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Research Progress on Inflammation-Related Biomarkers for Prognosis after Emergency PCI in STEMI Patients

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Abstract

Although emergency percutaneous coronary intervention (PCI) can rapidly restore myocardial perfusion in patients with acute ST-segment elevation myocardial infarction (STEMI), these patients still face a relatively high risk of major adverse cardiovascular events (MACE) after the procedure. The inflammatory response plays a central role in myocardial injury, ventricular remodeling, and thrombosis following STEMI. This article reviews the predictive value of inflammation-related biomarkers—including C-reactive protein, fibrinogen, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation index, systemic inflammatory response index, and red cell distribution width—for prognosis after emergency PCI in STEMI patients, aiming to provide a reference for clinical risk assessment and stratification.

Keywords

Acute ST-Segment Elevation Myocardial Infarction (STEMI), Percutaneous Coronary Intervention (PCI), Inflammatory Biomarkers, Disease Prognosis, Major Adverse Cardiovascular Events (MACE)

1. Introduction

In the past decade, coronary heart disease has remained one of the two leading causes of death in many regions worldwide, including developed countries such as the United States and Europe [1], posing a serious threat to human health. In China, the incidence of coronary heart disease continued to rise as of 2023, largely due to the accelerating aging of the population [2]. Acute ST-segment elevation

myocardial infarction (STEMI) is a severe clinical manifestation of coronary heart disease. Its pathogenesis involves plaque rupture with subsequent thrombosis, leading to coronary artery occlusion, regional myocardial ischemia and necrosis, and a high early mortality rate [3]. Emergency percutaneous coronary intervention (PCI) is an effective method for rapidly restoring blood flow in the culprit vessel and improving hemodynamics, thereby reducing the risk of death in the acute phase.

Although PCI can effectively achieve myocardial reperfusion in ischemic heart, numerous related factors still influence the prognosis of STEMI patients. It should be noted that different clinical studies adopt varying definitions of major adverse cardiovascular events (MACE), with different combinations of endpoints. To help readers accurately understand the findings and avoid inappropriate comparisons, this review will specify the exact MACE components used in each study in parentheses when reporting key results. Clinical studies have shown that the probability of MACE within one year in specific high-risk groups of AMI patients after emergency PCI can reach 5.1%, mainly including all-cause death, cardiac death, recurrent myocardial infarction, unplanned revascularization, heart failure, and stroke [4]. According to the results of a large-sample prospective cohort study with a median follow-up of 4 years on the prognosis of STEMI patients treated with emergency PCI, the incidence of MACE was 17.8% [5] (defined here as: all-cause death, recurrent myocardial infarction, and stroke). Based on this finding, patients still face a relatively high risk of subsequent MACE.

In addition to traditional risk factors, such as advanced age, diabetes, and chronic renal insufficiency [6]-[8], an increasing number of researchers have recognized the critical role of inflammatory responses in myocardial injury, ventricular remodeling, and thrombosis following STEMI. Several inflammation-related biomarkers have been shown to be independently associated with patient prognosis. Compared with imaging modalities, such as echocardiography and coronary angiography, these inflammatory markers offer advantages including ease of detection, low cost, and the ability for dynamic monitoring. This article reviews the research progress on C-reactive protein, fibrinogen, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation index, systemic inflammatory response index, and red cell distribution width, with the aim of providing a reference for clinical practice.

2. Overview of the Pathophysiological Mechanisms of the Inflammatory Response in the Occurrence, Development, and Prognosis of STEMI

