Original Article

# Analytical Study of Admission Inflammatory Markers and In-Hospital Events among Acute ST-Elevation Myocardial Infarction Patients

**Ved Prakash Verma[1], Ajay Chaurasia[2], S.S Mohanty[1], Satish Suryavanshi[1]**

[1]Senior Consultant, Interventional Cardiologist, SMC Heart Institute, VIP Estate, Raipur, Chhattisgarh, India. [2]Senior Consultant, Cardio Thoracic & Vascular Surgeon, SMC Heart Institute, VIP Estate, Raipur, Chhattisgarh, India

## Abstract

**Background:** ST-elevation myocardial infarction has high early complication risk, especially in patients presenting late or with haemodynamic instability. Admission inflammatory markers may help in simple risk stratification at medium-facility hospitals. **Material and Methods:** This retrospective observational study included 148 adult patients admitted with ST-elevation myocardial infarction. Admission high-sensitivity C-reactive protein, complete blood count, neutrophil–lymphocyte ratio, renal function, blood sugar, troponin I and echocardiographic left ventricular ejection fraction were recorded. Patients were followed during hospital stay for adverse cardiovascular events and mortality. Receiver operating characteristic curve analysis and logistic regression were used to assess predictors. **Results:** Adverse cardiovascular events occurred in 37 patients (25.0%) and in-hospital mortality occurred in 12 patients (8.1%). hs-CRP was higher in patients with adverse events than those without events [11.6 (5.9–23.4) vs 4.8 (2.1–9.7) mg/L, p<0.001]. hs-CRP was also higher in patients who died [18.9 (10.8–36.5) vs 5.8 (2.6–11.4) mg/L, p<0.001]. hs-CRP ≥6.0 mg/L independently predicted adverse cardiovascular events and hs-CRP ≥10.0 mg/L independently predicted in-hospital mortality. **Conclusion:** Admission hs-CRP was a useful inflammatory marker for early risk assessment in STEMI. Neutrophil–lymphocyte ratio showed supportive value but did not remain an independent predictor after adjustment. Bedside clinical severity, failed reperfusion and hs-CRP together may help in identifying high-risk patients. This approach is practical for Indian medium-facility hospitals.

**Keywords:** ST-elevation myocardial infarction; high-sensitivity C-reactive protein; neutrophil–lymphocyte ratio; adverse cardiovascular events.

**Received: 10 April 2026** **Revised: 30 April 2026** **Accepted: 16 May 2026** **Published: 21 May 2026**

## Introduction

Ischaemic heart disease remains a major cause of death in India. ST-elevation myocardial infarction is one of its most acute and high-risk presentations. Early diagnosis, early risk stratification and timely reperfusion remain the main parts of STEMI care.[1,2]

STEMI is diagnosed using clinical symptoms, electrocardiographic ST elevation and evidence of myocardial injury. In-hospital outcome is affected by age, Killip class, left ventricular function, renal function, hyperglycaemia and delay in treatment. These variables are commonly available during admission and are useful for bedside risk assessment.[3,4]

Inflammation has an important role in plaque rupture, coronary thrombosis and myocardial injury. It may also contribute to microvascular dysfunction, no-reflow and ventricular dysfunction after infarction. Recent evidence on anti-inflammatory treatment after myocardial infarction also supports the clinical relevance of inflammation in post-infarct outcome.[5] High-sensitivity C-reactive protein is a simple inflammatory marker. It is available in many hospital laboratories and can be measured at admission. Neutrophil–lymphocyte ratio is still simpler, as it is calculated from the routine complete blood count. Both markers have shown prognostic value in acute myocardial infarction, but Indian medium-facility data are still limited.[6,7] The present study was planned to assess admission hs-CRP and neutrophil–lymphocyte ratio as predictors of in-hospital adverse cardiovascular events and mortality in STEMI patients managed at a medium-facility Indian teaching hospital.

## Materials and Methods

**Study design and setting:** This was a single-centre retrospective observational study conducted in the emergency department and intensive cardiac care unit of a tertiary care hospital in Raipur, Chhattisgarh. Consent of the patients were taken prior to the study & obtaining data. Consecutive patients admitted with ST-elevation myocardial infarction were included during the study period.

