Relative Hyperglycemia and Coronary Thrombosis in Diabetic STEMI: Current Evidence on the Prognostic Utility of Stress Hyperglycemia Ratio

Alaa El Sayed Salama, Mahmoud Hassan Abdelqader Shah, Ibrahim Mohamed Fathy Mohamed, Nada Hassan Metwally

Cardiology Department, Faculty of Medicine - Zagazig University
Corresponding Author: Ibrahim Mohamed Fathy Mohamed

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Abstract

Background: Diabetic patients presenting with ST-segment elevation myocardial infarction (STEMI) represent a particularly high-risk population characterized by more extensive coronary artery disease, enhanced platelet reactivity, impaired fibrinolysis, and an increased propensity for intracoronary thrombus formation. Large thrombus burden during primary percutaneous coronary intervention (PCI) is associated with distal embolization, no-reflow phenomenon, microvascular obstruction, stent thrombosis, and adverse cardiovascular outcomes. Although admission hyperglycemia has long been recognized as a marker of poor prognosis in acute myocardial infarction, it cannot reliably distinguish acute stress-induced glycemic responses from chronic hyperglycemia, particularly in patients with pre-existing diabetes mellitus. The stress hyperglycemia ratio (SHR), which integrates admission glucose with glycated hemoglobin-derived estimated average glucose, has emerged as a novel biomarker reflecting relative hyperglycemia and the magnitude of the acute metabolic response to myocardial injury.

Aim: This review aims to comprehensively summarize current evidence regarding the association between relative hyperglycemia, assessed by SHR, and coronary thrombosis in diabetic patients with STEMI undergoing primary PCI. Particular emphasis is placed on the pathophysiological mechanisms linking elevated SHR with thrombogenesis, the clinical studies evaluating the relationship between SHR and intracoronary thrombus burden, and the prognostic implications of SHR in contemporary STEMI management.

Discussion: Available evidence suggests that elevated SHR is associated with increased thrombus burden, greater angiographic complexity, impaired myocardial reperfusion, and a higher incidence of adverse clinical outcomes. Potential mechanisms underlying this association include endothelial dysfunction, oxidative stress, inflammatory activation, enhanced platelet aggregation, activation of coagulation pathways, and suppression of endogenous fibrinolysis. Several observational studies have demonstrated that SHR independently predicts large intracoronary thrombus burden in patients undergoing primary PCI, outperforming admission glucose alone in risk stratification. Furthermore, elevated SHR has been linked to increased mortality, major adverse cardiovascular events, no-reflow phenomenon, microvascular obstruction, and impaired ventricular recovery following STEMI.

Conclusion: Stress hyperglycemia ratio represents a promising and readily obtainable biomarker that provides a more individualized assessment of acute metabolic stress in diabetic STEMI patients. Current evidence supports its potential role in identifying individuals at increased thrombotic risk and poor prognosis during primary PCI. Incorporation of SHR into existing risk assessment models may enhance early stratification and facilitate tailored therapeutic strategies. Nevertheless, prospective multicenter studies are warranted to establish standardized cutoff values, validate its incremental prognostic value, and determine whether SHR-guided interventions can improve clinical outcomes in this vulnerable patient population.

Keywords: *Hyperglycemia ,Coronary Thrombosis, Diabetic, STEMI, Stress Hyperglycemia Ratio*

Introduction

ST-segment elevation myocardial infarction (STEMI) remains one of the most severe manifestations of acute coronary syndrome and continues to be a major contributor to cardiovascular morbidity and mortality worldwide. Despite substantial advances in pharmacological therapy, systems of care, and widespread implementation of primary percutaneous coronary intervention (PCI), STEMI continues to carry significant risks of adverse short- and long-term outcomes. Although timely reperfusion remains the cornerstone of management, patient-related factors substantially influence prognosis following myocardial infarction. Among these factors, diabetes mellitus (DM) has consistently been identified as a major determinant of unfavorable clinical outcomes after STEMI [1].

Diabetes mellitus is highly prevalent among patients presenting with STEMI and is associated with a unique cardiovascular phenotype characterized by advanced age, a greater burden of cardiovascular risk factors, and more frequent comorbid conditions. Numerous studies have demonstrated that diabetic patients experience higher rates of in-hospital complications, recurrent ischemic events, heart failure, and mortality despite contemporary reperfusion strategies. These findings have led to the recognition of diabetic STEMI as a distinct high-risk clinical entity requiring enhanced risk stratification and individualized therapeutic approaches [2].

The adverse prognosis associated with diabetic STEMI is largely attributable to the complex pathophysiological consequences of chronic hyperglycemia and insulin resistance. Diabetes promotes endothelial dysfunction, oxidative stress, chronic inflammation, and accelerated atherosclerosis, resulting in diffuse coronary artery disease and increased plaque vulnerability. Furthermore, diabetes alters platelet function and coagulation pathways, creating a prothrombotic state that predisposes affected individuals to more extensive intracoronary thrombosis during acute coronary occlusion [3].

