Open Access Emergency Medicine

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Diagnostic Value of D-Dimer in Patients Visiting the Emergency Department with Symptoms of Acute Coronary Syndrome: A Retrospective Observational Study

Mohammad K Alsuleiman[1], Ahmad A Oqlat[1], Abdel Salam Ewais[2], Mosab Said[3], Fadi Jalal Alkhawaja[3], Liqaa A Raffee[1], Anwar Mustafa[1]

[1]Department of Accident and Emergency Medicine, Faculty of Medicine, Jordan University of Science and Technology, Irbid, 22110, Jordan; [2]Department of Family Medicine, Radiology and Emergency Medicine, Faculty of Medicine, the Hashemite University, Zarqa, 13133, Jordan; [3]Faculty of Medicine, Yarmouk University, Irbid, 21163, Jordan

Correspondence: Mohammad K Alsuleiman, Email mkalsuleiman@just.edu.jo

Background: D-dimer is a fibrin degradation product formed during fibrinolysis and reflects the activation of coagulation and fibrinolytic pathways. The role of D-dimer in acute coronary syndrome (ACS) remains uncertain. This study was designed to analyse differences in D-dimer levels in patients with different forms of ACS and the diagnostic efficiency and possible complementary role of the D-dimer biomarker.

Methods: This study applied a retrospective observational design and included adult patients presenting to the emergency department with chest pain. Differences in D-dimer levels across diagnostic categories were analysed. To evaluate the diagnostic performance of D-dimer, receiver operating characteristic (ROC) analysis was conducted, which involved calculations of area under the curve (AUC), sensitivity, specificity, and likelihood ratios. Also, the incremental value of D-dimer over clinical data and cardiac troponin was determined through a multivariate logistic regression model.

Results: The study found that the level of D-dimer was significantly higher in patients with ST elevation myocardial infarction (STEMI) and Non-ST elevation myocardial infarction (NSTEMI) compared to patients with unstable angina (UA) and non-specific chest pain ($p < 0.001$). As per ROC analysis, the level of D-dimer demonstrated modest discrimination between STEMI/NSTEMI and unstable angina (AUC 0.618; 95% CI 0.538–0.697), and a fair discrimination between STEMI/NSTEMI and non-specific chest pain (AUC 0.668; 95% CI 0.601–0.736). At a cutoff of 0.50 mg/L, sensitivity and specificity were limited. In multivariable models, D-dimer did not provide incremental diagnostic value beyond clinical variables and cardiac troponin (ΔAUC not significant).

Conclusion: Despite higher D-dimer concentrations in myocardial infarction, the marker exhibited modest diagnostic performance and did not provide additional diagnostic value over cardiac troponin. Therefore, D-dimer is suggested to be used as an adjunct rather than primary diagnostic instrument in patients presenting with suspected acute coronary syndrome.

Keywords: D-dimer, acute coronary syndrome, unstable angina, myocardial infarction, chest pain, emergency department

Introduction

Acute coronary syndrome (ACS) is a spectrum of clinical conditions caused by acute reduction in coronary blood flow resulting in myocardial ischemia. It remains a leading cause of morbidity and mortality worldwide.[1] Early diagnosis and appropriate risk stratification are essential to improve patient outcomes and reduce complications.

Current guideline-based diagnostic strategies for ACS rely on clinical assessment, electrocardiography (ECG), and cardiac biomarkers with a particular emphasis on high-sensitivity cardiac troponin as the cornerstone for the diagnosis of myocardial infarction.[2–4] High-sensitivity troponin assays have enhanced the detection of the earliest myocardial injury and stratification of risks in patients who present with suspected ACS.[2–4] Nevertheless, cardiac troponin levels may be

https://doi.org/10.2147/OAEM.S600069
Received: 30 January 2026
Accepted: 28 April 2026
Published: 5 May 2026

normal, especially during the early presence of the disease, and ECG results may be non-diagnostic in some patients, leading to diagnostic uncertainty in individuals presenting with chest pain and suspected ACS.[2,4]

D-dimer is a fibrin degradation product formed during fibrinolysis and reflects activation of coagulation and fibrinolytic pathways.[5] Thrombus formation has a central role in the pathophysiology of ACS; elevated levels of D-dimer can be a marker of the thrombotic process in a patient with myocardial infarction.[6] Several studies have suggested that there may be a relationship between elevated D-dimer and ACS, but its place in diagnosis has not been adopted, and it is not included in guideline-recommended diagnostic algorithms.[7,8]

