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# Predictors of in-hospital mortality in high Killip class acute ST-segment elevation myocardial infarction after reperfusion

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## Abstract:

**Objective**: This study aimed to identify the predictors of in-hospital mortality in acute ST-segment Elevation Myocardial Infarction (STEMI) patients presenting with high Killip class following reperfusion.

**Material and Methods:** A retrospective cohort study was conducted at Surat Thani Hospital from October 1, 2020, to September 30, 2022. Acute STEMI patients who received percutaneous coronary intervention (PCI) were enrolled. The primary outcome was in-hospital mortality.

**Results:** A total of 234 patients classified as high Killip class (III or IV) were enrolled in this cohort study. The in-hospital mortality rate was 18.8%. Independent predictors associated with in-hospital mortality were hemoglobin level <12 g/dL (OR=5.57; 95% CI=1.92–16.17, p-value=0.002), mean arterial pressure <65 mmHg (OR=5.80; 95% CI=1.93–17.43, p-value=0.002), receiving more than two vasopressors (OR=7.19; 95% CI=2.49–20.77, p-value<0.001), and multivessel PCI (OR=14.65; 95% CI=2.84–75.67, p-value=0.001). Intubation as a supportive intervention prior to reperfusion therapy was associated with decreased in-hospital mortality (OR=0.27; 95% CI=0.10–0.73, p-value<0.010).

**Conclusion:** STEMI patients with a high Killip class receiving reperfusion therapy exhibited high in-hospital mortality. Predictive factors included low hemoglobin, low mean arterial pressure, multiple vasopressors, and multivessel PCI. Early intubation is associated with improved clinical outcomes.

**Keywords:** cardiogenic shock, in-hospital mortality, reperfusion therapy, STEMI

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## Introduction

Cardiovascular diseases (CVDs) are a major global health challenge, especially in low- and middle-income nations, with a mortality rate of 17.9 million deaths annually[1]. Acute coronary syndrome (ACS) is a broad spectrum of disease depending on its clinical manifestation, electrocardiographic (ECG) findings, and elevation of cardiac enzymes, especially cardiac troponin[2]. Of these, ST-segment elevation myocardial infarction (STEMI) is the most serious condition, requiring immediate management. The treatment goals of STEMI are rapid diagnosis and immediate reperfusion therapy, either by medication or primary percutaneous coronary intervention (PPCI). For patients who received medication therapy with a fibrinolytic agent but failed to restore coronary perfusion, rescue PCI could be considered to improve patient outcomes.

Thailand's national guideline framework was developed to improve health outcomes through population-based prevention programs, management of risk factors and non-communicable diseases, and acute care for conditions such as acute coronary syndrome and stroke[3]. A significant proportion of STEMI patients are unstable, with complications of congestive heart failure or cardiogenic shock. STEMI patients with congestive heart failure can be grouped by the Killip classification. The most severe forms of pulmonary edema and pulmonary edema with cardiogenic shock are classified into Killip class III and class IV, respectively. Other clinical risk assessments that could be used include the GRACE score, biomarkers, and echocardiography[4,5].

The mortality rate of patients with STEMI and Killip class I-II declined after the implementation of current clinical practice guidelines[6,7]. However, the mortality rate remains high among patients with a high Killip class. Several clinical factors, as well as the treatment strategy, may impact the in-hospital mortality of STEMI patients, including cardiogenic shock, anterior wall infarction, low left ventricular ejection fraction (LVEF), and old age[8,9]. Common comorbidities such as diabetes mellitus, chronic kidney disease, and hemodynamic instability at the time of presentation have also been associated with in-hospital mortality[10,11].

The PCI strategy, such as complete or culprit vessel-only revascularization in STEMI patients during hospitalization, the number of or preferred inotropic drugs in unstable patients, and the use of temporary mechanical assist devices (e.g., intra-aortic balloon pump (IABP), Impella, or veno-arterial extracorporeal membrane oxygenation (VA-ECMO), could be considered modifiable factors with the ability to determine patient outcomes[6,12,13]. Consequently, the objective of this study was to probe the predictors associated with in-hospital mortality among patients who presented with a high Killip class as a complication of acute STEMI.

## Material and Methods

The current study was carried out at Surat Thani Hospital. The study design was a retrospective cohort, in which patients' medical records were retrospectively reviewed from October 1, 2020, to September 30, 2022. The Ethics Committee of Surat Thani Hospital approved the research (Study Code: REC-66-0133 Certificate of Approval No. 134/2566). The inclusion criteria were patients who were admitted to the cardiac intensive care unit (CCU) and diagnosed with acute STEMI according to ICD codes I21.0-I21.3. The severity of each patient was categorized according to the Killip classification prior to cardiac catheterization, based on clinical evaluation performed in the catheterization laboratory. The Killip classification ranges from Killip class I (no heart failure), class II (heart failure with rales and/or a third heart sound), class III (pulmonary edema), and class IV (pulmonary edema and cardiogenic shock). The diagnosis of AMI and the determination of the Killip classification depended on the CCU attending physician. A specialized nurse collected the data.

