Predictive Value of Left Atrial Volume Index and C-Reactive Protein/Albumin Ratio for New-Onset Arrhythmias in STEMI Patients Undergoing PCI

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Abstract

Background: New-onset arrhythmias remain one of the most frequent and clinically significant complications in patients presenting with ST-segment elevation myocardial infarction (STEMI), even in the contemporary era of primary percutaneous coronary intervention (PCI). Atrial fibrillation, ventricular tachyarrhythmias, and conduction disturbances not only prolong hospitalization but also increase the risk of heart failure, recurrent ischemic events, and mortality. Traditional predictors, such as left ventricular ejection fraction and infarct size, fail to fully capture arrhythmic risk. Increasing attention has been directed toward structural and inflammatory markers as novel tools for risk stratification. Two such parameters are the Left Atrial Volume Index (LAVI), a robust measure of atrial remodeling and diastolic dysfunction, and the C-reactive protein/albumin ratio (CAR), a composite biomarker reflecting systemic inflammation and nutritional status.

Aim: This review explores the predictive value of LAVI and CAR in identifying patients at increased risk for new-onset arrhythmias after PCI-treated STEMI. We aim to (1) examine the mechanistic rationale linking atrial remodeling and inflammation to arrhythmogenesis; (2) summarize clinical evidence on the association of LAVI and CAR with arrhythmic events; and (3) evaluate their incremental utility over established risk models.

Conclusion: Both LAVI and CAR have emerged as promising, easily obtainable, and cost-effective predictors of arrhythmias following STEMI managed with PCI. Elevated LAVI reflects atrial structural and functional abnormalities predisposing to electrical instability, while high CAR indicates a heightened pro-inflammatory milieu that facilitates atrial and ventricular arrhythmias. When assessed together, these markers may offer complementary prognostic information, bridging structural and biochemical pathways of arrhythmogenesis. Incorporating LAVI and CAR into post-PCI risk stratification algorithms may refine patient selection for intensive monitoring, early rhythm surveillance, and preventive therapies. Nevertheless, available data are derived primarily from observational and single-center studies, and prospective validation in larger, multicenter cohorts is needed. Future research should also investigate whether interventions targeting atrial remodeling and systemic inflammation can reduce arrhythmic risk and improve clinical outcomes. By integrating structural and inflammatory parameters, clinicians may achieve a more precise and individualized assessment of arrhythmia risk in STEMI patients undergoing PCI.

Keywords: *Left Atrial Volume Index, C-Reactive Protein/Albumin Ratio, STEMI, PCI*

Introduction

ST-segment elevation myocardial infarction (STEMI) remains one of the most severe clinical manifestations of acute coronary syndrome, associated with high rates of morbidity and mortality despite advances in reperfusion therapy. The introduction of primary percutaneous coronary intervention (PCI) has dramatically improved survival and reduced the incidence of recurrent ischemic events, yet arrhythmias remain a common and clinically significant complication in this population. New-onset arrhythmias—including atrial fibrillation (AF), ventricular tachyarrhythmias, and conduction

disturbances—frequently complicate the acute and subacute phases of STEMI, and their occurrence has been linked to worse hemodynamic stability, prolonged hospital stay, and increased long-term mortality [1–3].

Traditional predictors of arrhythmia risk after STEMI, such as left ventricular ejection fraction (LVEF), infarct size, and Killip class, provide important insights but fail to capture the full spectrum of pathophysiological processes involved in arrhythmogenesis. The complex interplay between myocardial ischemia, atrial remodeling, neurohormonal activation, and systemic inflammation suggests that additional markers may enhance risk prediction. This has led to growing interest in structural and biochemical biomarkers that reflect atrial substrate vulnerability and systemic inflammatory status [4,5].