Although high-sensitivity troponin is widely used, the diagnosis can still be difficult, especially in early presenters as well as in individuals with unstable angina in whom troponin levels may remain within the normal range despite the presence of myocardial ischemia. Consequently, there has been a continued interest in identifying more biomarkers that can help in the assessment and risk stratification of suspected patients with ACS. Past researchers examining the association between D-dimer and ACS have shown mixed findings, and the clinical usefulness of D-dimer in this context is debatable.[7,8]

Therefore, this study aimed to assess the variation in D-dimer levels in patients with various forms of acute coronary syndrome and to determine the diagnostic performance and its ability to potentially add value as a complementary biomarker in patients presenting with chest pain.

Methodology

Study Design and Setting

This is a retrospective observational study conducted at King Abdullah University Hospital (KAUH), which is a tertiary care hospital in North Jordan.

Study Population

Adult patients presenting to the emergency department with chest pain between January 2020 and December 2024 were retrospectively included in the study. Only patients who had D-dimer measured at the time of presentation were eligible for inclusion. Typical chest pain was defined as retrosternal discomfort of a heavy nature, precipitated by exertion and relieved by rest or nitroglycerin.

The D-dimer testing was done based on the clinical judgment of the treating emergency physician as part of the primary examination of patients with suspected cardiac chest pain. Owing to the retrospective characteristics of the study, patients whose D-dimer were measured at the time of presentation only were included in the analysis. The demographic (age and sex), clinical variables (medical history (diabetes mellitus, hypertension, dyslipidemia, etc) as well as cardiac troponin level), were extracted from electronic medical records.

Diagnostic Group Classification

Patients were categorized based on their final clinical diagnosis into the following groups:

(1) ST-elevation myocardial infarction (STEMI),
(2) Non-ST-elevation myocardial infarction (NSTEMI),
(3) Unstable angina (UA), and
(4) Non-specific chest pain.

For selected analyses, patients with STEMI and NSTEMI were grouped together as myocardial infarction, and comparisons were performed against unstable angina or non-specific chest pain, as appropriate.

Inclusion Criteria

The following inclusion criteria were applied;

1. Age ≥ 18 years.
2. Presentation to the emergency department with chest pain as the chief complaint.
3. The present D-dimer was documented at the time of presentation.
4. D-dimer and troponin measurements were taken within one hour of emergency department presentation.

Exclusion Criteria

Patients were excluded in case of the following:

- Recent surgical procedures, such as the coronary artery bypass grafting.
- Past venous thromboembolism.
- Active malignancy.
- Long-term liver/kidney disease.
- Hematologic diseases (eg. lymphoma, leukaemia).
- Pregnancy.
- Recent myocardial infarction.
- Additional clinical/laboratory evidence of infection (38°C or increased C-reactive protein, if present).
- The pre-hospital use of anticoagulant therapy (heparin, low-molecular-weight heparin or warfarin).

Study Sample Size

A total of 349 patients were included in the study and categorized into two groups: 190 patients with ACS and 159 patients with non-cardiac chest pain.

Definition and Classification of ACS

The diagnosis of ACS was based on clinical presentation, electrocardiographic (ECG) findings, cardiac troponin levels, and results of coronary angiography.

ST-elevation myocardial infarction (STEMI) was defined by the presence of ischemic chest pain with ST-segment elevation on ECG and elevated cardiac troponin levels. Non-ST-elevation myocardial infarction (NSTEMI) was defined by ischemic symptoms with elevated troponin levels in the absence of ST-segment elevation. Unstable angina was defined by ischemic chest pain without elevated troponin levels but with ≥50% coronary artery stenosis confirmed by coronary angiography.

Non-specific chest pain was defined as chest pain in patients in whom ACS was excluded and no alternative diagnosis was established after clinical evaluation. Final diagnoses were based on the discharge diagnosis documented in the medical record after completion of all clinical assessments, laboratory investigations, ECG evaluation, and cardiology consultation.

Laboratory Measurements

Blood samples for D-dimer and cardiac troponin I were collected within the first hour of presentation to the emergency department. The initial troponin level was recorded for analysis, while serial troponin measurements were performed when clinically indicated, according to institutional protocols.