Intracoronary thrombus formation represents the principal pathological mechanism underlying STEMI. Following plaque rupture or erosion, exposure of thrombogenic material initiates platelet activation and aggregation together with activation of the coagulation cascade, ultimately leading to thrombus formation and vessel occlusion. Importantly, the extent of thrombus burden has important clinical implications, as large intracoronary thrombi are associated with distal embolization, no-reflow phenomenon, impaired myocardial reperfusion, stent thrombosis, and increased mortality following primary PCI [4].

Hyperglycemia during acute myocardial infarction has long been recognized as an indicator of adverse prognosis. Acute elevations in blood glucose levels occur secondary to activation of neurohormonal and inflammatory pathways during physiological stress, resulting in increased hepatic glucose production and peripheral insulin resistance. Several studies have shown that admission hyperglycemia is associated with larger infarct size, impaired coronary reperfusion, and increased mortality among patients with acute coronary syndromes [5].

However, interpretation of admission glucose values in diabetic patients remains problematic because absolute glucose concentrations cannot reliably distinguish acute stress-induced hyperglycemia from chronically elevated glucose levels resulting from poor glycemic control. Consequently, the prognostic significance of admission hyperglycemia may be underestimated or misinterpreted, particularly in patients with established diabetes mellitus. This limitation has stimulated growing interest in biomarkers that better reflect the relative degree of metabolic stress experienced during acute illness [6].

The stress hyperglycemia ratio (SHR) has emerged as a novel indicator of relative hyperglycemia by integrating admission glucose concentrations with estimated average glucose derived from glycated hemoglobin (HbA1c). Unlike isolated measurements of admission glucose, SHR provides a more individualized assessment of acute glycemic disturbances by accounting for baseline glycemic status. Recent investigations have suggested that SHR may offer superior prognostic utility across a range of cardiovascular conditions, including acute myocardial infarction [7].

Accumulating evidence indicates that elevated SHR is associated with adverse outcomes in patients presenting with STEMI. Higher SHR values have been linked to increased risks of mortality, major

adverse cardiovascular events, microvascular obstruction, impaired ventricular recovery, and no-reflow phenomenon. More recently, emerging studies have demonstrated a significant association between elevated SHR and increased intracoronary thrombus burden, suggesting that relative hyperglycemia may serve as a marker of enhanced thrombotic activity during acute coronary occlusion [8].

The biological plausibility of this association is supported by evidence demonstrating that acute hyperglycemia promotes oxidative stress, endothelial dysfunction, platelet hyperreactivity, inflammatory activation, and impaired fibrinolysis. In diabetic patients, these acute metabolic disturbances occur on a background of chronic vascular injury and pre-existing prothrombotic abnormalities, potentially amplifying the extent of coronary thrombosis during STEMI and contributing to poorer outcomes following primary PCI [9].

Although the prognostic significance of SHR has been increasingly recognized, evidence specifically addressing its relationship with intracoronary thrombus burden in diabetic patients with STEMI remains relatively limited. Furthermore, uncertainties persist regarding the clinical applicability of SHR, its incremental value beyond established prognostic markers, and its potential role in guiding therapeutic decision-making during primary PCI.

Therefore, the aim of this review is to comprehensively evaluate current evidence regarding the relationship between relative hyperglycemia, assessed using the stress hyperglycemia ratio, and coronary thrombosis in diabetic patients presenting with STEMI undergoing primary PCI. Particular emphasis will be placed on the pathophysiological mechanisms linking SHR with thrombogenesis, the available clinical studies investigating its association with intracoronary thrombus burden and adverse outcomes, and the potential implications of incorporating SHR into contemporary risk stratification models for this high-risk population [10].

2. Diabetes Mellitus and STEMI: A High-Risk Clinical Phenotype

Diabetes mellitus (DM) is one of the most important comorbid conditions affecting patients with ST-segment elevation myocardial infarction (STEMI). The coexistence of diabetes substantially influences the clinical presentation, angiographic characteristics, response to reperfusion therapy, and overall prognosis of acute myocardial infarction. Despite significant advances in primary percutaneous coronary intervention (PCI) and contemporary pharmacotherapy, diabetic patients continue to experience disproportionately higher rates of adverse cardiovascular outcomes compared with non-diabetic individuals. These observations have established diabetic STEMI as a distinct high-risk clinical phenotype requiring more comprehensive risk stratification and individualized therapeutic strategies [11].

The prevalence of diabetes among patients presenting with STEMI has increased steadily over recent decades, reflecting the global rise in obesity and metabolic syndrome. Epidemiological studies suggest that approximately 16%–25% of STEMI patients have established diabetes mellitus, while a substantial proportion have previously undiagnosed abnormalities in glucose metabolism. Compared with non-diabetic patients, diabetic individuals presenting with STEMI are generally older and exhibit a higher prevalence of hypertension, dyslipidemia, chronic kidney disease, and previous cardiovascular disease. These characteristics contribute to greater clinical complexity and are associated with increased rates of both in-hospital and long-term mortality [12].