The Left Atrial Volume Index (LAVI), a widely available echocardiographic parameter, has emerged as a reliable indicator of left atrial remodeling and diastolic dysfunction. Enlarged LAVI not only reflects chronic pressure and volume overload but also provides prognostic information regarding atrial electrical instability and propensity for AF. In parallel, the C-reactive protein/albumin ratio (CAR) has gained attention as a novel inflammatory marker with prognostic significance in cardiovascular disease. CRP, an acute-phase reactant, reflects systemic inflammation and myocardial injury, while hypoalbuminemia indicates poor nutritional and anti-inflammatory reserves; their ratio therefore integrates both harmful inflammatory and protective counter-regulatory processes [6–8].

Despite promising findings, the predictive roles of LAVI and CAR in new-onset arrhythmias following STEMI treated with PCI have not been comprehensively synthesized in the literature. Existing data are fragmented, often limited to single-center observational studies, and have not been fully integrated into current risk stratification algorithms. Therefore, this review aims to critically evaluate the evidence supporting LAVI and CAR as predictors of arrhythmic events in PCI-treated STEMI patients. By bridging structural and inflammatory pathways, we explore their potential role in refining risk prediction and guiding targeted management strategies [9,10].

Pathophysiological Basis of Arrhythmias in STEMI

The development of arrhythmias in the setting of ST-segment elevation myocardial infarction (STEMI) is multifactorial, involving ischemia-related electrical instability, structural remodeling, and systemic factors that modify the electrophysiological substrate. Acute ischemia disrupts myocardial cell membrane potentials, leading to alterations in depolarization and repolarization that increase the likelihood of re-entrant circuits and triggered activity. Ventricular arrhythmias are particularly common in the first 48 hours after infarction due to acute ischemia, catecholamine surge, and electrolyte disturbances, whereas atrial arrhythmias such as atrial fibrillation (AF) often appear later, reflecting atrial remodeling and elevated filling pressures [11,12].

Beyond direct ischemic injury, myocardial inflammation plays a pivotal role in arrhythmogenesis. The infarcted myocardium recruits neutrophils, macrophages, and cytokines that promote oxidative stress and interstitial edema. These processes can impair gap junction conductivity and prolong action potential duration, fostering both atrial and ventricular arrhythmias. Moreover, systemic inflammatory responses can exacerbate atrial stretch, conduction heterogeneity, and autonomic imbalance, further predisposing patients to new-onset arrhythmias [13,14].

Another key factor is the hemodynamic burden imposed by STEMI. Left ventricular systolic dysfunction increases left atrial pressure and volume, resulting in atrial dilatation and wall stress. This structural remodeling alters atrial electrophysiology by slowing conduction and shortening refractory periods, creating a substrate favorable for AF. The combination of atrial stretch and neurohormonal activation—particularly the renin-angiotensin-aldosterone system (RAAS)—accelerates the process of atrial fibrosis, which is strongly associated with arrhythmia initiation and perpetuation [15,16].

Furthermore, autonomic dysregulation contributes significantly to arrhythmogenesis. Heightened sympathetic activity during acute ischemia enhances triggered activity via delayed afterdepolarizations, while parasympathetic withdrawal shortens atrial refractory periods. In the post-PCI setting, reperfusion

injury may also contribute by generating reactive oxygen species, calcium overload, and transient electrophysiological disturbances. Together, these factors highlight the multifaceted and overlapping mechanisms that underlie arrhythmia development in STEMI patients [17,18].

Left Atrial Remodeling and Arrhythmogenesis

Left atrial (LA) remodeling is a central determinant of arrhythmogenesis in patients with cardiovascular disease, particularly after ST-segment elevation myocardial infarction (STEMI). The left atrium is highly sensitive to pressure and volume overload, conditions that commonly accompany left ventricular dysfunction following myocardial infarction. Chronic exposure to elevated filling pressures leads to atrial enlargement, structural fibrosis, and electrophysiological alterations that together create a vulnerable substrate for atrial fibrillation (AF). These changes are most reliably quantified using the Left Atrial Volume Index (LAVI), which normalizes atrial volume to body surface area, thereby providing a reproducible measure of atrial remodeling [19,20].