**Study population:** Adult patients diagnosed with ST-elevation

**Address for correspondence:** Dr. Satish Suryavanshi, Senior Consultant, Interventional Cardiologist, SMC Heart Institute, VIP Estate, Raipur, Chhattisgarh, India.
E-mail: drsatish_suryavanshi@yahoo.in

DOI:
10.21276/amit.2026.v13.i2.682

**How to cite this article:** Verma VP, Chaurasia A, Mohanty SS, Suryavanshi S. Analytical Study of Admission Inflammatory Markers and In-Hospital Events Among Acute ST-Elevation Myocardial Infarction Patients. Acta Med Int. 2026;13(2):245-249.

myocardial infarction within 24 hours of chest pain onset were included. Diagnosis was based on typical chest pain, ST-segment elevation on electrocardiogram and raised cardiac biomarker. Patients with active infection, sepsis, chronic inflammatory disease, malignancy, advanced chronic kidney disease, chronic liver disease, recent surgery or prior thrombolysis before admission were excluded.

**Data collection:** Clinical data were recorded at admission. Age, sex, body mass index, diabetes mellitus, hypertension, smoking status, time from chest pain onset to hospital arrival, infarct site, Killip class and blood pressure were noted. Admission blood samples were taken before reperfusion therapy wherever possible. Complete blood count, serum creatinine, random blood sugar, troponin I and hs-CRP were measured. Neutrophil–lymphocyte ratio was calculated from differential leucocyte count. Two-dimensional echocardiography was done after initial stabilisation.

**Treatment profile:** Reperfusion therapy was given as per facility protocol and cardiology decision. Thrombolysis was used when primary PCI was not immediately available. Successful thrombolysis was assessed clinically and by electrocardiographic ST-segment resolution. Patients with failed thrombolysis or high-risk features were referred for rescue PCI where feasible. Standard medical treatment was given according to clinical condition.

**Outcome measures:** The primary outcome was in-hospital adverse cardiovascular events. It included cardiac death, acute heart failure, cardiogenic shock, sustained ventricular tachycardia or ventricular fibrillation and reinfarction. The secondary outcome was in-hospital mortality. Patients were followed until discharge or death.

**Statistical analysis:** Data were analysed using standard statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Categorical variables were expressed as number and percentage. Student's t-test or Mann–Whitney U test was used for continuous variables. Chi-square test or Fisher's exact test was used for categorical variables. Receiver operating characteristic curve analysis was used to identify cut-off values for hs-CRP and neutrophil–lymphocyte ratio. Logistic regression was used to assess predictors of adverse cardiovascular events and mortality. A p value <0.05 was considered statistically significant.

## Results

Adverse cardiovascular events occurred in 37 of 148 patients (25.0%). Patients with adverse events were older than those without events (61.4 ± 10.6 vs 55.2 ± 11.4 years, p=0.004). Diabetes mellitus was more frequent in the adverse event group (45.9% vs 28.8%, p=0.050). Chest pain to hospital time was longer in patients with adverse events (8.9 ± 5.2 vs 6.7 ± 4.3 hours, p=0.012). Killip class ≥II was higher in the adverse event group (48.6% vs 17.1%, p<0.001). Systolic blood pressure was lower (112.3 ± 24.6 vs 126.8 ± 22.2 mmHg, p=0.001). Serum creatinine, random blood sugar, leucocyte count, neutrophil–lymphocyte ratio and hs-CRP were also significantly higher in patients with adverse events. LVEF <40% was more common in this group (51.4% vs 21.6%, p<0.001). Successful thrombolysis was lower among patients with adverse events (45.5% vs 75.6%, p=0.006).