The pathophysiological mechanisms underlying the adverse outcomes observed in diabetic STEMI are multifactorial. Chronic hyperglycemia and insulin resistance promote endothelial dysfunction through increased oxidative stress, reduced nitric oxide bioavailability, and enhanced inflammatory signaling. These abnormalities accelerate the development and progression of atherosclerosis, leading to more extensive coronary artery involvement. In addition, diabetes is associated with qualitative alterations in lipoproteins and persistent low-grade inflammation, further contributing to plaque instability and the propensity for acute coronary events [13].

Coronary angiographic studies have consistently demonstrated that diabetic patients exhibit more severe and diffuse coronary artery disease than their non-diabetic counterparts. Multivessel disease, longer

lesion length, greater plaque burden, and more extensive coronary calcification are commonly encountered in diabetic STEMI patients undergoing primary PCI. These anatomical characteristics increase procedural complexity and may partially explain the lower rates of optimal myocardial reperfusion observed in this population despite successful restoration of epicardial coronary flow [14].

Another important characteristic of diabetic STEMI is the presence of an enhanced prothrombotic state. Diabetes mellitus is associated with increased platelet activation, upregulation of platelet surface receptors, augmented thrombin generation, and impaired endogenous fibrinolysis. Elevated levels of inflammatory mediators and plasminogen activator inhibitor-1 further contribute to thrombus propagation and persistence. Consequently, diabetic patients are more likely to develop larger intracoronary thrombi during STEMI, increasing the risk of distal embolization, microvascular obstruction, and the no-reflow phenomenon during primary PCI [15].

The impact of diabetes on STEMI outcomes extends beyond the acute phase of myocardial infarction. Numerous studies have demonstrated that diabetic patients experience higher rates of cardiogenic shock, acute heart failure, recurrent myocardial infarction, repeat revascularization, and cardiovascular mortality during follow-up. Importantly, these adverse outcomes persist even after adjustment for conventional cardiovascular risk factors and differences in treatment strategies, suggesting that diabetes independently contributes to worse prognosis following STEMI [16].

Advanced imaging modalities have provided additional insights into the mechanisms responsible for the unfavorable prognosis associated with diabetic STEMI. Cardiac magnetic resonance studies have demonstrated that diabetic patients frequently exhibit larger infarct size, more pronounced microvascular obstruction, and greater impairment of ventricular function following acute myocardial infarction. Persistent microvascular dysfunction despite successful primary PCI may explain why diabetic individuals often experience worse clinical outcomes despite comparable angiographic success rates [17].

The recognition of diabetic STEMI as a unique high-risk entity has important implications for clinical practice. Traditional risk assessment tools may not fully capture the complex interplay between chronic metabolic abnormalities and the acute physiological response to myocardial infarction in this population. Consequently, there is growing interest in identifying novel biomarkers capable of improving prognostic assessment among diabetic STEMI patients. The stress hyperglycemia ratio (SHR), which integrates acute and chronic glycemic status, has emerged as a promising marker that may provide additional information regarding thrombotic risk and clinical outcomes beyond that offered by admission glucose measurements alone [18].

Collectively, current evidence indicates that diabetic patients presenting with STEMI represent a particularly vulnerable population characterized by extensive coronary disease, heightened thrombogenicity, impaired myocardial reperfusion, and persistently elevated mortality despite advances in contemporary cardiovascular care. Understanding the unique pathophysiological features of diabetic STEMI provides the foundation for exploring the potential role of relative hyperglycemia and SHR in identifying patients at increased risk of extensive coronary thrombosis and adverse outcomes following primary PCI.

3. Intracoronary Thrombosis in STEMI

Intracoronary thrombosis is the pathological hallmark of ST-segment elevation myocardial infarction (STEMI) and represents the final common pathway linking atherosclerotic plaque disruption to acute coronary artery occlusion. The abrupt interruption of coronary blood flow following thrombus formation results in myocardial ischemia and, if prolonged, irreversible myocardial necrosis. Although primary percutaneous coronary intervention (PCI) has significantly improved outcomes in STEMI, the presence of large intracoronary thrombus burden remains an important determinant of procedural success and clinical prognosis. Consequently, understanding the mechanisms, determinants, and clinical implications of coronary thrombosis is essential for optimizing the management of patients undergoing primary PCI [19].

The development of intracoronary thrombus is most commonly triggered by rupture or erosion of an atherosclerotic plaque. Plaque rupture exposes highly thrombogenic components of the necrotic core, including collagen and tissue factor, to circulating blood elements. This process initiates platelet adhesion through interactions with von Willebrand factor and glycoprotein receptors, followed by platelet activation and aggregation. Simultaneously, activation of the coagulation cascade promotes thrombin generation and fibrin formation, resulting in the development of an organized thrombus capable of partially or completely occluding the culprit vessel [20].

The extent of thrombus formation during STEMI varies considerably among patients and is influenced by multiple clinical and angiographic factors. Delayed presentation after symptom onset allows continued thrombus propagation and is consistently associated with larger thrombus burden. Other determinants include smoking, diabetes mellitus, elevated inflammatory activity, larger plaque burden, and hypercoagulable states. In particular, diabetes contributes to enhanced platelet reactivity, increased thrombin generation, and impaired fibrinolysis, thereby promoting the formation of larger and more resistant intracoronary thrombi [21].