All clinical data were collected prior to cardiac catheterization, including baseline characteristics, underlying diseases, presenting symptoms, initial laboratory and angiographic findings, as well as pharmacologic interventions and treatment outcomes. Patients who did not receive reperfusion therapy by either pharmacotherapy or PCI were excluded, as were patients with Killip classes I or II. The in-hospital mortality was the primary outcome of this study.

### Statistical analyses

Descriptive statistics were used to examine the baseline characteristics and clinical data. Categorical data were presented as frequency and percentage, and evaluated using Fisher's exact test. The normal distribution of continuous data was assessed using the Kolmogorov-Smirnov test, presented as the mean±standard deviation (S.D.), and analysed using the Student's t-test. The non-normal distribution was presented as the median and interquartile range (IQR), and the results were evaluated using the Mann-Whitney U test. Receiver operating characteristic (ROC) curve analysis was employed to ascertain the accuracy of continuous variable data and identify cut-off values.

The potential predictive factors associated with in-hospital mortality were identified through univariate analysis. Significant factors identified through univariate analysis (p-value<0.10) were integrated into a multivariate binary logistic regression forward stepwise model to determine the independent predictors of in-hospital mortality. Odds ratios (ORs) and 95% confidence intervals (CIs) were computed. All analyses were performed utilizing PASW Statistics, version 18 (SPSS Inc., Chicago, IL, USA), with statistical significance established by two-sided p-value<0.05.

## Results

During the study period, a total of 415 patients were diagnosed with STEMI. Of these, 330 patients (79.5%) received reperfusion therapy. Among those, 234 patients (71.0%) who were classified as Killip class III or IV were included in the final cohort for analysis (Figure 1). The baseline characteristics of the study population are summarized in Table 1. Of the enrolled patients, 190 (81.2%) survived to hospital discharge, while 44 (18.8%) died during hospitalization.

Compared to survivors, non-survivors were significantly older (67.5±14.7 vs. 61.0±11.1 years; p-value=0.008), more frequently female (36.4% vs. 21.6%; p-value=0.040), and less likely to be current smokers (27.3% vs. 43.7%; p-value=0.046). In addition, non-survivors had a higher prevalence of hypertension (68.2% vs. 50.5%; p-value=0.034) and chronic kidney disease (27.3% vs. 7.4%; p-value<0.001). The most frequent infarct location was the anterior wall (50.9%), followed by the inferior wall (36.3%) and lateral wall (12.8%). There was no statistically significant difference in the distribution of myocardial infarction location between survivors and non-survivors.

Hemodynamic and laboratory parameters at baseline also differed significantly between groups. Non-survivors had lower mean systolic blood pressure (87.9±29.6 vs. 101.2±24.2 mmHg; p-value=0.002), diastolic blood pressure (58.3±18.7 vs. 68.5±14.7 mmHg; p-value=0.001), and mean arterial pressure (68.1±21.5 vs. 79.5±16.7 mmHg; p-value=0.002). They also exhibited a significantly lower estimated glomerular filtration rate (46.4±27.5 vs. 75.3±26.3 mL/min/1.73 $m^2$; p-value<0.001) and hemoglobin concentration (11.9±3.6 vs. 14.1±7.2 g/dL; p-value=0.015).

The incidence of out-of-hospital cardiac arrest was significantly higher among non-survivors (29.5% vs. 11.6%; p-value=0.003). However, there was no statistically significant difference in the proportion of patients initially presenting to non-percutaneous coronary intervention (PCI)-capable centers between the two groups (75.0% vs. 82.6%; p-value=0.243).

**Table 1** Comparison of baseline characteristics among non-survived and survived STEMI patients after reperfusion therapy (n=234)