**Table 1: Clinical and laboratory profile according to adverse cardiovascular events**

| Parameter | Adverse events present n=37 | Adverse events absent n=111 | p value |
|---|---|---|---|
| Age in years | 61.4 ± 10.6 | 55.2 ± 11.4 | 0.004* |
| Male sex | 25 (67.6%) | 83 (74.8%) | 0.384 |
| BMI kg/m² | 25.8 ± 3.7 | 24.9 ± 3.4 | 0.173 |
| Diabetes mellitus | 17 (45.9%) | 32 (28.8%) | 0.050* |
| Hypertension | 20 (54.1%) | 48 (43.2%) | 0.254 |
| Current smoking | 18 (48.6%) | 62 (55.9%) | 0.445 |
| Chest pain to hospital time in hours | 8.9 ± 5.2 | 6.7 ± 4.3 | 0.012* |
| Anterior wall STEMI | 24 (64.9%) | 55 (49.5%) | 0.105 |
| Killip class ≥II at admission | 18 (48.6%) | 19 (17.1%) | <0.001* |
| Systolic BP mmHg | 112.3 ± 24.6 | 126.8 ± 22.2 | 0.001* |
| Serum creatinine mg/dL | 1.42 ± 0.55 | 1.12 ± 0.33 | <0.001* |
| Random blood sugar mg/dL | 231.4 ± 91.2 | 178.6 ± 72.5 | 0.001* |
| Leucocyte count ×10³/mm³ | 13.8 ± 4.4 | 11.5 ± 3.2 | 0.001* |
| Neutrophil–lymphocyte ratio | 6.9 (4.6–9.8) | 4.3 (2.8–6.1) | <0.001* |
| hs-CRP mg/L | 11.6 (5.9–23.4) | 4.8 (2.1–9.7) | <0.001* |
| Troponin I ng/mL | 7.9 (2.6–18.5) | 4.6 (1.5–11.2) | 0.047* |
| LVEF <40% | 19 (51.4%) | 24 (21.6%) | <0.001* |
| Thrombolysis given | 22 (59.5%) | 82 (73.9%) | 0.092 |
| Successful thrombolysis | 10/22 (45.5%) | 62/82 (75.6%) | 0.006* |
| Primary PCI | 6 (16.2%) | 13 (11.7%) | 0.474 |
| Referral for PCI | 16 (43.2%) | 31 (27.9%) | 0.084 |

**Table 2: Clinical and laboratory profile according to in-hospital mortality**

| Parameter | Mortality present n=12 | Mortality absent n=136 | p value |
|---|---|---|---|
| Age in years | 64.6 ± 8.8 | 56.4 ± 11.5 | 0.018* |
| Male sex | 8 (66.7%) | 100 (73.5%) | 0.720 |
| Diabetes mellitus | 6 (50.0%) | 43 (31.6%) | 0.200 |
| Hypertension | 7 (58.3%) | 61 (44.9%) | 0.370 |
| Delayed presentation >6 hours | 9 (75.0%) | 59 (43.4%) | 0.040* |

| | | | |
|---|---|---|---|
| Anterior wall STEMI | 8 (66.7%) | 71 (52.2%) | 0.340 |
| Killip class ≥II at admission | 9 (75.0%) | 28 (20.6%) | <0.001* |
| Systolic BP mmHg | 101.7 ± 22.4 | 125.3 ± 22.9 | 0.001* |
| Serum creatinine mg/dL | 1.61 ± 0.63 | 1.16 ± 0.37 | 0.003* |
| Random blood sugar mg/dL | 254.2 ± 96.3 | 186.5 ± 76.1 | 0.006* |
| Leucocyte count ×10³/mm³ | 14.6 ± 4.9 | 11.9 ± 3.5 | 0.018* |
| Neutrophil–lymphocyte ratio | 8.4 (5.9–12.6) | 4.7 (3.0–6.9) | 0.002* |
| hs-CRP mg/L | 18.9 (10.8–36.5) | 5.8 (2.6–11.4) | <0.001* |
| Troponin I ng/mL | 10.8 (4.1–25.4) | 5.0 (1.7–12.7) | 0.048* |
| LVEF <40% | 8 (66.7%) | 35 (25.7%) | 0.006* |
| Successful thrombolysis | 3/8 (37.5%) | 69/96 (71.9%) | 0.053 |

In-hospital mortality occurred in 12 patients (8.1%). Patients who died were older than survivors (64.6 ± 8.8 vs 56.4 ± 11.5 years, p=0.018). Delayed presentation >6 hours was more frequent in the mortality group (75.0% vs 43.4%, p=0.040). Killip class ≥II was also higher among patients who died (75.0% vs 20.6%, p<0.001). Systolic blood pressure was lower in the mortality group (101.7 ± 22.4 vs 125.3 ± 22.9 mmHg, p=0.001). Serum creatinine, random blood sugar, leucocyte count, neutrophil–lymphocyte ratio, hs-CRP and troponin I were significantly higher among patients who died. LVEF <40% was more frequent in the mortality group (66.7% vs 25.7%, p=0.006).