The pathophysiological link between LA remodeling and arrhythmogenesis is multifaceted. Atrial dilatation results in mechanical stretch of cardiomyocytes, which disrupts gap junction integrity and leads to conduction slowing and heterogeneity. Fibrotic replacement of atrial myocardium further exacerbates conduction block and promotes re-entrant circuits. Moreover, altered calcium handling and stretch-activated ion channel activity contribute to abnormal triggered activity. Collectively, these factors explain why patients with increased LAVI are at heightened risk of new-onset AF, even in the absence of overt structural heart disease [21,22].

Several studies have demonstrated that increased LAVI is a strong predictor of AF following acute coronary syndromes. In STEMI patients undergoing primary PCI, higher baseline LAVI has been independently associated with incident AF and with worse cardiovascular outcomes, including heart failure and stroke. Notably, LAVI has shown incremental predictive value over left ventricular ejection fraction (LVEF), suggesting that atrial remodeling provides unique prognostic information not captured by ventricular function alone. Thresholds above 34 mL/m² are frequently cited as abnormal, but even modest elevations have been associated with arrhythmic risk, emphasizing the sensitivity of LAVI as a marker [23–25].

Beyond AF, LA remodeling may also predispose to ventricular arrhythmias. Experimental data suggest that atrial enlargement and fibrosis can influence autonomic tone and neurohormonal activity, which in turn affect ventricular electrophysiology. Increased atrial pressure and wall stress can elevate sympathetic drive and impair baroreceptor sensitivity, creating a pro-arrhythmic environment that extends beyond the atrium. Thus, LAVI is not only a marker of atrial arrhythmogenesis but may also indirectly reflect systemic vulnerability to malignant ventricular arrhythmias [26,27].

Inflammation and Oxidative Stress in STEMI

Inflammation plays a pivotal role in the pathophysiology of ST-segment elevation myocardial infarction (STEMI) and is increasingly recognized as a critical contributor to arrhythmogenesis. The ischemic myocardium undergoes a robust inflammatory response characterized by leukocyte infiltration, cytokine release, and activation of matrix metalloproteinases. While this response is essential for tissue repair, excessive or dysregulated inflammation leads to adverse remodeling, scar formation, and electrical instability. Elevated systemic inflammatory markers, particularly C-reactive protein (CRP), have consistently been associated with worse outcomes and an increased risk of arrhythmias in STEMI patients [28,29].

Oxidative stress is intimately linked with the inflammatory cascade in myocardial infarction. The abrupt restoration of blood flow during primary percutaneous coronary intervention (PCI) induces reperfusion

injury, characterized by the generation of reactive oxygen species (ROS). Excess ROS disrupt cellular ion homeostasis, impair mitochondrial function, and damage gap junction proteins such as connexin-43, thereby promoting conduction heterogeneity and triggered activity. These electrophysiological disturbances contribute both to atrial fibrillation (AF) and to malignant ventricular arrhythmias such as ventricular tachycardia and ventricular fibrillation [30,31].

The interaction between inflammation and oxidative stress creates a self-perpetuating cycle that amplifies arrhythmic risk. Pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor-alpha, upregulate ROS production, while oxidative stress further stimulates inflammatory signaling pathways such as NF-κB. This cross-talk exacerbates atrial and ventricular remodeling, increases fibrosis, and prolongs action potential duration. Clinically, patients with higher levels of inflammatory and oxidative biomarkers tend to develop arrhythmias more frequently after STEMI, highlighting the importance of targeting these pathways for risk assessment and management [32,33].

Systemic inflammation also contributes to atrial remodeling independent of ischemic injury. Studies have shown that high-sensitivity CRP levels correlate with left atrial volume and fibrosis, suggesting that inflammation may directly drive structural changes that predispose to AF. In addition, albumin, a negative acute-phase reactant with antioxidant properties, decreases during systemic inflammation, further reducing the body's protective capacity against oxidative damage. This interrelationship forms the biological rationale for composite markers such as the CRP/albumin ratio (CAR), which integrate both pro-inflammatory and protective factors into a single index of systemic inflammatory burden [34,35].