Atherosclerosis is the pathological basis of coronary heart disease, and its development is closely associated with abnormal lipoprotein deposition and chronic inflammation. Low-density lipoprotein (LDL) undergoes oxidative modification to form oxidized LDL, which is phagocytosed by macrophages to create foam cells, thereby promoting plaque progression [9]-[11]. Foam cells release cytokines that

attract smooth muscle cells to migrate and synthesize a fibrous cap. Meanwhile, activated macrophages secrete matrix metalloproteinases (MMPs) that degrade collagen, thinning the fibrous cap and rendering the plaque vulnerable. Multiple inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α), participate in all stages of atherosclerosis and influence plaque stability [12]. The inflammatory response plays a critical role in plaque destabilization by weakening the fibrous cap and increasing plaque vulnerability, serving as a bridge between atherosclerosis and acute thrombotic events. When plaque rupture or erosion occurs, exposed tissue factor activates the coagulation cascade, leading to rapid platelet aggregation. Thrombin then catalyzes the conversion of fibrinogen to fibrin, forming platelet-rich thrombi that cause coronary artery thrombosis and trigger STEMI.

Although atherosclerotic plaque rupture with secondary thrombosis is the direct cause of STEMI, the local and systemic inflammatory cascade triggered by myocardial ischemia and necrosis after STEMI is a key factor influencing long-term patient prognosis. Damaged myocardium releases damage-associated molecular patterns (DAMPs), which activate Toll-like receptors and the NLRP3 inflammasome, initiating innate immune responses. Cardiac fibroblasts in the infarct zone are induced to produce large amounts of pro-inflammatory cytokines (such as IL-6 and TNF-α) and chemokines, resulting in massive infiltration of inflammatory cells. Neutrophils and macrophages recruited to the infarct area generate reactive oxygen species and proteases, exacerbating tissue damage. While this process participates in the clearance of necrotic tissue and subsequent repair, excessive or prolonged inflammation can aggravate cardiomyocyte injury, promote interstitial fibrosis, and lead to left ventricular remodeling and impaired contractile function, ultimately increasing the risk of heart failure, malignant arrhythmias, and death [13]-[15].

Therefore, biomarkers reflecting the inflammatory state after acute myocardial infarction have become important tools for predicting long-term patient prognosis.

3. Inflammatory Biomarkers

3.1. Traditional Biomarkers

3.1.1. C-Reactive Protein (CRP) and High-Sensitivity C-Reactive Protein (hs-CRP)

C-reactive protein (CRP) is a traditional inflammatory marker synthesized by the liver upon stimulation by inflammatory cytokines such as interleukin-6 (IL-6). After the onset of STEMI, necrotic cardiomyocytes release large amounts of damage-associated molecular patterns, activating resident macrophages and dendritic cells in the heart to produce pro-inflammatory cytokines including IL-6 and IL-1β. IL-6 circulates to the liver, binds to the gp130 receptor on hepatocytes, and upregulates CRP gene transcription through the JAK/STAT3 signaling pathway. CRP is rapidly synthesized and released, with serum levels beginning to rise within 6 - 12 hours and peaking at 24 - 48 hours. CRP itself also exerts pro-inflammatory