**Table 3: ROC analysis for prediction of adverse events and mortality**

| Markers | Outcome predicted | AUC | Cut-off | Sensitivity | Specificity | p value |
|---|---|---|---|---|---|---|
| hs-CRP | Adverse cardiovascular events | 0.742 | ≥6.0 mg/L | 75.7% | 64.9% | <0.001* |
| Neutrophil–lymphocyte ratio | Adverse cardiovascular events | 0.706 | ≥5.2 | 67.6% | 67.6% | <0.001* |
| Leucocyte count | Adverse cardiovascular events | 0.638 | ≥12.8 ×10³/mm³ | 59.5% | 60.4% | 0.012* |
| Random blood sugar | Adverse cardiovascular events | 0.665 | ≥190 mg/dL | 64.9% | 62.2% | 0.004* |
| hs-CRP | In-hospital mortality | 0.829 | ≥10.0 mg/L | 83.3% | 72.1% | <0.001* |
| Neutrophil–lymphocyte ratio | In-hospital mortality | 0.769 | ≥6.4 | 75.0% | 70.6% | 0.002* |
| Leucocyte count | In-hospital mortality | 0.650 | ≥13.5 ×10³/mm³ | 58.3% | 64.7% | 0.081 |
| Serum creatinine | In-hospital mortality | 0.733 | ≥1.4 mg/dL | 66.7% | 78.7% | 0.008* |

hs-CRP showed good discrimination for adverse cardiovascular events with AUC 0.742 (95% CI 0.652–0.833, p<0.001). At a cut-off of ≥6.0 mg/L, sensitivity was 75.7% and specificity was 64.9%. Neutrophil–lymphocyte ratio also predicted adverse events with AUC 0.706 (95% CI 0.610–0.802, p<0.001). For in-hospital mortality, hs-CRP showed the highest AUC of 0.829 (95% CI 0.721–0.937, p<0.001). At a cut-off of ≥10.0 mg/L, sensitivity was 83.3% and specificity was 72.1%. Neutrophil–lymphocyte ratio showed fair discrimination for mortality with AUC 0.769 (95% CI 0.632–0.906, p=0.002). Leucocyte count did not show significant prediction for mortality.

**Table 4: Predictors of adverse cardiovascular events**

| Variable | Unadjusted OR | 95% CI | p value | Adjusted OR | 95% CI | p value |
|---|---|---|---|---|---|---|
| Age ≥60 years | 2.29 | 1.08–4.87 | 0.031* | 1.72 | 0.70–4.24 | 0.238 |
| Delayed presentation >6 hours | 2.08 | 0.97–4.45 | 0.060 | 1.63 | 0.66–4.01 | 0.289 |
| Killip class ≥II | 4.58 | 2.02–10.38 | <0.001* | 3.02 | 1.15–7.95 | 0.025* |
| LVEF <40% | 3.82 | 1.73–8.45 | 0.001* | 2.47 | 0.97–6.28 | 0.058 |
| hs-CRP ≥6.0 mg/L | 4.11 | 1.87–9.03 | <0.001* | 2.83 | 1.15–6.97 | 0.024* |
| Neutrophil–lymphocyte ratio ≥5.2 | 3.06 | 1.42–6.59 | 0.004* | 2.09 | 0.86–5.09 | 0.104 |
| Failed reperfusion after thrombolysis | 3.87 | 1.49–10.05 | 0.005* | 2.74 | 1.02–7.36 | 0.046* |

On unadjusted analysis, age ≥60 years, Killip class ≥II, LVEF <40%, hs-CRP ≥6.0 mg/L, neutrophil–lymphocyte ratio ≥5.2 and failed reperfusion after thrombolysis were associated with adverse cardiovascular events. After adjustment, Killip class ≥II remained an independent predictor (adjusted OR 3.02, 95% CI 1.15–7.95, p=0.025). hs-CRP ≥6.0 mg/L independently predicted adverse cardiovascular events (adjusted OR 2.83, 95% CI 1.15–6.97, p=0.024). Failed reperfusion after thrombolysis also remained significant (adjusted OR 2.74, 95% CI 1.02–7.36, p=0.046).