CRP/Albumin Ratio as a Prognostic Marker

The C-reactive protein/albumin ratio (CAR) has recently emerged as a powerful prognostic biomarker in cardiovascular disease, integrating two complementary yet opposing biological processes: inflammation and nutritional/anti-inflammatory reserve. CRP, an acute-phase reactant synthesized in the liver under the influence of interleukin-6, reflects systemic inflammatory activity and has been linked with adverse outcomes in myocardial infarction. Conversely, serum albumin functions not only as a nutritional marker but also as an endogenous antioxidant and anti-inflammatory protein. Hypoalbuminemia is frequently observed in acute coronary syndromes (ACS), reflecting both systemic inflammation and poor baseline physiological reserve. By combining CRP and albumin, CAR amplifies prognostic power and accounts for the balance between pro-inflammatory drive and protective buffering capacity [36,37].

Several clinical studies have demonstrated that CAR outperforms either CRP or albumin alone in predicting short- and long-term outcomes in STEMI patients undergoing PCI. Elevated CAR values have been independently associated with in-hospital mortality, major adverse cardiovascular events (MACE), and heart failure development. In addition, CAR has shown a robust correlation with Killip class and troponin levels, suggesting that it reflects both systemic disease severity and myocardial injury burden. Importantly, CAR is inexpensive, universally available, and requires no additional diagnostic testing beyond routine laboratory work, making it an attractive candidate for widespread risk stratification [38,39].

The role of CAR in arrhythmogenesis is supported by evidence linking systemic inflammation with atrial and ventricular electrical instability. High CRP levels promote atrial fibrosis, ion channel dysregulation, and conduction abnormalities, while hypoalbuminemia diminishes the body's antioxidant defenses, facilitating oxidative injury and autonomic imbalance. Clinical observations confirm that patients with elevated CAR are at higher risk of developing new-onset atrial fibrillation (AF) and ventricular tachyarrhythmias after STEMI. In fact, some studies have suggested that CAR may be a stronger predictor of arrhythmias than traditional markers such as white blood cell count or isolated CRP [40,41].

Beyond arrhythmias, CAR has prognostic significance across a spectrum of cardiovascular conditions.

In patients with heart failure, elevated CAR has been associated with worse New York Heart Association (NYHA) class and increased mortality. Similarly, in stable coronary artery disease and peripheral vascular disease, high CAR levels predict cardiovascular death and recurrent ischemic events. These findings highlight the systemic nature of inflammation in cardiovascular pathology and reinforce the clinical utility of CAR as a global marker of disease activity and prognosis [42,43].

Interaction Between LAVI and CRP/Albumin Ratio

While both Left Atrial Volume Index (LAVI) and the C-reactive protein/albumin ratio (CAR) independently predict adverse outcomes in ST-segment elevation myocardial infarction (STEMI), their combined assessment may provide a more comprehensive reflection of arrhythmic risk. LAVI represents the structural and hemodynamic burden imposed on the atrium, while CAR reflects the systemic inflammatory state. Given that arrhythmogenesis is driven by both substrate vulnerability and inflammatory triggers, integrating these markers allows for dual-pathway risk stratification. Patients with both increased LAVI and elevated CAR are therefore at particularly high risk for new-onset arrhythmias after primary percutaneous coronary intervention (PCI) [44,45].

Mechanistically, inflammation and atrial remodeling are tightly interconnected. Inflammatory mediators, including interleukin-6 and tumor necrosis factor-alpha, promote atrial fibrosis, collagen deposition, and gap junction dysfunction, thereby enlarging LAVI over time. Conversely, atrial stretch and pressure overload augment local cytokine production, perpetuating systemic inflammation and raising CRP levels. Low albumin further weakens antioxidant defenses, facilitating oxidative stress and myocardial injury. This bidirectional relationship suggests that CAR and LAVI may act synergistically rather than independently, amplifying arrhythmic risk in STEMI patients [46,47].

Emerging evidence supports the prognostic value of combining structural and inflammatory biomarkers in cardiovascular disease. For example, studies have demonstrated that patients with both elevated CRP and abnormal LAVI are at substantially higher risk of atrial fibrillation compared with those with either abnormality alone. Similarly, high CAR levels have been shown to accentuate the prognostic impact of increased atrial size on arrhythmic outcomes. These findings highlight the additive and potentially multiplicative risk when atrial remodeling coexists with systemic inflammation [48,49].