Cardiac troponin I was measured using the Atellica IM High-Sensitivity Troponin I assay (Siemens Healthineers), with values ≤0.05 ng/mL considered within the normal range.

D-dimer levels were measured using the INNOVANCE® D-Dimer assay (Siemens Healthineers), an automated particle-enhanced immunoturbidimetric assay for the quantitative determination of cross-linked fibrin degradation products, with a predefined cutoff value of 0.5 mg/L. The same assays were used throughout the study period.

Ethical Approval

Approval for the research was granted by the Institutional Review Board (IRB number: 2024/542) from the faculty of Medicine, Jordan University of Science and Technology. The research was conducted in accordance with the Declaration

of Helsinki. Informed consent was not required due to the retrospective nature of the research and the fact that the anonymity of the participants could be maintained.

Statistical Analysis

Analyses were performed using Stata/SE version 16.1 (StataCorp, College Station, TX, USA) with a two-sided significance level of $\alpha = 0.05$.

Categorical variables were presented as counts and percentages and compared using the chi-square test. Continuous variables were presented as mean ± standard deviation or median with interquartile range, depending on distribution.

Because D-dimer and troponin levels were markedly right-skewed, they were compared across diagnostic groups using the Kruskal–Wallis test.

Associations with D-dimer levels were evaluated using linear regression with unstable angina (UA) as the reference diagnosis category. A prespecified multivariable model adjusted for age, sex, diabetes mellitus, hypertension, ischemic heart disease, heart failure, dyslipidemia, and troponin levels. Robust (Huber–White) standard errors were used. A sensitivity analysis was performed using log-transformed D-dimer values [ln (D-dimer + 0.01)], with exponentiated coefficients interpreted as ratios.

Diagnostic performance was assessed using receiver operating characteristic (ROC) curves and area under the curve (AUC) for STEMI/NSTEMI versus unstable angina and STEMI/NSTEMI versus non-specific chest pain. Sensitivity, specificity, and likelihood ratios were calculated at a predefined cutoff of 0.50 mg/L.

Incremental diagnostic value was evaluated by comparing AUCs from logistic regression models with and without D-dimer. Firth-penalized logistic regression was used as a sensitivity analysis to account for potential separation. Complete-case analysis was applied for model covariates.

Results

A total of 349 emergency department presentations with chest pain were analyzed. Representing 30.9%, 108 participants had STEMI/ NSTEMI, 23.5% (n = 82) had unstable angina (UA), and 45.6% (159) had non-specific chest pain. As shown in Table 1, the mean age of the participants was 55.2 (11.9) years, with 75% being male.

Table 1 Baseline Characteristics of the Study Population According to Final Diagnosis

	Total N=349	**STEMI/NSTEMI n=108**	**UA n=82**	**Non-specific n=159**	**p value**
Age in years, mean (SD)	55.2 (11.9)	57.9 (11.2)	58.2 (11.6)	51.7 (11.7)	<0.001
Gender					<0.001
Male	265 (75.9)	84 (77.8)	76 (92.7)	105 (66.0)	
Female	84 (24.1)	24 (22.2)	6 (7.3)	54 (34.0)	
Medical history					
DM	145 (41.6)	51 (47.2)	42 (51.2)	52 (32.7)	0.008
HTN	183 (52.4)	58 (53.7)	52 (63.4)	73 (45.9)	0.034
IHD	123 (35.2)	43 (39.8)	47 (57.3)	33 (20.8)	<0.001
HF	21 (6.0)	5 (4.6)	9 (11.0)	7 (4.4)	0.097
Dyslipidemia	12 (3.4)	0 (0.0)	6 (7.3)	6 (3.8)	0.022
Biomarkers, median (IQR)					

(*Continued*)

Table 1 (Continued).

	Total N=349	STEMI/NSTEMI n=108	UA n=82	Non-specific n=159	p value
Troponin level	0.01 (0.00–0.13)	0.95 (0.14–5.09)	0.01 (0.00–0.02)	0.00 (0.00–0.01)	<0.001
D-Dimer value (mg/L)	0.37 (0.23–0.68)	0.54 (0.31–1.05)	0.35 (0.24–0.60)	0.31 (0.20–0.49)	<0.001

Abbreviations: STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; UA, unstable angina; DM, diabetes mellitus; HTN, hypertension; IHD, ischemic heart disease; HF, heart failure; SD, standard deviation; IQR: Interquartile Range.