**Table 5: Predictors of in-hospital mortality**

| Variable | Unadjusted OR | 95% CI | p value | Adjusted OR | 95% CI | p value |
|---|---|---|---|---|---|---|
| Age ≥60 years | 3.45 | 1.00–11.89 | 0.050* | 2.15 | 0.51–9.08 | 0.296 |
| Killip class ≥II | 12.62 | 3.11–51.24 | <0.001* | 5.94 | 1.24–28.41 | 0.026* |
| Serum creatinine ≥1.4 mg/dL | 5.12 | 1.45–18.05 | 0.011* | 3.48 | 0.82–14.80 | 0.089 |
| LVEF <40% | 5.78 | 1.63–20.55 | 0.007* | 2.41 | 0.52–11.16 | 0.262 |
| hs-CRP ≥10.0 mg/L | 10.18 | 2.55–40.61 | 0.001* | 6.21 | 1.31–29.42 | 0.021* |
| Neutrophil–lymphocyte ratio ≥6.4 | 5.88 | 1.57–22.03 | 0.008* | 2.72 | 0.59–12.48 | 0.198 |
| Failed reperfusion after thrombolysis | 4.82 | 1.28–18.11 | 0.020* | 2.96 | 0.64–13.75 | 0.166 |

On unadjusted analysis, age ≥60 years, Killip class ≥II, serum creatinine ≥1.4 mg/dL, LVEF <40%, hs-CRP ≥10.0 mg/L, neutrophil–lymphocyte ratio ≥6.4 and failed reperfusion after thrombolysis were associated with in-hospital mortality. After adjustment, Killip class ≥II remained an independent predictor of mortality (adjusted OR 5.94, 95% CI 1.24–28.41, p=0.026). hs-CRP ≥10.0 mg/L was the strongest independent inflammatory predictor of mortality (adjusted OR 6.21, 95% CI 1.31–29.42, p=0.021). Other variables lost statistical significance after adjustment.

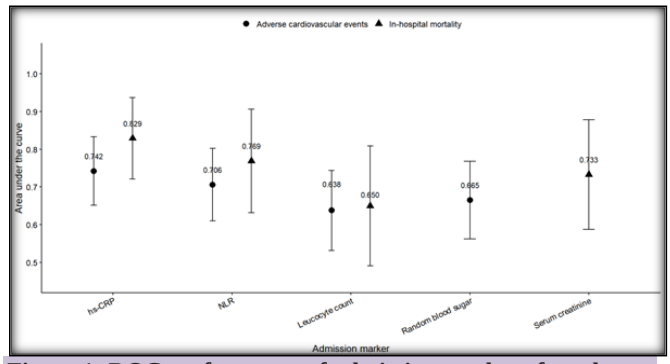


**Figure 1: ROC performance of admission markers for adverse events and mortality**

## Discussion

In the present study, adverse cardiovascular events occurred in 37 patients (25.0%) and in-hospital mortality occurred in 12 patients (8.1%). Admission hs-CRP was higher in patients with adverse events and also in patients who died during hospital stay. hs-CRP showed good discrimination for adverse events with AUC 0.742 and stronger discrimination for mortality with AUC 0.829. On multivariable analysis, hs-CRP ≥6.0 mg/L independently predicted adverse cardiovascular events and hs-CRP ≥10.0 mg/L independently predicted in-hospital mortality.

These findings suggest that admission hs-CRP is not only an inflammatory value but also a clinical risk marker in STEMI. The association remained significant after adjustment for clinical severity variables. This is important because hs-CRP can be measured easily in many Indian hospitals. It may help to identify patients who need closer monitoring in coronary care unit, especially when PCI facility is not available round the clock. The findings are in agreement with Hartopo et al., who reported that admission hs-CRP independently predicted in-hospital adverse cardiovascular events and mortality in STEMI patients. Their study used different cut-off values, but the direction of association was similar.[8] The higher cut-off in the present study may be related to delayed presentation, higher thrombolysis use and medium-facility referral pattern.