From a clinical standpoint, simultaneous evaluation of LAVI and CAR could improve post-PCI risk stratification in STEMI. Incorporating both markers into prediction models may allow earlier identification of patients who require intensive rhythm monitoring, tailored pharmacologic strategies, or preventive interventions such as closer telemetry surveillance. This integrated approach aligns with the paradigm of precision medicine, in which combining structural, functional, and biochemical parameters yields a more individualized assessment of arrhythmic risk [50].

Arrhythmias in the Setting of Primary PCI

Primary percutaneous coronary intervention (PCI) has become the standard of care for patients presenting with ST-segment elevation myocardial infarction (STEMI), significantly reducing mortality and limiting myocardial damage. However, despite timely reperfusion, arrhythmias remain a frequent complication in the acute and subacute phases. Ventricular tachycardia (VT) and ventricular fibrillation (VF) are particularly common within the first 48 hours post-PCI, largely driven by acute ischemia, reperfusion injury, and electrolyte imbalances. Although many early ventricular arrhythmias are transient, their occurrence is associated with increased short-term mortality and often reflects larger infarct size or incomplete myocardial salvage [51,52].

Atrial fibrillation (AF) is the most prevalent supraventricular arrhythmia following STEMI, with reported incidence ranging from 6% to 21% in contemporary PCI cohorts. New-onset AF is not a benign finding; it is linked with higher risks of thromboembolic events, recurrent ischemia, and heart failure. Unlike ventricular arrhythmias, AF often arises later during hospitalization or in the subacute period, reflecting the contribution of left atrial remodeling, elevated filling pressures, and systemic inflammation. Notably, AF after STEMI has been shown to confer a worse long-term prognosis, even

in patients who spontaneously revert to sinus rhythm [53,54].
Bradyarrhythmias and conduction disturbances also occur commonly after PCI in STEMI, particularly when the infarction involves the right coronary artery supplying the atrioventricular node. High-degree atrioventricular block and sinus node dysfunction may necessitate temporary pacing in the acute phase. Although some conduction abnormalities resolve after reperfusion, persistent bradyarrhythmias may reflect more extensive conduction system damage and predict adverse outcomes. Thus, arrhythmic complications following PCI are diverse in type and mechanism, and their prognostic implications extend well beyond the acute setting [55,56].
Traditional clinical predictors of arrhythmias after PCI include advanced age, higher Killip class, reduced left ventricular ejection fraction, anterior infarction, and delayed reperfusion. However, these factors fail to account for the full spectrum of arrhythmic risk, particularly in patients with preserved ventricular function. This gap underscores the need for novel markers, such as Left Atrial Volume Index (LAVI) and the C-reactive protein/albumin ratio (CAR), which provide insight into atrial remodeling and systemic inflammation—two central processes in post-PCI arrhythmogenesis [57,58].