Assessment of thrombus burden during coronary angiography is commonly performed using the Thrombolysis in Myocardial Infarction (TIMI) thrombus grading system. This classification ranges from Grade 0, indicating the absence of angiographic evidence of thrombus, to Grade 5, representing total vessel occlusion due to thrombotic obstruction. In clinical practice and research settings, high thrombus burden is generally defined as TIMI thrombus grades 4 or 5. The TIMI grading system remains the most widely utilized method for evaluating thrombus burden and has demonstrated important associations with procedural and clinical outcomes in STEMI patients undergoing primary PCI [22].

Large intracoronary thrombus burden poses several challenges during primary PCI. Mechanical disruption of the thrombus during wire passage, balloon inflation, or stent deployment may result in distal embolization of thrombotic debris into the coronary microcirculation. This process contributes to microvascular obstruction and the no-reflow phenomenon, characterized by inadequate myocardial perfusion despite successful restoration of epicardial coronary artery patency. No-reflow has been consistently associated with larger infarct size, reduced left ventricular function, increased incidence of heart failure, and higher mortality rates [23].

In addition to its impact on myocardial reperfusion, high thrombus burden has been linked to increased procedural complexity and adverse cardiovascular events. Patients with extensive thrombus frequently require additional interventional maneuvers and may experience lower rates of optimal post-procedural TIMI flow. Furthermore, large thrombus burden has been identified as an independent predictor of recurrent myocardial infarction, early stent thrombosis, major adverse cardiovascular events (MACE), and both short- and long-term mortality following STEMI [24].

Advances in intracoronary imaging techniques have improved understanding of the underlying substrate responsible for coronary thrombosis. Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) allow detailed characterization of plaque morphology and thrombus composition, facilitating differentiation between plaque rupture and plaque erosion. These modalities have demonstrated that STEMI patients with large thrombus burden frequently exhibit vulnerable plaque features, including thin-cap fibroatheroma, large lipid pools, and substantial inflammatory infiltration. Although their routine use during primary PCI remains limited, these technologies provide valuable mechanistic insights into coronary thrombosis [25].

Given the profound influence of thrombus burden on procedural outcomes and prognosis, considerable effort has been directed toward identifying clinical and biochemical markers capable of predicting extensive thrombosis before coronary intervention. Traditional predictors such as delayed presentation, inflammatory biomarkers, and angiographic characteristics provide useful information but often lack sufficient specificity. Consequently, there is growing interest in novel biomarkers reflecting the underlying biological processes involved in thrombogenesis. Relative hyperglycemia, quantified by the stress hyperglycemia ratio (SHR), has emerged as a potential marker of thrombotic risk by integrating

the acute metabolic response to STEMI with baseline glycemic status [26].

The recognition that intracoronary thrombus burden significantly influences both immediate and long-term outcomes has important therapeutic implications. Early identification of patients at increased risk of extensive thrombosis may facilitate more intensive antithrombotic strategies, closer procedural monitoring, and improved risk stratification following primary PCI. In this context, exploring the relationship between relative hyperglycemia and coronary thrombosis may enhance understanding of residual thrombotic risk among diabetic STEMI patients and contribute to the development of more personalized approaches to care.

4. Relative Hyperglycemia and Stress Hyperglycemia Ratio

Hyperglycemia is a common metabolic disturbance observed during acute myocardial infarction and has long been recognized as an indicator of poor clinical prognosis. Acute elevations in blood glucose occur as part of the physiological stress response and are mediated by activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis. Increased circulating concentrations of catecholamines, cortisol, glucagon, and inflammatory cytokines stimulate hepatic gluconeogenesis and glycogenolysis while simultaneously promoting peripheral insulin resistance. The resulting imbalance between glucose production and utilization leads to stress-induced hyperglycemia, even in individuals without a prior diagnosis of diabetes mellitus. Numerous studies have demonstrated associations between stress hyperglycemia and larger infarct size, impaired myocardial reperfusion, heart failure, and increased mortality in patients with acute coronary syndromes [27].

Traditionally, admission blood glucose has been used to assess the prognostic significance of hyperglycemia in patients presenting with STEMI. However, the clinical interpretation of isolated glucose measurements remains problematic, particularly among diabetic individuals. Elevated admission glucose concentrations may reflect either an acute physiological response to severe illness or chronically poor glycemic control resulting from longstanding diabetes. Consequently, absolute glucose values fail to distinguish transient stress-related dysglycemia from baseline metabolic status, potentially limiting their ability to accurately estimate risk and predict adverse outcomes following myocardial infarction [28].

Recognition of these limitations has led to the development of the concept of relative hyperglycemia, which reflects the magnitude of acute glucose elevation in relation to an individual's chronic glycemic state. Unlike absolute hyperglycemia, relative hyperglycemia provides insight into the degree of metabolic stress imposed by the acute illness itself. This approach acknowledges that identical admission glucose values may carry different clinical implications depending on a patient's baseline glycemic control. Thus, relative hyperglycemia may represent a more meaningful marker of disease severity than admission glucose alone [29].