Age differed significantly across diagnostic categories ($p < 0.001$), with patients diagnosed with STEMI/NSTEMI and UA being older (57.9 ± 11.2 and 58.2 ± 11.6 years, respectively) compared with those with non-specific chest pain (51.7 ± 11.7 years). Sex distribution also differed significantly ($p < 0.001$), with the highest proportion of males observed in the UA group (92.7%), followed by STEMI/NSTEMI (77.8%) and non-specific chest pain (66.0%).

Regarding medical history, the proportions between diabetes mellitus, hypertension, ischemic heart disease, and dyslipidemia were shown to be significant between the groups of diagnoses, but not between the cases of heart failure ($p = 0.097$).

D-dimer levels differed significantly across diagnostic categories (Kruskal–Wallis $p < 0.001$), with the highest median levels observed in STEMI/NSTEMI (0.54; IQR 0.31–1.05 mg/L), followed by UA (0.35; IQR 0.24–0.60) and non-specific chest pain (0.31; IQR 0.20–0.49). Troponin levels also differed significantly ($p < 0.001$), with the highest median values in STEMI/NSTEMI (0.95; IQR 0.14–5.09), compared with UA (0.01; IQR 0.00–0.02) and non-specific chest pain (0.00; IQR 0.00–0.01) (Table 1).

Associations between diagnosis category and D-dimer levels were further examined using multivariable regression models with unstable angina as the reference category (Table 2). After adjustment for age, sex, diabetes mellitus, hypertension, ischemic heart disease, heart failure, dyslipidemia, and troponin, STEMI/NSTEMI was associated with higher D-dimer levels compared with UA (adjusted $\beta = 0.99$; 95% CI 0.16–1.82; $p = 0.019$), whereas non-specific chest

Table 2 Unadjusted and Adjusted Linear Regression for Factors Associated with the Level of D-Dimer

	Univariable		Multivariable		Sensitivity Analysis ln(D-dimer+0.01)	
	β (95% CI)	p-value	β (95% CI)	p-value	Exp(β) Ratio (95% CI)	p-value
Age in years, mean (SD)	0.02 (−0.01, 0.04)	0.181	0.02 (−0.01, 0.05)	0.161	1.02 (1.01 to 1.03)	0.001
Male	0.05 (−0.59, 0.70)	0.867	0.17 (−0.53, 0.87)	0.642	0.91 (0.73 to 1.12)	0.373
Medical history						
DM	−0.12 (−0.67, 0.44)	0.676	−0.12 (−0.77, 0.53)	0.712	0.98 (0.81 to 1.19)	0.858
HTN	−0.23 (−0.78, 0.32)	0.415	−0.24 (−0.91, 0.43)	0.48	0.99 (0.81 to 1.19)	0.879
IHD	−0.25 (−0.82, 0.33)	0.402	−0.31 (−0.97, 0.35)	0.353	0.80 (0.65 to 0.99)	0.042
HF	−0.18 (−1.34, 0.97)	0.754	−0.02 (−1.23, 1.19)	0.978	1.07 (0.72 to 1.59)	0.733
Dyslipidemia	−0.18 (−1.69, 1.32)	0.811	0.13 (−1.39, 1.65)	0.866	0.99 (0.65 to 1.51)	0.962
Biomarkers						
Troponin level	0.03 (−0.03, 0.09)	0.358	−0.02 (−0.08, 0.04)	0.545	1.01 (0.99 to 1.03)	0.389

(*Continued*)

Table 2 (Continued).

	Univariable		Multivariable		Sensitivity Analysis ln(D-dimer+0.01)	
	β (95% CI)	p-value	β (95% CI)	p-value	Exp(β) Ratio (95% CI)	p-value
Final diagnosis						
STEMI/NSTEMI	0.94 (0.20, 1.68)	0.013	0.99 (0.16, 1.82)	0.019	1.46 (1.11 to 1.92)	0.008
UA	Reference		Reference		Reference	
Non-specific chest pain	0.00 (−0.69, 0.69)	0.998	0.01 (−0.78, 0.76)	0.980	0.92 (0.74 to 1.16)	0.495

Notes: Reference category for diagnosis = Unstable Angina.
Abbreviations: DM, diabetes mellitus; HTN, hypertension; IHD, ischemic heart disease; HF, heart failure; STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; UA, unstable angina; SD, standard deviation.