Evidence Linking LAVI to Post-PCI Arrhythmias

Clinical studies have consistently highlighted the predictive role of Left Atrial Volume Index (LAVI) in arrhythmia development after ST-segment elevation myocardial infarction (STEMI). Increased LAVI reflects chronic exposure to elevated left ventricular filling pressures, which predisposes patients to atrial dilatation, fibrosis, and electrical remodeling. In the context of STEMI, this pre-existing atrial vulnerability combines with acute ischemic stress to trigger arrhythmias, particularly atrial fibrillation (AF). Several prospective cohorts have demonstrated that patients with elevated LAVI values are more likely to develop new-onset AF during hospitalization for STEMI, even after successful primary percutaneous coronary intervention (PCI) [59,60].
In a study by Kim et al., patients with STEMI undergoing PCI who developed new-onset AF had significantly higher baseline LAVI compared with those who maintained sinus rhythm. Importantly, LAVI remained an independent predictor of AF after adjusting for confounders such as age, left ventricular ejection fraction (LVEF), and infarct location. The authors identified a cutoff value of 34 mL/m^2 as predictive of AF occurrence, consistent with guideline-based thresholds for abnormal atrial size. This suggests that echocardiographic evaluation of LAVI on admission provides valuable prognostic information beyond traditional markers [61].
Subsequent studies have extended these findings to long-term outcomes. Shin et al. reported that higher LAVI at the time of PCI was associated not only with incident AF during hospitalization but also with recurrent arrhythmias and heart failure readmissions during follow-up. The prognostic impact of LAVI persisted regardless of preserved or reduced LVEF, underscoring its role as a marker of atrial vulnerability rather than ventricular dysfunction alone. This reinforces the concept that atrial remodeling is an independent contributor to adverse outcomes in STEMI patients [62].
Beyond AF, there is evidence suggesting that increased LAVI may correlate with ventricular arrhythmias. While the mechanism is less direct, it is hypothesized that atrial enlargement contributes to autonomic imbalance and heightened sympathetic activity, which may predispose to ventricular tachyarrhythmias. A study by Aksu et al. observed that patients with elevated LAVI and larger atrial dimensions had higher rates of both AF and ventricular arrhythmias during STEMI hospitalization. These findings indicate that LAVI, while primarily a marker of atrial remodeling, may also serve as a broader indicator of electrophysiological instability in STEMI [63].

Evidence Linking CRP/Albumin Ratio to Post-PCI Arrhythmias

The prognostic utility of the C-reactive protein/albumin ratio (CAR) in predicting arrhythmias after primary percutaneous coronary intervention (PCI) for ST-segment elevation myocardial infarction (STEMI) has been highlighted in multiple clinical studies. Elevated CAR reflects the coexistence of

heightened inflammation and reduced antioxidant/nutritional reserve, both of which are implicated in arrhythmic risk. In a study by Kocyigit et al., a high CAR on admission was independently associated with increased incidence of in-hospital atrial fibrillation (AF) in STEMI patients. Importantly, CAR retained predictive value after adjusting for age, left ventricular ejection fraction (LVEF), infarct size, and renal function, suggesting its role as a robust biomarker that integrates systemic stress with cardiovascular vulnerability [64].

Other observational cohorts have validated these findings. Oh et al. demonstrated that CAR not only predicted AF but also correlated with ventricular arrhythmias during the acute phase of myocardial infarction. Their analysis showed that patients in the highest tertile of CAR had a significantly higher risk of sustained ventricular tachycardia and ventricular fibrillation compared with those in the lowest tertile. Notably, CAR provided incremental predictive value when added to conventional risk scores, such as the GRACE score, underscoring its potential integration into existing prognostic models for post-PCI arrhythmias [65].

In addition to acute arrhythmias, CAR has been linked with adverse long-term outcomes, including recurrent AF, major adverse cardiovascular events (MACE), and mortality in post-MI populations. Ucar et al. observed that STEMI patients with elevated CAR were more likely to experience recurrent arrhythmias and heart failure hospitalizations during follow-up. This finding highlights the dual role of CAR as both an acute-phase predictor and a marker of chronic cardiovascular instability. Furthermore, the continuous rather than threshold-dependent nature of CAR's prognostic significance suggests that even modest elevations may have clinical relevance [66].

Mechanistically, CAR may be seen as a dynamic biomarker reflecting both trigger and substrate conditions for arrhythmogenesis. Elevated CRP levels promote atrial fibrosis, conduction slowing, and ion channel dysregulation, while reduced albumin weakens antioxidant defense and increases oxidative stress burden. Together, this imbalance facilitates both atrial and ventricular electrical instability. By integrating these opposing biological processes into a single index, CAR captures the full spectrum of inflammatory stress relevant to arrhythmia development, thereby offering a more comprehensive risk assessment than either marker alone [67].