The stress hyperglycemia ratio (SHR) has emerged as the most widely studied indicator of relative hyperglycemia. SHR integrates acute and chronic glycemic information by relating admission glucose concentration to estimated average glucose derived from glycated hemoglobin (HbA1c). Because HbA1c reflects average glycemic exposure over the preceding two to three months, SHR provides a measure of the acute deviation from baseline glucose homeostasis occurring during the index event. This characteristic allows SHR to more accurately capture the metabolic response to acute myocardial infarction [30].

The most commonly utilized formula for calculating SHR involves estimation of average glucose using HbA1c values according to the following equation:

Estimated average glucose (mg/dL) = (28.7 × HbA1c) – 46.7

SHR is subsequently calculated as:

SHR = Admission glucose (mg/dL) / Estimated average glucose (mg/dL)

This method has been extensively employed in cardiovascular studies and has demonstrated good reproducibility across diverse patient populations. The simplicity and availability of the required laboratory parameters make SHR an attractive biomarker for routine clinical application [31].

Compared with admission glucose alone, SHR offers several potential advantages in the evaluation of STEMI patients. By incorporating baseline glycemic status, SHR reduces the confounding effect of chronic hyperglycemia and improves discrimination between acute metabolic stress and established diabetes. Furthermore, SHR appears to exhibit stronger associations with adverse cardiovascular outcomes than either admission glucose or HbA1c individually. This enhanced prognostic performance may reflect its ability to integrate information regarding both chronic metabolic dysfunction and the intensity of the acute stress response [32].

Accumulating evidence supports the prognostic significance of SHR in patients with acute myocardial infarction. Elevated SHR has been associated with increased risks of in-hospital mortality, major adverse cardiovascular events, impaired left ventricular function, and microvascular obstruction following STEMI. Several studies have also demonstrated that SHR predicts poor outcomes independently of traditional cardiovascular risk factors, suggesting that relative hyperglycemia provides incremental prognostic information beyond established clinical variables [33].

Importantly, emerging evidence indicates that SHR may have a direct relationship with coronary thrombosis. Higher SHR values have been associated with increased intracoronary thrombus burden, suggesting that relative hyperglycemia may reflect activation of biological pathways involved in thrombogenesis. The mechanisms underlying this association are likely multifactorial and include enhanced platelet activation, increased thrombin generation, endothelial dysfunction, oxidative stress, and impaired fibrinolysis. These findings have generated considerable interest in SHR as a potential marker for identifying STEMI patients at increased thrombotic risk before primary PCI [34].

The prognostic utility of SHR appears to extend to both diabetic and non-diabetic populations; however, its clinical relevance may be particularly important among diabetic patients. In this setting, traditional glucose measurements are especially difficult to interpret because of pre-existing abnormalities in glycemic control. By accounting for chronic hyperglycemia through incorporation of HbA1c, SHR may provide a more accurate representation of acute metabolic stress and facilitate improved risk stratification in diabetic STEMI patients undergoing primary PCI [35].

Taken together, current evidence suggests that SHR represents a practical and biologically plausible biomarker capable of enhancing prognostic assessment in acute myocardial infarction. Its ability to integrate acute and chronic glycemic status offers important advantages over conventional glucose measurements. As interest in precision cardiovascular medicine continues to expand, SHR may emerge as a valuable tool for identifying diabetic STEMI patients at increased risk of extensive coronary thrombosis and adverse clinical outcomes.

5. Biological Links Between Relative Hyperglycemia and Coronary Thrombosis

The relationship between stress hyperglycemia ratio (SHR) and intracoronary thrombus burden is supported by several biologically plausible mechanisms involving endothelial dysfunction, oxidative stress, platelet hyperreactivity, activation of coagulation pathways, impaired fibrinolysis, and inflammation. Relative hyperglycemia reflects the magnitude of the acute metabolic response to physiological stress and may therefore represent more than a simple biomarker of disease severity. Instead, elevated SHR may actively contribute to the development of a prothrombotic environment that promotes coronary thrombosis during ST-segment elevation myocardial infarction (STEMI) [36].

One of the earliest consequences of acute hyperglycemia is endothelial dysfunction. Elevated glucose concentrations increase the generation of reactive oxygen species, reduce nitric oxide bioavailability, and impair endothelial-dependent vasodilation. Dysfunctional endothelial cells exhibit increased expression of adhesion molecules and procoagulant factors while losing their antithrombotic properties. This shift toward a pro-inflammatory and prothrombotic phenotype facilitates leukocyte recruitment, platelet adhesion, and local thrombus formation at the site of plaque disruption [37].

Oxidative stress represents another important mechanism linking relative hyperglycemia to thrombogenesis. Acute fluctuations in glucose concentrations may generate oxidative injury that exceeds that observed with sustained chronic hyperglycemia. Excessive production of reactive oxygen

species contributes to endothelial damage, oxidation of lipoproteins, and amplification of inflammatory pathways within the vascular wall. Furthermore, oxidative stress enhances platelet activation and potentiates thrombin generation, thereby promoting growth and stabilization of intracoronary thrombi [38].