effects by binding to phosphorylcholine on damaged cell membranes, activating the classical complement pathway, and thereby exacerbating tissue injury. Thus, elevated CRP levels not only reflect the systemic inflammatory burden but also directly contribute to the amplification of myocardial damage. High-sensitivity C-reactive protein (hs-CRP) allows for more precise detection of low-grade inflammation. It has demonstrated predictive value for both short-term in-hospital prognosis and long-term outcomes after discharge in patients with acute myocardial infarction. Lin X *et al.* [16] found that in a study of 590 elderly AMI patients (the study population was not STEMI-specific; the included patients were AMI patients, but relevant evidence was provided), multivariate analysis (including age, preoperative systolic and diastolic blood pressure, history of stroke, LVEF, GRACE score, first white blood cell count and hemoglobin, eGFR, blood glucose, creatine kinase-MB, high-sensitivity troponin, and whether it was STEMI) showed that the risk of in-hospital adverse events (defined as a composite endpoint of all-cause death, malignant arrhythmia, and cardiac mechanical complications) in the third tertile (Q3) of hs-CRP at admission was 2.21 times that of the Q1 group. After adding hs-CRP to the traditional prediction model, the AUC increased from 0.676 to 0.712, and the net reclassification index (NRI) was 0.461. In a study investigating the prognosis of ACS patients undergoing emergency PCI [17] (this study was based on the overall ACS population, providing indirect reference for STEMI prognosis assessment), admission hs-CRP > 2 mg/L was independently associated with a 34% increased risk of 1-year ischemic events. However, hs-CRP has the limitation of poor specificity, so the predictive efficacy of a single biomarker remains limited. In recent years, research trends have gradually shifted toward combined use of multiple indicators to construct more accurate predictive models by integrating information from various aspects. For example, the hs-CRP to albumin ratio (hsCAR), as a novel composite indicator, has shown good predictive value. A retrospective study including 1177 STEMI patients [18] found that elevated hsCAR at admission was an independent predictor of MACE (defined here as: all-cause death, ischemia-driven revascularization, non-fatal myocardial infarction, heart failure hospitalization, and cerebrovascular events) (HR = 1.84, 95% CI: 1.53 - 2.21), with a median follow-up of 461 days. Moreover, when combined with the HbA1c/HDL-C biomarker, it demonstrated greater value than the traditional GRACE score. These results suggest that a combined model integrating metabolic and inflammatory indicators can effectively improve postoperative risk stratification in STEMI patients. In addition to CRP and its derived indicators, fibrinogen, as an acute-phase reactant and a key protein in the coagulation system, also plays an important role in inflammation and hypercoagulability after STEMI. In recent years, a growing body of evidence has shown that it has independent predictive value for the long-term prognosis of AMI patients.

3.1.2. Fibrinogen (Fib)

Fibrinogen is a key protein in the coagulation cascade and also serves as an acute-

phase reactant, with its synthesis in the liver increasing under inflammatory stimulation. The intense inflammatory response following STEMI acts on hepatocytes through cytokines such as IL-6 and IL-1β, upregulating the gene expression of the three polypeptide chains of fibrinogen (Aα, Bβ, and γ). Elevated fibrinogen levels increase blood viscosity and promote platelet aggregation, thereby creating a prothrombotic state. In addition, its degradation products, such as D-dimer, can feedback to activate inflammasomes, forming a positive "inflammation-coagulation" feedback loop. The combination of this hypercoagulable state and persistent inflammation increases the risk of adverse events, including in-stent thrombosis, recurrent myocardial infarction, and heart failure. A. Tuluhong [19] explored the relationship between the first fibrin level measured after admission and the 1-year prognosis of STEMI patients. The results showed that the AUC of this indicator for predicting MACE (defined here as: all-cause death, unplanned revascularization, recurrent myocardial infarction after 28 days, heart failure hospitalization, stroke, unstable angina, and in-stent restenosis) was 0.629, with a sensitivity of 71.79%. Multivariate Cox regression analysis including traditional prognostic factors such as age, history of diabetes, smoking history, and medication history indicated that elevated fibrin was an independent risk factor. However, the specificity of the single fibrinogen indicator is insufficient. He Rong *et al.* [20] combined it with admission hs-CRP to evaluate the 2-year prognosis of STEMI patients from the multidimensional perspectives of inflammatory response and hypercoagulable state. The results showed that the combined indicator was more predictive of mortality risk. Under conditions of both hypercoagulability (>510 mg/L) and high inflammation (14 mg/L), the 2-year survival rate was 69%, which was better than that of other subgroups. Both factors were independent risk factors, and the conclusions remained stable after adjusting for traditional influencing factors such as age, smoking history, diabetes, and Killip class in the multivariate Cox model. Furthermore, recent studies have explored other combinations involving fibrinogen. Zufei W *et al.* [21] reported that both admission blood glucose and fibrinogen levels were predictive of slow flow during PCI, and their combination showed superior predictive performance compared with either marker alone. Since slow flow during PCI often indicates inadequate myocardial perfusion and is associated with worse outcomes, survival curve analysis in their study further supported these findings.