Table 3 Diagnostic Performance of D-Dimer for Differentiating STEMI/NSTEMI from Unstable Angina and Non-Specific Chest Pain (Cutoff Evaluated at 0.50 mg/L)

Comparison	AUC (95% CI)	Cutoff (mg/L)	Sensitivity (%)	Specificity (%)	LR+	LR-	Correctly Classified (%)
STEMI/NSTEMI vs unstable angina, n=190	0.618 (0.538–0.697)	0.50	53.7	68.3	1.69	0.68	60.0
STEMI/NSTEMI vs non-specific chest pain, n=297	0.668 (0.601–0.736)	0.50	53.7	77.4	2.37	0.60	67.8

Abbreviations: STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; AUC, Area Under the Curve; CI, confidence interval; LR+, positive likelihood ratio; LR-, negative likelihood ratio.

pain did not differ from UA (p = 0.980). A sensitivity analysis using ln (D-dimer + 0.01) yielded consistent results (STEMI/NSTEMI vs UA ratio 1.46; 95% CI 1.11–1.92; p = 0.008).

On ROC analysis, D-dimer demonstrated modest discrimination for STEMI/NSTEMI versus UA (AUC 0.618; 95% CI 0.538–0.697) and fair discrimination for STEMI/NSTEMI versus non-specific chest pain (AUC 0.668; 95% CI 0.601–0.736). At a cutoff of 0.50 mg/L, sensitivity and specificity were limited. For STEMI/NSTEMI versus UA, sensitivity and specificity were 53.7% and 68.3%, respectively (LR+ 1.69; LR− 0.68). For STEMI/NSTEMI versus non-specific chest pain, sensitivity and specificity were 53.7% and 77.4%, respectively (LR+ 2.37; LR− 0.60) (Table 3).

To evaluate incremental diagnostic value, logistic regression models incorporating clinical variables and troponin were constructed. For STEMI/NSTEMI versus UA, the clinical/troponin model demonstrated excellent discrimination (AUC 0.962; 95% CI 0.935–0.988). The addition of D-dimer did not improve model performance (AUC 0.960; 95% CI 0.931–0.988; ΔAUC −0.002 p = 0.495), and D-dimer was not independently associated with the outcome in the diagnostic model (p = 0.319) (Table 4). Firth-penalized logistic regression yielded consistent findings, with troponin remaining strongly associated (p < 0.001).

Similarly, for STEMI/NSTEMI versus non-specific chest pain, the clinical/troponin model demonstrated excellent discrimination (AUC 0.978; 95% CI 0.963–0.993). The addition of D-dimer did not change the AUC (0.978; 95% CI 0.963–0.993; ΔAUC 0.0 p = 1.000), and D-dimer was not independently associated (β = 0.40; SE 0.32; p = 0.211) (Table 4).

Discussion

Principal Findings

In this retrospective cohort of patients presenting to the emergency department with chest pain, D-dimer levels differed significantly across diagnostic groups, with the highest levels observed in patients with ST elevation myocardial

Table 4 Discriminative Performance of Multivariable Models Including Clinical Covariates and Cardiac Troponin, with and without D-Dimer, for the Diagnosis of Myocardial Infarction

Comparison	Model	AUC (95% CI)	ΔAUC	p-value	D-dimer P value
STEMI/NSTEMI vs UA	Clinical and Troponin without D-dimer	0.962 (0.935–0.988)	—	—	
	Clinical and Troponin with D-dimer	0.960 (0.931–0.988)	−0.002	0.495	0.319
STEMI/NSTEMI vs non-specific chest pain	Clinical and Troponin without D-dimer	0.978 (0.963–0.993)	—	—	
	Clinical and Troponin with D-dimer	0.978 (0.963–0.993)	0.000	1.000	0.211

Notes: Clinical covariates were defined as age, sex, and cardiovascular comorbidities (diabetes mellitus, hypertension, ischemic heart disease, heart failure, and dyslipidemia).
Abbreviations: STEMI, ST elevation myocardial infarction; NSTEMI, Non-ST elevation myocardial infarction; AUC, Area under the curve; ΔAUC, Delta area under the curve.

infarction (STEMI) and Non-ST elevation myocardial infarction (NSTEMI), followed by unstable angina (UA) and non-specific chest pain. These findings suggest that elevated D-dimer levels are associated with myocardial infarction.