Platelet hyperreactivity is a hallmark of both diabetes mellitus and stress hyperglycemia. Acute elevations in glucose have been shown to increase platelet aggregation through multiple mechanisms, including increased intracellular calcium mobilization, enhanced expression of glycoprotein receptors, and reduced sensitivity to endogenous inhibitors such as nitric oxide and prostacyclin. Activated platelets release pro-inflammatory mediators and provide a catalytic surface for thrombin generation, contributing to the development of larger and more resistant thrombi. Consequently, patients with elevated SHR may exhibit heightened platelet activity that predisposes them to increased thrombus burden during STEMI [39].

Relative hyperglycemia may also influence the coagulation cascade. Hyperglycemic states are associated with increased concentrations of coagulation factors, enhanced tissue factor expression, and accelerated thrombin generation. Thrombin serves a central role in thrombus propagation by converting fibrinogen to fibrin and amplifying platelet activation. Increased thrombin generation results in the formation of dense fibrin networks that are more resistant to degradation. Therefore, elevated SHR may identify patients with a heightened procoagulant response during acute myocardial infarction [40].

In addition to promoting coagulation, acute hyperglycemia impairs endogenous fibrinolytic activity. Increased expression of plasminogen activator inhibitor-1 (PAI-1), commonly observed in both diabetes and acute stress states, suppresses tissue plasminogen activator activity and limits fibrin degradation. The resulting imbalance between coagulation and fibrinolysis favors thrombus persistence and continued thrombus growth. This mechanism may partly explain the association between elevated SHR and larger intracoronary thrombus burden observed in clinical studies [41].

Inflammation constitutes another critical link between relative hyperglycemia and coronary thrombosis. Acute myocardial infarction triggers a robust inflammatory response characterized by activation of leukocytes and release of cytokines such as interleukin-6 and tumor necrosis factor-alpha. Hyperglycemia further amplifies this response by stimulating inflammatory signaling pathways and promoting endothelial activation. The close interaction between inflammation and coagulation, often referred to as thrombo-inflammation, enhances platelet recruitment and fibrin formation, thereby contributing to thrombus propagation [42].

Microvascular dysfunction may represent an additional consequence of stress-induced metabolic disturbances. Hyperglycemia-mediated endothelial injury, platelet activation, and distal embolization of thrombotic material impair coronary microcirculatory function even after successful restoration of epicardial blood flow. Microvascular obstruction and the no-reflow phenomenon are associated with larger infarct size, impaired ventricular recovery, and worse clinical outcomes. Since high thrombus burden is a major determinant of no-reflow, the association between elevated SHR and impaired myocardial reperfusion may reflect, at least in part, enhanced thrombogenic activity [43].

Importantly, these mechanisms are likely to be particularly relevant in diabetic patients presenting with STEMI. Chronic exposure to hyperglycemia induces persistent endothelial dysfunction, oxidative stress, platelet abnormalities, and impaired fibrinolysis. During acute myocardial infarction, superimposed stress hyperglycemia may further intensify these pathological processes, creating a synergistic effect that promotes extensive coronary thrombosis. Thus, SHR may serve not only as a marker of acute physiological stress but also as an indicator of amplified thrombotic susceptibility in diabetic individuals [44].

Collectively, current evidence suggests that elevated SHR reflects the convergence of multiple biological pathways involved in coronary thrombogenesis. The interaction between relative hyperglycemia, inflammation, endothelial dysfunction, platelet activation, and impaired fibrinolysis provides a mechanistic framework supporting the observed relationship between SHR and intracoronary

thrombus burden. Understanding these mechanisms may help explain the prognostic significance of SHR and reinforce its potential utility in identifying diabetic STEMI patients at increased risk of adverse outcomes following primary PCI.

6. Clinical Evidence Linking Stress Hyperglycemia Ratio to Coronary Thrombus Burden

The relationship between stress hyperglycemia ratio (SHR) and intracoronary thrombus burden has emerged as an area of growing interest in interventional cardiology. Although hyperglycemia has long been associated with adverse outcomes in acute myocardial infarction, recent studies suggest that relative hyperglycemia, rather than absolute glucose elevation, may better reflect the pathophysiological processes contributing to coronary thrombosis. Available evidence indicates that SHR is associated with angiographic markers of thrombus burden and may provide incremental prognostic value in patients undergoing primary percutaneous coronary intervention (PCI) [45].

One of the earliest studies directly evaluating this association was conducted by Chu and colleagues, who investigated the relationship between SHR and intracoronary thrombus burden in diabetic patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary PCI. The study included 227 diabetic STEMI patients and demonstrated that individuals with large thrombus burden exhibited significantly higher SHR values than those with smaller thrombi. Furthermore, SHR remained an independent predictor of large thrombus burden after multivariable adjustment for conventional cardiovascular risk factors. Patients with SHR values ≥1.19 had a markedly greater incidence of large thrombus burden compared with those with lower SHR values, highlighting the potential utility of SHR in identifying diabetic STEMI patients at increased thrombotic risk [46].