Combined Role of LAVI and CRP/Albumin Ratio

The integration of structural and inflammatory biomarkers has emerged as a promising approach to refining arrhythmia risk prediction in ST-segment elevation myocardial infarction (STEMI) patients treated with percutaneous coronary intervention (PCI). Left Atrial Volume Index (LAVI) and the C-reactive protein/albumin ratio (CAR) reflect complementary yet distinct aspects of arrhythmogenesis: atrial remodeling as a substrate and systemic inflammation as a trigger. Evaluating these markers together offers a dual-lens view of arrhythmic vulnerability, where elevated LAVI identifies patients with structurally compromised atria, while high CAR signals an environment primed for electrical instability [68,69].

Evidence suggests that patients with both abnormal LAVI and elevated CAR are at substantially higher risk for new-onset atrial fibrillation (AF) compared with those with only one abnormality. In a prospective study by Kurtul et al., combining CAR with echocardiographic measures of atrial size significantly improved risk stratification for AF in acute coronary syndrome (ACS) patients. Patients with elevated values in both markers had nearly a two-fold higher incidence of AF than those with isolated abnormalities, supporting their additive predictive utility. This synergistic effect likely arises because atrial stretch enhances cytokine release and inflammatory signaling, while systemic inflammation accelerates atrial fibrosis and conduction heterogeneity, creating a feedback loop for arrhythmogenesis [70].

The combined use of LAVI and CAR has also been investigated in relation to ventricular arrhythmias. Aksu et al. reported that STEMI patients with high CAR and enlarged atria experienced greater rates of both AF and sustained ventricular tachycardia during hospitalization. These findings suggest that

systemic inflammation not only amplifies the arrhythmic substrate within the atria but also modulates autonomic activity and oxidative stress pathways that predispose to ventricular arrhythmias. Thus, integrating LAVI and CAR may help identify patients at risk for a broader spectrum of arrhythmic events, extending beyond atrial fibrillation alone [71].

From a clinical standpoint, combined assessment of LAVI and CAR could enhance decision-making in post-PCI care. Patients with concurrent elevation in both markers may benefit from closer rhythm monitoring, prolonged telemetry, or consideration for early anticoagulation if AF develops. Additionally, these patients may represent candidates for more aggressive anti-inflammatory or neurohormonal therapies aimed at modifying arrhythmic risk. By embedding both structural and biochemical parameters into predictive models, clinicians could move toward a precision-medicine strategy that more accurately identifies high-risk individuals and tailors management accordingly [72].

Conclusion

New-onset arrhythmias remain one of the most clinically significant complications following ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. Traditional predictors such as left ventricular ejection fraction, infarct size, and clinical risk scores, while valuable, do not fully capture the complex interplay of structural and systemic factors that contribute to electrical instability.

The Left Atrial Volume Index (LAVI) provides a robust measure of atrial remodeling and diastolic burden, directly reflecting a vulnerable substrate for arrhythmogenesis. In parallel, the C-reactive protein/albumin ratio (CAR) integrates inflammatory activity and nutritional/antioxidant reserve, identifying a systemic environment conducive to arrhythmic triggers. Individually, both parameters offer important prognostic information, but when combined, they provide a dual-pathway assessment that more comprehensively reflects arrhythmic vulnerability.

The integration of LAVI and CAR into post-PCI risk stratification frameworks has the potential to transform clinical practice. Their use could enable earlier identification of high-risk patients, guide monitoring intensity, and inform preventive strategies such as targeted rhythm surveillance, anticoagulation for atrial fibrillation, or therapies aimed at reducing inflammation and remodeling. Importantly, these markers are accessible, cost-effective, and easily incorporated into routine clinical workflows.

Future research should aim to validate their predictive power in larger, multicenter cohorts and evaluate whether interventions that modify atrial remodeling or systemic inflammation can mitigate arrhythmic risk. Such efforts may pave the way toward precision-medicine approaches, where structural and biochemical markers are integrated into personalized management strategies.

In summary, the combined use of LAVI and CAR represents a promising step forward in identifying and managing arrhythmia risk in STEMI patients undergoing PCI. By bridging the gap between structural vulnerability and systemic inflammatory activation, these parameters provide clinicians with a practical and powerful tool to refine risk prediction and improve outcomes.

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