Although both C-reactive protein and fibrinogen are traditional inflammatory markers with established prognostic value in STEMI, their individual limitations have prompted researchers to explore the combined use of these and other biomarkers for more comprehensive risk assessment from multiple pathophysiological dimensions. In addition to conventional inflammatory markers such as CRP and fibrinogen, composite inflammatory indices derived from routine blood tests—including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and systemic inflammatory response index (SIRI)—have gained attention in recent years due to

their accessibility, repeatability, and potential value in prognostic evaluation of STEMI patients.

3.2. New Composite Indices

3.2.1. Neutrophil-to-Lymphocyte Ratio (NLR)

The neutrophil-to-lymphocyte ratio (NLR) is a composite inflammatory parameter derived from routine blood count indices, reflecting the balance between systemic inflammation and immune regulation. Compared with absolute white blood cell count, NLR is less affected by transient physiological changes such as exercise or dehydration and integrates two distinct yet complementary immune pathways. NLR has also demonstrated important prognostic value in STEMI patients after emergency PCI. A study by Chinese scholars [22] in 2015, which included 326 STEMI patients, showed that NLR calculated from admission routine blood test results revealed significantly higher incidences of heart failure, arrhythmias, and cardiogenic shock during hospitalization, significantly lower LVEF, and a significantly higher rate of heart failure readmission during 6 months of follow-up in the high NLR group (>6.75) compared with the low NLR group ($P < 0.05$). Another study by Song G *et al.* [23] in 2025 indicated that in the prognostic study of ACS patients (including STEMI) treated with PCI, with a median follow-up of 1014 days, the cumulative survival rate of the low NLR group at admission was higher than that of the high NLR group, and elevated NLR was an independent risk factor for MACE (the findings should be interpreted cautiously in STEMI-specific cohorts; MACE was defined here as: cardiac death, severe heart failure hospitalization, recurrent myocardial infarction, and in-stent restenosis).

3.2.2. Platelet-to-Lymphocyte Ratio (PLR)

The platelet-to-lymphocyte ratio (PLR), first proposed by Smith RA [24], is considered a composite indicator of systemic inflammation. It reflects both the acute inflammatory and prothrombotic state through platelet counts and the immune regulatory status through lymphocyte counts. This parameter has since been widely applied in prognostic studies and has shown meaningful clinical value. Wang Yingshuang [25] found in a study of short-term prognosis within 6 months after emergency PCI in STEMI patients that the PLR indicator, obtained from fasting blood tests on the second day after surgery, had an AUC of 0.800 for predicting MACE (defined here as: malignant arrhythmia, cardiogenic shock, heart failure, cardiac arrest, unplanned revascularization, recurrent angina, and all-cause death), with a sensitivity of 77.4% and a specificity of 79.8%. In recent years, Luo Yonggang *et al.* [26] proposed that combining PLR with NT-proBNP could further improve predictive performance. The predictive efficacy of the combined model for MACE (defined here as: revascularization, cardiac death, arrhythmia, and heart failure) was superior to that of either single indicator, with an AUC of 0.898. Another study [27] based on preoperative routine blood test results established a systemic inflammatory marker score using PLR and NLR. The study found that AMI patients with PLR > 155.93 had significantly higher postoperative all-cause mor-

tality, with an HR of 42.427 in univariate Cox analysis (this study included AMI patients as a whole and did not separately analyze the STEMI subgroup). In addition, Li T *et al.* [28] found that PLR at admission was an independent risk factor for new-onset atrial fibrillation during hospitalization in STEMI patients, with an incidence of approximately 8.1%. Restricted cubic spline (RCS) regression showed a linear dose-response relationship between PLR and NOAF (new-onset atrial fibrillation).

3.2.3. Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI)

The systemic immune-inflammation index (SII) and the systemic inflammatory response index (SIRI) are novel composite inflammatory markers proposed in recent years. SII is calculated as platelet count × neutrophil count / lymphocyte count, integrating information from platelets, neutrophils, and lymphocytes. SIRI is calculated as neutrophil count × monocyte count / lymphocyte count, with greater emphasis on reflecting monocyte activation status. Both indices can be derived solely from routine blood test parameters.