Subsequent investigations expanded these observations to broader acute coronary syndrome populations. In a recent study involving patients with acute coronary syndromes, elevated SHR was independently associated with high thrombus burden as assessed angiographically. Importantly, this relationship persisted after adjustment for traditional clinical variables and markers of disease severity, suggesting that relative hyperglycemia may provide unique prognostic information not captured by established risk factors alone. These findings reinforced the concept that SHR reflects underlying biological processes directly involved in thrombus formation and propagation [47].

Evidence supporting the prognostic significance of relative hyperglycemia has also been reported in STEMI populations using alternative measures of acute glycemic stress. Studies evaluating stress-induced hyperglycemia have demonstrated significant associations with impaired myocardial reperfusion, increased thrombotic complications, and adverse clinical outcomes following primary PCI. Although not all investigations specifically employed SHR, their results support the broader hypothesis that acute metabolic disturbances contribute to coronary thrombogenesis and influence procedural success [48].

Beyond its association with thrombus burden, elevated SHR has consistently been linked to markers of impaired myocardial reperfusion. Patients with higher SHR values exhibit increased rates of microvascular obstruction and no-reflow phenomenon despite successful restoration of epicardial coronary blood flow. Because extensive thrombus burden is a major determinant of distal embolization and microvascular dysfunction, these findings provide indirect evidence supporting the relationship between SHR and enhanced thrombogenic activity during STEMI [49].

The prognostic implications of SHR extend to both short- and long-term clinical outcomes. Several studies have demonstrated that elevated SHR independently predicts in-hospital mortality, major adverse cardiovascular events (MACE), and impaired ventricular recovery after acute myocardial infarction. Importantly, SHR appears to outperform admission glucose concentrations in risk stratification models, emphasizing the importance of accounting for baseline glycemic status when assessing the impact of acute hyperglycemia. These observations suggest that SHR captures clinically relevant information beyond traditional metabolic markers [50].

Despite the promising findings reported to date, several limitations characterize the current body of evidence. Most available studies have employed retrospective observational designs, thereby limiting

causal inference. Sample sizes have frequently been modest, and considerable heterogeneity exists regarding patient populations, definitions of thrombus burden, and SHR cutoff values. Moreover, diabetic and non-diabetic patients are often analyzed together despite important differences in baseline glycemic status and thrombotic risk. These methodological considerations highlight the need for cautious interpretation of existing data [51].

Another important limitation relates to the absence of standardized thresholds for defining high-risk SHR values. Although individual studies have proposed specific cutoff points associated with increased thrombus burden or adverse outcomes, these values vary substantially across populations. Differences in laboratory methodologies, timing of glucose assessment, and patient characteristics may contribute to this variability. Establishing universally applicable SHR thresholds will therefore require large prospective multicenter investigations [52].

Nevertheless, the consistency of findings across diverse cardiovascular settings supports the biological plausibility and potential clinical relevance of SHR. The repeated observation that elevated SHR is associated with larger thrombus burden, impaired myocardial reperfusion, and worse clinical outcomes suggests that relative hyperglycemia may represent an important component of the residual risk profile in diabetic STEMI patients. Consequently, SHR has emerged as a promising biomarker capable of enhancing risk stratification before primary PCI [53].

Collectively, current evidence indicates that SHR may provide valuable prognostic information regarding coronary thrombosis in diabetic patients presenting with STEMI. Although further validation is required before widespread clinical implementation, available studies support the hypothesis that relative hyperglycemia reflects an amplified thrombotic response during acute myocardial infarction. Future prospective studies are needed to determine whether incorporation of SHR into clinical decision-making algorithms can improve outcomes through more individualized therapeutic strategies.

7. Prognostic Implications of Stress Hyperglycemia Ratio in Diabetic STEMI

Beyond its association with intracoronary thrombus burden, accumulating evidence suggests that the stress hyperglycemia ratio (SHR) has important prognostic implications in patients presenting with ST-segment elevation myocardial infarction (STEMI). Because SHR integrates acute metabolic stress with chronic glycemic status, it may provide a more accurate reflection of disease severity than isolated admission glucose measurements. In diabetic STEMI patients, in whom interpretation of absolute glucose values is often challenging, SHR has emerged as a potentially valuable marker for identifying individuals at increased risk of adverse clinical outcomes following primary percutaneous coronary intervention (PCI) [54].

Several studies have demonstrated a significant association between elevated SHR and increased in-hospital mortality among patients with acute myocardial infarction. Relative hyperglycemia likely reflects activation of neurohormonal, inflammatory, and thrombotic pathways that contribute to hemodynamic instability and myocardial injury. Importantly, the prognostic value of SHR appears to persist after adjustment for traditional cardiovascular risk factors and baseline glycemic control, suggesting that acute metabolic disturbances provide independent information regarding patient outcomes [55].

The relationship between SHR and major adverse cardiovascular events (MACE) has also been increasingly recognized. Higher SHR values have been associated with increased risks of recurrent myocardial infarction, repeat revascularization, heart failure hospitalization, and cardiovascular death during follow-up. This association may reflect the combined effects of enhanced thrombogenicity, impaired myocardial reperfusion, and greater susceptibility to adverse ventricular remodeling among patients experiencing pronounced stress hyperglycemia during STEMI [56].

