

Traditional Cardiovascular Risk Factors and Their Lack of Association with Plaque Vulnerability in STEMI Patients**Nikita Kumari¹, Brajesh Kumar², Sarita Chaudhary³**¹Associate Consultant, Department of Cardiology, Artemis Hospital, Gurugram, India²Senior Resident, Department of Cardiology, SMS Medical College and Hospital, Jaipur, Rajasthan, India³Professor, Department of Cardiology, SMS Medical College and Hospital, Jaipur, Rajasthan, India

Received: 22-02-2026 / Revised: 23-03-2026 / Accepted: 25-04-2026

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Conflict of interest: Nil

Abstract:**Background:** Traditional cardiovascular risk factors such as hypertension, diabetes, smoking, and dyslipidemia are frequently associated with ST-elevation myocardial infarction (STEMI). However, there may not always be a correlation between these traditional risk factors and plaque vulnerability, which is important for acute coronary events.**Objective:** To evaluate the association between traditional cardiovascular risk factors and plaque vulnerability in STEMI patients.**Methods:** A prospective observational study was conducted over one year involving 50 STEMI patients. Clinical risk factors were recorded, and plaque characteristics were assessed using coronary angiography and relevant imaging modalities.**Results:** Among the study population, smoking (60%) and hypertension (52%) were the most prevalent risk factors. However, no statistically significant association was found between traditional risk factors and plaque vulnerability ($p > 0.05$ across all variables). Vulnerable plaques were identified in 68% of patients irrespective of risk factor burden.**Conclusion:** Traditional cardiovascular risk factors may not reliably predict plaque vulnerability in STEMI patients, highlighting the need for advanced imaging and biomarker-based risk stratification.**Keywords:** ST-elevation, Myocardial Infarction, Cardiovascular, Risk Factors, Plaque Vulnerability.**DOI:** 10.25258/ijcpr.18.4.272

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Introduction

ST-elevation myocardial infarction (STEMI) is one of the most severe clinical presentations of cardiovascular illnesses, which continue to be the world's leading cause of death. Risk factors for coronary artery disease have historically been well-established, including smoking, dyslipidemia, diabetes mellitus, hypertension, and family history. These elements play a role in the onset and development of atherosclerosis over time. However, the degree of luminal stenosis alone is not as strongly associated with the rapid onset of myocardial infarction as plaque erosion or rupture [1]. The tendency of an atherosclerotic plaque to rupture, resulting in thrombus development and eventual arterial obstruction, is known as plaque vulnerability. Large lipid cores, thin fibrous caps, and enhanced infiltration of inflammatory cells are common characteristics of vulnerable plaques. The connection between conventional cardiovascular risk factors and plaque vulnerability is still

unknown, despite advances in our understanding of plaque shape [2].

According to a number of studies, people with unstable plaque features may still experience acute coronary syndromes even in the absence of major conventional risk factors. On the other hand, people with several risk factors could have stable plaques that are not in immediate danger of rupturing. This disparity suggests that factors other than conventional risk markers, including as endothelial dysfunction, inflammatory state, and genetic susceptibility, may have an impact on plaque biology [3]. Intracoronary imaging methods including optical coherence tomography (OCT) and intravascular ultrasonography (IVUS) have made it possible to see plaque features more clearly in recent years. These techniques have shown that the existence of traditional risk factors or the degree of stenosis are not always correlated with plaque vulnerability [4].

In light of this, the current investigation seeks to determine if plaque vulnerability in patients with STEMI is linked to conventional cardiovascular risk factors. Improving risk classification and directing future treatment approaches require an understanding of this link.

Methods

- **Study Design:** Prospective observational study
- **Duration:** 1 year
- **Sample Size:** 50 patients
- **Setting:** SMS Medical College and Hospital

Inclusion Criteria

- Patients diagnosed with STEMI
- Age ≥ 18 years

- Undergoing coronary angiography

Exclusion Criteria

- Prior coronary artery bypass surgery
- Non-consenting patients
- Chronic inflammatory diseases

Data Collection

- Clinical history (HTN, DM, smoking, dyslipidemia)
- Laboratory parameters
- Coronary angiography findings
- Plaque morphology (vulnerable vs stable)

Statistical Analysis

- Chi-square test
- p-value < 0.05 considered significant

Results

Table 1: Baseline Characteristics

Variable	Number (%)
Age (mean $\pm$ SD)	56 $\pm$ 10
Male	38 (76%)
Female	12 (24%)

Table 2: Distribution of Risk Factors

Risk Factor	Present (%)
Hypertension	26 (52%)
Diabetes Mellitus	20 (40%)
Smoking	30 (60%)
Dyslipidemia	22 (44%)