SII has certain value in predicting short-term prognosis after PCI in STEMI patients. Guo Zhichao *et al.* [29], in a retrospective study of 172 STEMI patients undergoing emergency PCI, found that SII measured within two days postoperatively was an independent risk factor for in-hospital MACE (defined here as: cardiogenic shock, acute heart failure, recurrent myocardial infarction, unplanned revascularization, malignant arrhythmia, and non-cardiac death), with an AUC of 0.777, indicating that higher SII was associated with worse in-hospital outcomes. In another multicenter, large-sample prospective study [30], it was found that pre-procedural SII ≥ 914 was independently associated with a higher risk of MACE (defined here as: stroke, heart failure, non-fatal recurrent myocardial infarction, and all-cause death) after PCI in STEMI patients, and the high SII group had a larger myocardial infarct size.

Additional studies have also explored the predictive value of SII and SIRI for 1-year prognosis. Yan H *et al.* [31], in a retrospective study of 1222 STEMI patients undergoing emergency PCI, concluded that SII and SIRI calculated from admission routine blood tests were both independent risk factors for 1-year all-cause death (SII: HR = 8.994; SIRI: HR = 3.671) and MACE (SII: HR = 6.465; SIRI: HR = 4.739, defined here as: recurrent myocardial infarction, non-fatal stroke, and cardiac death). In summary, as novel inflammatory biomarkers, SII and SIRI have certain predictive value for both short-term and long-term prognosis in STEMI patients after PCI.

Although these novel composite inflammatory indices based on routine blood parameters show promise in prognostic prediction for STEMI patients, multicenter and large-sample studies remain relatively limited. Moreover, unlike traditional markers, these indices are largely derived from statistical analysis and are relatively abstract, lacking clear pathophysiological significance. In contrast, red cell distribution width (RDW), a conventional blood routine parameter, has also

been found to be associated with prognosis in STEMI patients.

3.3. Red Cell Distribution Width (RDW)

Red cell distribution width (RDW) is a routine blood parameter traditionally used in the evaluation of anemia-related disorders. Although it is not considered a classic inflammatory marker, numerous studies in recent years have shown that RDW levels are closely associated with systemic inflammatory burden, oxidative stress, and neuroendocrine activation. It can indirectly reflect the inflammatory state following STEMI and has therefore been investigated by many researchers as a non-traditional inflammation-related prognostic indicator. Perlstein *et al.* [32] proposed that inflammatory cytokines can suppress bone marrow hematopoiesis and disrupt iron metabolism, leading to impaired erythrocyte maturation and consequently elevated RDW. Other studies have suggested that intense oxidative stress can shorten red blood cell lifespan, resulting in the premature release of immature erythrocytes into the peripheral circulation and thereby increasing RDW [33] [34]. In addition, excessive neuroendocrine activation may promote abnormal secretion of erythropoietin [35], stimulating erythroid progenitor cell proliferation and affecting red blood cell size uniformity, ultimately elevating RDW levels. Although the precise molecular mechanisms linking RDW to inflammation have not been fully elucidated, existing evidence supports its role as an indirect and integrative inflammation-related biomarker. These mechanisms collectively suggest that RDW may serve as a comprehensive indicator reflecting inflammation, oxidative stress, and nutritional status, thereby correlating with adverse cardiovascular outcomes.

Numerous clinical studies have confirmed that RDW is an independent predictor of prognosis in STEMI patients after emergency PCI. Azab *et al.* [36] followed 619 STEMI patients for 4 years and found that for every 1% increase in RDW at admission, the risk of death increased by 10%. Ting *et al.* [37] conducted a propensity score-matched cohort study based on 84,811 AMI patients and showed that a high RDW (≥13.5%) at admission was independently associated with a 77% increased risk of 1-year all-cause death (HR = 1.77), and this effect remained significant even in non-anemic patients (HR = 1.67; this result was derived from a broad AMI cohort and should be interpreted with caution when extrapolating to STEMI). In China, Lu Hongjing *et al.* [38] performed multivariate Cox regression analysis on 625 elderly STEMI patients (median follow-up of 72 months) and found that RDW at admission was an independent predictor of all-cause death. At a cutoff value of 13.45%, the AUC for predicting all-cause death was 0.630, and survival curves showed that the all-cause mortality rate in the high RDW group was significantly higher than that in the low RDW group.

