cascades directly involved in vascular inflammation. Moreover, TNF- α , a pro-inflammatory cytokine secreted by activated macrophages, contributes to endothelial dysfunction, upregulation of adhesion molecules and apoptosis within atherosclerotic plaques. Elevated TNF- α levels have been observed in patients with unstable angina and

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myocardial infarction and they often correlate with poor prognosis and adverse cardiac remodeling post-ACS.

Another key biomarker, Myelo Per Oxidase (MPO), is released from activated neutrophils and monocytes and plays a role in oxidative stress and LDL modification. MPO levels rise in patients prior to the onset of myocardial infarction and high circulating levels have been shown to predict risk independent of troponins and CRP. Similarly, soluble CD40 Ligand (sCD40L), a marker of platelet activation and immune signaling, has also been implicated in plaque destabilization and thrombus formation, making it a candidate for risk stratification in ACS patients. In addition, Pregnancy-Associated Plasma Protein A (PAPP-A) and Lipoprotein-Associated Phospholipase A2 (Lp-PLA2) have emerged as novel biomarkers involved in plaque inflammation and rupture, offering further granularity in assessing vascular inflammation. From a clinical standpoint, the integration of these biomarkers into predictive models enhances diagnostic accuracy, especially in patients presenting with atypical symptoms or non-diagnostic electrocardiograms. For example, combining hs-CRP with troponin assays can improve the identification of patients at higher risk for myocardial infarction or recurrent events. Moreover, serial measurements of inflammatory biomarkers can track disease progression and therapeutic response. Anti-inflammatory therapies, such as statins and colchicine, have shown efficacy in reducing both biomarker levels and cardiovascular event rates, underscoring the relevance of inflammation in ACS pathophysiology and management. The CANTOS trial further reinforced this paradigm by showing that targeting interleukin-1 β with canakinumab reduced cardiovascular events in patients with prior myocardial infarction and elevated CRP, without affecting lipid levels [2].

Conclusion

In summary, inflammatory biomarkers serve as important adjuncts in predicting the onset and progression of acute coronary syndromes by reflecting underlying processes of plaque inflammation, rupture and thrombosis. High-sensitivity CRP, IL-6, TNF- α , MPO and several emerging markers offer insights into cardiovascular risk that transcend traditional lipid-based metrics. Their incorporation into clinical practice enhances risk stratification, early diagnosis and therapeutic monitoring, contributing to a more precise and individualized approach to cardiovascular care.

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