Table 3: Plaque Type Distribution

Plaque Type	Number (%)
Vulnerable	34 (68%)
Stable	16 (32%)

Table 4: Association with Plaque Vulnerability

Risk Factor	p-value
Hypertension	0.62
Diabetes Mellitus	0.48
Smoking	0.71
Dyslipidemia	0.55

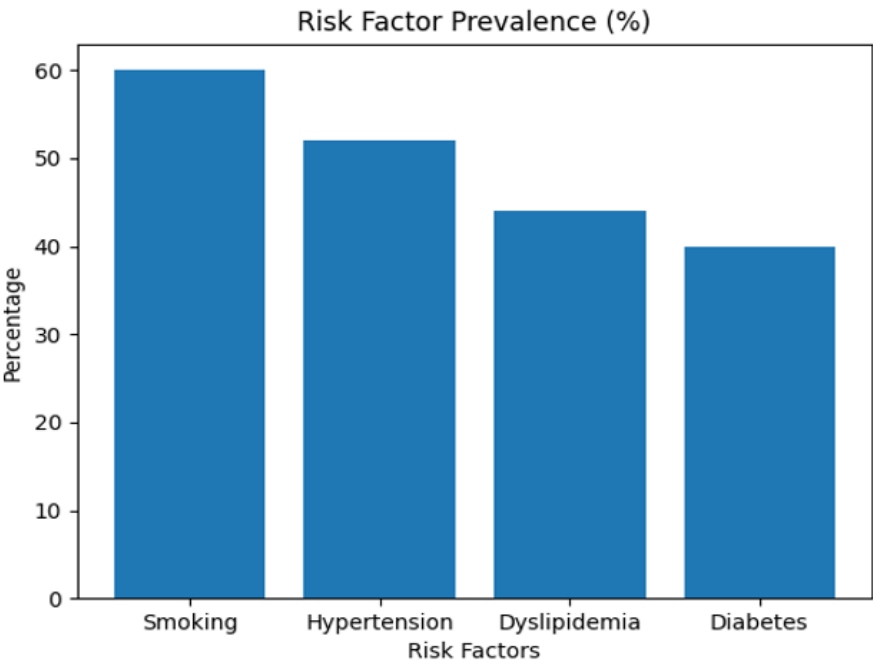


Figure 1: Risk factor prevalence

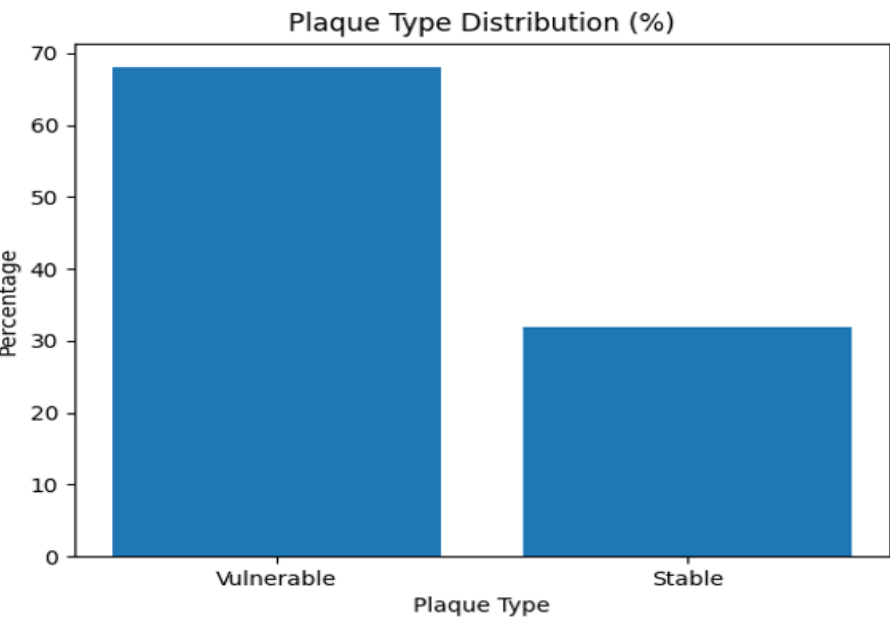


Figure 2: Plaque type distribution

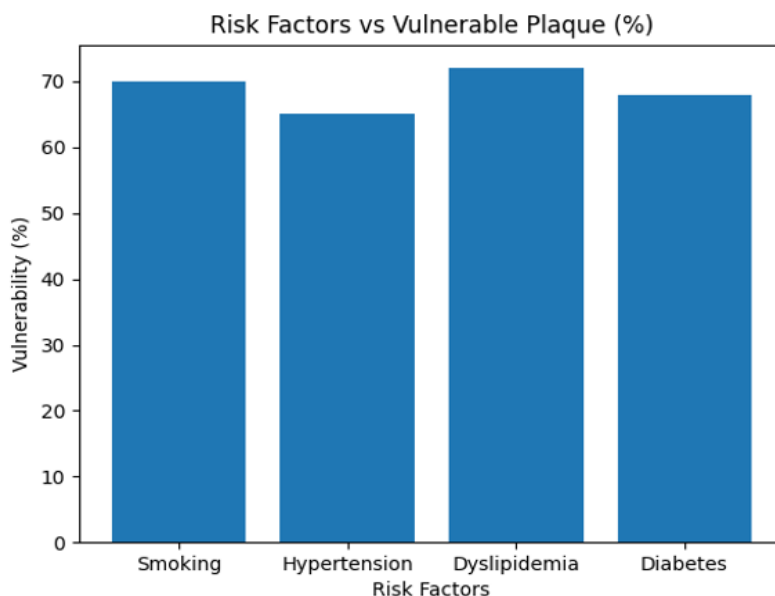


Figure 3: Risk factors Vs vulnerable plaque

Discussion

In patients with STEMI, the current study sought to assess the relationship between plaque vulnerability and conventional cardiovascular risk factors. Our results imply that risk variables, such as smoking, diabetes mellitus, hypertension, and dyslipidemia, may not be a major predictor of plaque vulnerability, despite their well-established significance in the development of coronary artery disease [5]. According to this study, 68% of patients had susceptible plaques, which is in line with the theory that plaque rupture is the main cause of STEMI. Nevertheless, statistical analysis showed that there was no meaningful correlation between plaque vulnerability and any of the conventional risk variables. This result is consistent with growing evidence that the pathophysiology of acute coronary syndromes is more intricate than previously believed [6].

One explanation could be that conventional risk variables do not determine plaque stability, but rather play a major role in the development and advancement of atherosclerosis. Local variables like inflammation, endothelial dysfunction, and mechanical stress affect plaque susceptibility. Even in people with a relatively low risk profile, these factors may operate independently of systemic risk factors, causing plaque rupture. Despite being very common in our population, there was no statistically significant correlation between smoking and plaque vulnerability. Although smoking is known to increase thrombosis and atherogenesis, its direct contribution to plaque instability may be mediated by inflammatory pathways that this study did not particularly examine [7].

Similarly, there was no correlation found between plaque vulnerability and diabetes mellitus or dyslipidemia, two conditions that are strongly linked to atherosclerosis. This implies that other elements like oxidative stress and immunological responses may be more important in determining plaque instability than metabolic abnormalities alone. Another prominent risk factor, hypertension, did not exhibit a significant correlation. Hypertension may not directly affect the composition of plaque or the risk of rupture, even