Lucci et al. also found that admission hs-CRP had prognostic value in acute myocardial infarction patients with and without diabetes mellitus.[9] In the present study, diabetes and high random blood sugar were more common in the adverse event group. This supports the view that inflammatory and metabolic stress may act together in acute infarction. A recent meta-analysis also showed that higher CRP after acute myocardial infarction was associated with all-cause mortality, cardiovascular death and major adverse cardiovascular events.[10] The present study showed a similar pattern. But the specificity of hs-CRP was moderate. So hs-CRP should not be used alone. It is better used along with Killip class, blood pressure, renal function, LVEF and reperfusion status.

Neutrophil–lymphocyte ratio was also higher in patients with adverse events and mortality. It showed fair ROC performance. But it did not remain an independent predictor after multivariable adjustment. Sharma et al. from Northeast India reported that NLR was useful as a preliminary low-cost prognostic marker in STEMI.[11] The difference may be due to sample size, outcome definition and inclusion of hs-CRP and Killip class in the same model. NLR may still be useful when hs-CRP report is delayed. Killip class ≥II was an independent predictor of both adverse events and mortality. This is clinically expected. Patients with heart failure signs at admission already have larger haemodynamic burden. In the present study, Killip class ≥II increased the adjusted odds of adverse events by 3.02 times and mortality by 5.94 times. This finding supports continued use of simple bedside clinical grading in STEMI.

Failed reperfusion after thrombolysis independently predicted adverse cardiovascular events. This has practical importance in Indian medium-facility hospitals where primary PCI may not be immediately available. The TRUST registry from Kerala showed that delayed reperfusion and delayed fibrinolysis were linked with worse outcomes in STEMI.[12] This supports early recognition of failed thrombolysis and timely referral for rescue PCI. The in-hospital mortality of 8.1% in the present study is close to major Indian ACS data. CREATE registry reported 30-day mortality of 8.6% in STEMI. Kerala ACS Registry reported higher in-hospital mortality and adverse events among STEMI patients compared with other ACS groups.[13,14] The adverse event rate in the present study was 25.0%, which is higher than some registry MACE values. This may be because the present study included acute heart failure, cardiogenic shock, arrhythmia and mortality in the event definition.

Indian STEMI care is affected by delay in presentation, referral pathway, availability of catheterisation laboratory and affordability. The Tamil Nadu-STEMI programme showed that organised hub-and-spoke STEMI care can improve reperfusion delivery. ACS QUIK showed that quality improvement can improve process measures, though major outcome reduction was not clearly shown. The CSI position statement also emphasises practical protocols suited to Indian settings.[15-17]

In thrombolysed patients, angiographic success remains variable even with guideline-directed therapy. Durdana et al. reported variable TIMI 3 flow after fibrinolysis in Indian STEMI patients.[18] This supports the present finding that failed reperfusion is a clinically important risk marker. hs-CRP may add further risk information in such patients but it cannot replace ECG-based reperfusion assessment. The admission hs-CRP was the strongest inflammatory marker in this study. NLR had supportive value but was not independent after adjustment. The findings support a practical risk approach where hs-CRP is used as an adjunct to clinical severity, LVEF, renal function and reperfusion response. This may be useful in medium-facility

Indian hospitals where early triage decisions are often made before advanced cardiac support is available.

## Conclusion

Admission hs-CRP was higher in STEMI patients who developed adverse cardiovascular events and in those who died during hospital stay. hs-CRP ≥6.0 mg/L predicted adverse cardiovascular events and hs-CRP ≥10.0 mg/L predicted in-hospital mortality after adjustment. Neutrophil–lymphocyte ratio was useful as a simple screening marker but its independent value was weaker. hs-CRP can be used as an adjunct to Killip class, reperfusion response and left ventricular function for early risk assessment in medium-facility Indian hospitals.


## Financial support and sponsorship

Nil.


## Conflicts of interest

There are no conflicts of interest.