The research evidence regarding RDW listed above indicates that it has certain predictive value for the prognosis of STEMI patients, and it also performs well in terms of medium- to long-term prognostic value. However, as a single indicator, it is easily influenced by various factors such as anemia, infection, and tumors,

lacks specificity, and its specific underlying mechanism remains unclear. Moreover, there may be superior alternative biomarkers available.

4. Clinical Utility and Evidence Comparison of Inflammatory Markers

The aforementioned biomarkers exhibit significant differences in clinical accessibility, evidence strength, and applicable scenarios. In terms of ease of detection, composite indicators based on routine blood tests (NLR, PLR, SII, SIRI) and RDW require no additional costs or equipment and can be obtained within minutes in the emergency setting, offering optimal timeliness. Hs-CRP and fibrinogen require immunoturbidimetric or coagulation function tests, which add some time but are more standardized. From the perspective of pathophysiological specificity, hs-CRP and fibrinogen are more specific due to their well-defined mechanisms, whereas the biological significance of blood routine-derived indicators is often inferred indirectly and is susceptible to interference from non-cardiac factors such as infection or dehydration. Regarding the temporal dimension of evidence, hs-CRP and NLR have accumulated sufficient data for short-term prognosis within hospitalization and 6 months. Fibrinogen, RDW, SII, and SIRI have demonstrated relatively stable independent predictive ability in long-term follow-up of one year or more. In summary, a single indicator is difficult to balance both sensitivity and specificity. Combining markers that reflect different pathological pathways (e.g., inflammatory markers combined with coagulation markers, or with hemodynamic markers such as NT-proBNP) can enhance risk stratification efficacy without significantly increasing costs. In clinical practice, these biomarkers can be flexibly selected and combined based on patient needs and available resources.

5. Conclusion and Prospects

Inflammatory responses play a critical role in the prognosis of STEMI patients after emergency PCI. Both traditional inflammatory markers such as C-reactive protein and fibrinogen, as well as novel composite inflammatory indices including NLR, PLR, SII, and SIRI, have been shown to be independently associated with the risk of major adverse cardiovascular events (MACE). In addition, RDW, a routine parameter from complete blood count, has recently been recognized as a potential inflammatory marker and also demonstrates independent predictive value for prognosis after PCI in STEMI patients. The combined use of multiple biomarkers can further improve predictive performance and provides clinicians with a simple and cost-effective tool for risk stratification.

There are still some limitations in this field: the cutoff values for inflammatory markers vary considerably across studies, and there is a lack of unified standards; most studies are single-center retrospective designs, with insufficient large-sample prospective validation; most studies have not considered the dynamic evolution of inflammatory markers after discharge, and a single baseline value may not reflect the persistent inflammatory state; the biological significance of some novel

composite markers is not yet fully understood, and many are derived from data-driven combinations, with most markers having poor specificity. In addition, the levels of these inflammatory markers are often influenced by confounding factors, such as infection, anemia, chronic kidney disease, and concurrent use of statins or anti-inflammatory drugs. Therefore, their "independent predictive" value should be interpreted cautiously in comprehensive assessments. Given the good accessibility and reproducibility of these markers, future research should focus on: unifying detection methods and cutoff values, conducting multicenter prospective cohort studies, exploring the value of dynamic monitoring, or investigating combined approaches with metabolic markers, traditional risk factors, imaging data, and clinical scoring systems—leveraging big data and artificial intelligence technologies—to further improve the accuracy of risk prediction after emergency PCI in STEMI patients and achieve individualized treatment.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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