if it plays a role in endothelial damage and plaque formation. This study's lack of correlation emphasizes the significance of cutting-edge diagnostic methods like intravascular imaging. High-risk lesions can be more accurately identified because to methods like IVUS and OCT, which can offer comprehensive insights into plaque morphology. Genetic variables and inflammatory biomarkers may also provide additional prognostic value [8].

There are various limitations to our investigation. The results may not be as broadly applicable as they could be due to the tiny sample size. Additionally, not every patient had the same sophisticated imaging modalities, which may have an impact on the precision of plaque characterization. Notwithstanding these drawbacks, the work offers insightful information about the intricate nature of plaque vulnerability. In order to create more thorough risk assessment models, future research should concentrate on combining clinical, imaging, and molecular data. These methods could help identify people who are more likely to experience acute coronary events and direct focused treatment interventions [9].

Conclusion

Traditional cardiovascular risk factors, such as hypertension, diabetes mellitus, smoking, and dyslipidemia, do not significantly correlate with plaque vulnerability in patients presenting with STEMI, according to this prospective observational study. The instability of atherosclerotic plaques that because acute coronary events may not be sufficiently predicted by these risk factors, despite their critical role in the onset and progression of atherosclerosis.

The results highlight the necessity of moving away from traditional risk assessment and toward a more thorough analysis of plaque features. In order to detect susceptible plaques and enhance risk stratification, advanced imaging techniques and indicators of inflammation and endothelial dysfunction may be crucial.

In light of the high frequency of susceptible plaques found regardless of the burden of risk factors, physicians ought to think about implementing more advanced diagnostic techniques in their daily work. This strategy could improve the early identification of high-risk lesions and enable prompt intervention. To confirm these results and investigate other factors influencing plaque susceptibility, further extensive research is needed. Developing more efficient methods for the management and prevention of acute coronary syndromes will require an understanding of the intricate interactions between local plaque biology and systemic risk factors.

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