Multivariate regression analysis demonstrated that D-dimer levels remained independently associated with the final diagnosis after adjustment for demographic variables, cardiovascular comorbidities, and troponin levels. This supports an association between D-dimer and acute coronary syndromes beyond baseline clinical characteristics. The observed gradient in D-dimer levels between myocardial infarction and unstable angina may reflect increased thrombotic activity and fibrin turnover; however, given the observational design, these findings should be considered hypothesis-generating.

Despite statistically significant differences between diagnostic groups, D-dimer demonstrated only modest diagnostic performance on ROC analysis. Substantial overlap in D-dimer values across groups further limits its utility as a standalone diagnostic tool. In addition, D-dimer did not provide incremental diagnostic value beyond clinical variables and cardiac troponin, as inclusion in multivariable models did not improve discrimination. Together, these findings indicate that although D-dimer is associated with myocardial infarction, its clinical utility in diagnostic decision-making for acute coronary syndrome (ACS) is limited.

Comparison with Existing Literature

Our findings are consistent with previous studies demonstrating higher D-dimer levels in myocardial infarction compared with unstable angina but limited diagnostic specificity. Prior studies have reported considerable overlap in D-dimer values across diagnostic categories, reducing its usefulness as a standalone diagnostic marker.[6–8]

Shitrit et al similarly concluded that D-dimer is insufficient for distinguishing acute coronary syndromes.[6] Likewise, Reihani et al reported elevated D-dimer levels in myocardial infarction compared with unstable angina but did not support its diagnostic use due to low specificity.[7] Our findings extend this evidence by demonstrating that D-dimer does not provide incremental diagnostic value beyond cardiac troponin, further limiting its clinical applicability.

Study Contribution and Novelty

This study contributes to the existing literature by evaluating D-dimer levels across the full spectrum of emergency department chest pain presentations, including STEMI, NSTEMI, unstable angina, and non-cardiac chest pain, rather than comparing myocardial infarction with healthy controls.

In addition to assessing the association between D-dimer and myocardial infarction, we evaluated its diagnostic performance and incremental value alongside established clinical and troponin-based models. Our results demonstrate limited discriminatory ability and no meaningful improvement in diagnostic accuracy with the addition of D-dimer.

Furthermore, this study reflects a population from an emergency department setting in the Middle East, a region that is underrepresented in the current literature. These findings provide additional evidence supporting a limited and complementary role for D-dimer in this clinical context.

Clinical Implications and Limitations of D-Dimer Testing

Our findings suggest that while D-dimer levels are elevated in patients with myocardial infarction, their diagnostic utility in clinical practice is limited. No significant diagnostic value was demonstrated in unstable angina, and D-dimer did not improve diagnostic performance when combined with clinical assessment and cardiac troponin.

Current diagnostic strategies for suspected ACS rely primarily on clinical evaluation, electrocardiography, and cardiac troponin testing.[4] In this context, D-dimer appears to have a complementary rather than a primary diagnostic role.

The limited clinical utility of D-dimer is partly attributable to its low specificity, as elevated levels may be observed in a wide range of conditions, including advanced age, recent surgery, malignancy, and inflammatory states. These factors may confound interpretation and reduce its ability to distinguish between myocardial infarction and unstable angina. Additionally, systemic inflammation, an important component of ACS pathophysiology, may contribute to elevated D-dimer levels, reflecting broader systemic processes beyond coronary thrombosis alone.[5,9]

Pathophysiological Explanation

Higher D-dimer levels in myocardial infarction compared with unstable angina may be explained by differences in underlying pathophysiology. Myocardial infarction is characterized by the formation of a sustained, fibrin-rich intracoronary thrombus following plaque rupture, leading to marked activation of the coagulation cascade and subsequent fibrinolysis.[10,11]

This process, along with myocardial necrosis and the associated inflammatory response, results in increased fibrin turnover and elevated circulating D-dimer levels.[5,10] In contrast, unstable angina is typically associated with smaller, platelet-rich thrombi and minimal myocardial necrosis, resulting in less pronounced activation of coagulation and fibrinolytic pathways and consequently lower D-dimer levels.[10]

Limitations

This study has several limitations. First, as a single-center retrospective study, the findings may have limited generalizability to other populations and healthcare settings. Second, inclusion was restricted to patients in whom D-dimer was measured, which may introduce selection bias, as testing was performed at the discretion of the treating physician.