## References


1. India State-Level Disease Burden Initiative CVD Collaborators. The changing patterns of cardiovascular diseases and their risk factors in the states of India: The Global Burden of Disease Study 1990–2016. Lancet Glob Health. 2018;6(12):e1339-e1351. doi:10.1016/S2214-109X(18)30407-8.
2. Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2023;44(38):3720-3826. doi:10.1093/eurheartj/ehad191.
3. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction. Eur Heart J. 2019;40(3):237-269. doi:10.1093/eurheartj/ehy462.
4. Bergmark BA, Mathenge N, Merlini PA, et al. Acute coronary syndromes. Lancet. 2022;399(10332):1347-1358. doi:10.1016/S0140-6736(21)02391-6.
5. Tardif JC, Kouz S, Waters DD, et al. Efficacy and safety of low-dose colchicine after myocardial infarction. N Engl J Med. 2019;381(26):2497-2505. doi:10.1056/NEJMoa1912388.
6. Zhang X, Wang S, Fang S, Yu B. Prognostic role of high sensitivity C-reactive protein in patients with acute myocardial infarction. Front Cardiovasc Med. 2021;8:659446. doi:10.3389/fcvm.2021.659446.
7. Mikhael R, Hindoro E, Taner S, Lukito AA. Neutrophil-to-lymphocyte ratio for predictor of in-hospital mortality in ST-segment elevation myocardial infarction: A meta-analysis. Med J Indones. 2020;29(2):172-182. doi:10.13181/mji.oa.202795.
8. Hartopo AB, Sukmasari I, Inggriani MP, et al. On-admission serum high-sensitivity C-reactive protein level independently predicts in-hospital mortality and adverse cardiovascular events in ST-elevation acute myocardial infarction. Indian J Clin Cardiol. 2022;3(1):16-23. doi:10.1177/26324636211055370.
9. Lucci C, Cosentino N, Genovese S, et al. Prognostic impact of admission high-sensitivity C-reactive protein in acute myocardial infarction patients with and without diabetes mellitus. Cardiovasc Diabetol. 2020;19(1):183. doi:10.1186/s12933-020-01157-7.
10. Liu S, Hu H, Yin C, et al. Evaluation of C-reactive protein as predictor of adverse prognosis in acute myocardial infarction after percutaneous coronary intervention: A systematic review and meta-analysis from 18,715 individuals. Front Cardiovasc Med. 2022;9:1013501. doi:10.3389/fcvm.2022.1013501.
11. Sharma DJ Sr, Thongam K, Basumatary LJ, et al. Neutrophil-to-lymphocyte ratio useful as a cost-effective preliminary prognostic marker in ST-elevation myocardial infarction: An observational study from a tertiary care hospital in Northeast India. Cureus. 2023;15(3):e36885. doi:10.7759/cureus.36885.
12. Mathew A, Moolla M, Jeemon P, et al. Timeliness of reperfusion in ST-segment elevation myocardial infarction and outcomes in Kerala, India: Results of the TRUST outcomes registry. Postgrad Med J. 2025;101(1193):232-239. doi:10.1093/postmj/qgae129.
13. Xavier D, Pais P, Devereaux PJ, et al. Treatment and outcomes of acute coronary syndromes in India: CREATE registry. Lancet. 2008;371(9622):1435-1442. doi:10.1016/S0140-6736(08)60623-6.
14. Mohanan PP, Mathew R, Harikrishnan S, et al. Presentation, management and outcomes of 25,748 acute coronary syndrome admissions in Kerala, India: Results from the Kerala ACS Registry. Eur Heart J. 2013;34(2):121-129. doi:10.1093/eurheartj/ehs219.
15. Alexander T, Mullasari AS, Joseph G, et al. A system of care for patients with ST-segment elevation myocardial infarction in India: The Tamil Nadu-STEMI Program. JAMA Cardiol. 2017;2(5):498-505. doi:10.1001/jamacardio.2016.5977.
16. Huffman MD, Mohanan PP, Devarajan R, et al. Effect of a quality improvement intervention on clinical outcomes in patients in India with acute myocardial infarction: The ACS QUIK randomized clinical trial. JAMA. 2018;319(6):567-578. doi:10.1001/jama.2017.21906.
17. Guha S, Sethi R, Ray S, et al. Cardiological Society of India: Position statement for the management of ST elevation myocardial infarction in India. Indian Heart J. 2017;69(Suppl 1):S63-S97. doi:10.1016/j.ihj.2017.03.006.
18. Durdana S, Malik MA, Hasan A, Rabbani MU. Angiographic outcomes in STEMI patients receiving fibrinolysis with guideline directed optimal antithrombotic therapy. Indian Heart J. 2021;73(1):125-128. doi:10.1016/j.ihj.2020.11.011.