Microvascular dysfunction represents another important determinant of prognosis following primary PCI. Even after successful restoration of epicardial coronary flow, inadequate perfusion at the level of the coronary microcirculation may result in persistent myocardial ischemia and larger infarct size. Several investigations have reported that elevated SHR is associated with increased incidence of

microvascular obstruction and the no-reflow phenomenon, both of which are recognized predictors of poor functional recovery and adverse cardiovascular outcomes. These findings support the hypothesis that relative hyperglycemia contributes to impaired myocardial reperfusion through mechanisms involving endothelial dysfunction, inflammation, and distal embolization [57].

Emerging evidence has also suggested a possible relationship between SHR and the risk of stent thrombosis. Although the exact mechanisms remain incompletely understood, acute metabolic disturbances may potentiate platelet activation and coagulation pathway activity, thereby increasing susceptibility to thrombotic complications after PCI. Recent observational studies have reported higher SHR values among patients developing early stent thrombosis following STEMI, highlighting the need for further investigation into the potential role of SHR in identifying patients requiring more intensive antithrombotic surveillance [58].

Left ventricular dysfunction and adverse remodeling are major determinants of long-term prognosis after STEMI. The severity of myocardial injury influences the development of chronic heart failure and contributes substantially to cardiovascular mortality. Studies employing cardiac magnetic resonance imaging have demonstrated associations between elevated SHR and larger infarct size, increased microvascular obstruction, and impaired ventricular function. These observations suggest that relative hyperglycemia may identify patients at increased risk of progressive ventricular dysfunction despite successful reperfusion therapy [59].

Importantly, several studies have demonstrated that SHR provides superior prognostic information compared with admission glucose alone. While isolated glucose measurements fail to distinguish acute stress responses from chronic dysglycemia, SHR accounts for baseline glycemic status through incorporation of glycated hemoglobin values. This distinction is particularly relevant in diabetic populations, where chronic hyperglycemia may obscure the prognostic significance of acute metabolic derangements. Consequently, SHR may offer improved risk discrimination among diabetic STEMI patients undergoing primary PCI [60].

Despite the encouraging findings reported thus far, important limitations should be acknowledged. Most studies evaluating the prognostic significance of SHR have employed observational designs, and the majority have been conducted within single-center settings. Moreover, considerable heterogeneity exists regarding patient populations, follow-up duration, and optimal SHR cutoff values. Therefore, although SHR appears to be a promising biomarker for risk stratification, additional prospective multicenter studies are necessary before its routine incorporation into clinical practice can be recommended [61].

Nevertheless, the consistency of evidence linking elevated SHR with mortality, MACE, no-reflow, ventricular dysfunction, and thrombotic complications underscores its potential clinical relevance. Given its simplicity, low cost, and widespread availability, SHR may represent a practical tool for enhancing prognostic assessment in diabetic STEMI patients. Future studies should determine whether SHR-guided therapeutic strategies can improve clinical outcomes through more individualized management approaches [61].

In summary, SHR has emerged as a promising prognostic biomarker in diabetic patients presenting with STEMI. Beyond its association with intracoronary thrombus burden, elevated SHR appears to identify patients at increased risk of adverse in-hospital and long-term outcomes following primary PCI. Integration of SHR into existing risk assessment models may facilitate earlier recognition of high-risk individuals and contribute to the development of more personalized strategies aimed at improving prognosis in this vulnerable patient population.

Conclusion

Diabetic patients presenting with ST-segment elevation myocardial infarction constitute a particularly vulnerable population characterized by diffuse coronary artery disease, enhanced thrombogenicity, impaired myocardial reperfusion, and persistently worse clinical outcomes despite advances in primary percutaneous coronary intervention. Emerging evidence suggests that the stress hyperglycemia ratio, as a marker of relative hyperglycemia integrating both acute metabolic stress and baseline glycemic status,

provides superior prognostic information compared with admission glucose alone. Elevated SHR has been consistently associated with larger intracoronary thrombus burden, microvascular dysfunction, no-reflow phenomenon, adverse ventricular remodeling, and increased risks of mortality and major adverse cardiovascular events. The biological plausibility of these associations is supported by the interplay between acute hyperglycemia and key pathways involved in thrombogenesis, including endothelial dysfunction, oxidative stress, inflammation, platelet hyperreactivity, activation of coagulation cascades, and impaired fibrinolysis. Although current evidence supports the potential role of SHR as a practical and readily available biomarker for identifying diabetic STEMI patients at heightened thrombotic risk, most available data are derived from observational studies with heterogeneous methodologies. Therefore, large prospective multicenter investigations are warranted to establish standardized SHR thresholds, validate its incremental prognostic value, and determine whether SHR-guided therapeutic strategies can improve outcomes in this high-risk population. Incorporation of SHR into contemporary risk stratification models may ultimately facilitate a more individualized approach to the management of diabetic patients undergoing primary PCI, thereby contributing to improved prognostic assessment and optimization of clinical care.

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