Although patients with overt infection were excluded, residual confounding from subclinical inflammatory or prothrombotic conditions cannot be excluded and may have influenced D-dimer levels. In addition, the non-normal distribution and variability of D-dimer values may have affected the performance of parametric statistical analyses, although additional analyses were conducted to ensure robustness.

Given the observational design, causal relationships cannot be established. Furthermore, this study was not designed as a formal diagnostic accuracy study, and the findings should be interpreted accordingly. Finally, the relatively small sample size underscores the need for larger, prospective, multicenter studies to further clarify the role of D-dimer in patients presenting with suspected ACS.

Conclusion

D-dimer levels were significantly higher in patients with myocardial infarction compared with those with unstable angina and non-specific chest pain. However, D-dimer demonstrated limited diagnostic performance and did not provide incremental diagnostic value beyond clinical assessment and cardiac troponin, despite showing an independent association with the diagnosis.

These findings suggest that D-dimer should not be used as a standalone diagnostic tool but may have a complementary role within the overall clinical assessment. Cardiac troponin remains the cornerstone of current diagnostic strategies for acute coronary syndrome. Further prospective studies are needed to clarify the potential role of D-dimer in risk stratification and multimarker approaches in patients with suspected ACS.

Funding

There is no funding to report.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Virani SS, Alonso A, Aparicio HJ, et al. Heart Disease and Stroke Statistics—2021 update: a report from the American Heart Association. *Circulation*. 2021;143(8). doi:10.1161/cir.0000000000000950
2. Collet JP, Thiele H, Barbato E, et al. 2020 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. *Eur Heart J*. 2021;42(14):1289–1367. doi:10.1093/eurheartj/ehaa575
3. Ibanez B, James S, Agewall S, et al. 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J*. 2018;39(2):119–177. doi:10.1093/eurheartj/ehx393
4. Sandoval Y, Jaffe AS. High-sensitivity cardiac troponin for the diagnosis of acute myocardial infarction. *J Am Coll Cardiol*. 2019;73(5):629–643. doi:10.1016/j.jacc.2018.11.001
5. Adam SS, Key NS, Greenberg CS. D-dimer antigen: current concepts and future prospects. *Blood*. 2009;113(13):2878–2887. PMID: 19008457. doi:10.1182/blood-2008-06-165845
6. Türkoğlu C, Harbalıoğlu H, Şeker T, Baykan AO, Uysal OK. D-dimers are associated with coronary artery disease severity assessed using SYNTAX and SYNTAX II scores in patients with ST-elevation myocardial infarction. *Rev Port Cardiol*. 2020;39(12):687–693. doi:10.1016/j.repc.2020.08.006
7. Shitrit D, Bar-Gil Shitrit A, Rudensky B, Sulkes J, Gutterer N, Zviony D. Role of ELISA D-dimer test in patients with unstable angina pectoris presenting at the emergency department with a normal electrocardiogram. *Am J Hematol*. 2004;77(2):147–150. doi:10.1002/ajh.20167
8. Reihani H, Shamloo AS, Keshmiri A. Diagnostic value of D-dimer in acute myocardial infarction among patients with suspected acute coronary syndrome. *Cardiol Res*. 2018;9(1):17–22. doi:10.14740/cr.v9i1.620
9. Blockmans D, Maes A, Vanrenterghem Y, et al. Elevated levels of D-dimer are associated with inflammation and disease activity rather than risk of venous thromboembolism in patients with granulomatosis with polyangiitis during long-term follow-up. *Adv Med Sci*. 2020;65(1):85–91. doi:10.1016/j.advms.2019.12.007
10. Fuster V. Plaque disruption and the acute coronary syndromes of unstable angina and myocardial infarction: if the substrate is similar, why is the clinical presentation different? *J Am Coll Cardiol*. 1992;19(7):1421–1422. doi:10.1016/0735-1097(92)90632-W
11. Mody R, Dash D, Mody B, Dash U, Gupta R. Approach to large thrombus burden in ST-elevation myocardial infarction. *Front Cardiovasc Med*. 2026;12. doi:10.3389/fcvm.2025.1610917