| Clinical parameters | All, n (%) 234 (100) | Non-survived, n (%) 44 (18.8) | Survived, n (%) 190 (81.2) | p-value |
|---|---|---|---|---|
| Age, years, mean±S.D. | 62.2±12.1 | 67.5±14.7 | 61.0±11.1 | 0.008 |
| Female | 57 (24.4) | 16 (36.4) | 41 (21.6) | 0.040 |
| Body mass index, kg/m$^2$, mean±S.D. | 24.0±3.9 | 24.6±4.6 | 23.8±3.7 | 0.269 |
| **Underlying diseases** | | | | |
| Hypecholesterolemia | 155 (66.2) | 29 (65.9) | 126 (66.3) | 0.959 |
| Hypertension | 126 (53.8) | 36 (68.2) | 96 (50.5) | 0.034 |
| Current smoking | 95 (40.6) | 12 (27.3) | 43 (43.7) | 0.046 |
| Diabetes mellitus | 63 (26.9) | 14 (31.8) | 49 (25.8) | 0.417 |
| Chronic kidney disease | 26 (11.1) | 12 (27.3) | 14 (7.4) | <0.001 |
| **Myocardial infarction location** | | | | |
| Anterior wall | 119 (50.9) | 20 (45.5) | 99 (52.1) | 0.427 |
| Inferior wall | 85 (36.3) | 17 (38.6) | 68 (35.8) | 0.723 |
| Lateral wall | 30 (12.8) | 7 (15.9) | 23 (12.1) | 0.496 |
| **Congestive heart failure Killip classification, n (%)** | | | | |
| Congestive heart failure with high Killip classification (III, IV) | 234 (100) | 44 (18.8) | 190 (81.2) | 0.001 |
| **Baseline hemodynamic parameters, mean±S.D.** | | | | |
| Heart rate, per minute | 88.5±17.0 | 96.1±23.9 | 86.7±14.5 | 0.001 |
| Systolic blood pressure, mmHg | 98.7±25.8 | 87.9±29.6 | 101.2±24.2 | 0.002 |
| Diastolic blood pressure, mmHg | 66.5±16.0 | 58.3±18.7 | 68.5±14.7 | 0.001 |
| Mean blood pressure, mmHg | 77.4±18.2 | 68.1±21.5 | 79.5±16.7 | 0.002 |
| **Baseline laboratory test, mean±S.D.** | | | | |
| Serum Na, mmol/L | 137.7±4.5 | 137.0±5.8 | 137.8±4.1 | 0.378 |
| Serum K, mmol/L | 3.9±0.6 | 4.2±0.7 | 3.8±0.6 | 0.005 |
| Left ventricular ejection fraction, % | 43.1±14.9 | 39.1±17.8 | 43.4±14.8 | 0.477 |
| Glomerular filtration rate, mL/min/1.73 m$^2$ | 69.9±28.8 | 46.4±27.5 | 75.3±26.3 | <0.001 |
| Hemoglobin, g/dL | 13.7±6.7 | 11.9±3.6 | 14.1±7.2 | 0.015 |
| Out-hospital cardiac arrest | 35 (15.0) | 13 (29.5) | 22 (11.6) | 0.003 |
| Referred from non-PCI hospital | 190 (81.2) | 33 (75.0) | 157 (82.6) | 0.243 |

Na=sodium, K=potassium, Mg=magnesium, PCI=percutaneous coronary intervention, S.D.=standard devision

Treatment strategies for STEMI patients are summarized in Table 2. There were no statistically significant differences between survivors and non-survivors in the distribution of reperfusion modalities, including PPCI, rescue PCI, and pharmaco-invasive strategy.

However, certain procedural interventions were significantly more common among non-survivors. PCI involving the left main coronary artery was performed more frequently in non-survivors than in survivors (20.5% vs. 7.9%; p-value=0.013), as was multivessel PCI (23.3% vs. 3.2%; p-value<0.001). Additionally, non-survivors required more intensive supportive therapies. A significantly greater proportion received norepinephrine (90.9% vs. 49.4%; p-value<0.001), required the use of more than two vasopressors (77.3% vs. 23.2%; p-value<0.001), underwent renal replacement therapy (15.9% vs. 4.2%; p-value=0.010), and required endotracheal intubation prior to hospital admission (54.5% vs. 28.9%; p-value=0.001).

Continuous variables that demonstrated statistically significant associations with in-hospital mortality were

dichotomized based on optimal cut-off values derived from receiver operating characteristic (ROC) curve analysis, including age>65 years, eGFR <50 mL/min/1.73m$^2$, hemoglobin <12 g/dL, serum potassium>5 meq/L, heart rate >100 per minute, mean ABP <65 mmHg, procedure time >35 minutes. Significant predictive factors for in-hospital mortality by univariate and multivariate analysis are presented in Table 3. Stepwise multivariate logistic regression analysis identified six independent variables significantly associated with in-hospital mortality. Five of these were predictors of increased mortality risk, including hemoglobin level <12 g/dL (OR=5.57; 95% CI: 1.92–16.17; p-value=0.002), mean arterial pressure <65 mmHg (OR=5.80; 95% CI: 1.93–17.43; p-value=0.002), norepinephrine administration (OR=7.49; 95% CI: 1.67–33.55; p-value=0.008), use of more than two vasopressors (OR=7.19; 95% CI: 2.49–20.77; p-value<0.001), and multivessel PCI (RR=14.65; 95% CI: 2.84–75.67; p-value=0.001). In contrast, endotracheal intubation prior to hospital admission was independently associated with improved survival (OR=0.27; 95% CI: 0.10–0.73; p-value=0.010).

**Table 2** Treatment strategy of acute myocardial infarction (n=234)

| Treatment | All, n (%) 234 (100) | Non-survived, n (%) 44 (18.8) | Survived, n (%) 190 (81.2) | p-value |
|---|---|---|---|---|
| **Reperfusion strategy** | | | | |
| Primary PCI | 152 (65.0) | 34 (77.3) | 118 (62.1) | 0.057 |
| Rescue PCI | 49 (20.9) | 7 (15.9) | 42 (22.1) | 0.363 |
| Pharmacoinvasive | 33 (14.1) | 3 (6.8) | 30 (15.8) | 0.152 |
| **Target vessel PCI**[†] | | | | |
| Left main disease | 24 (10.3) | 9 (20.5) | 15 (7.9) | 0.013 |
| Multivessel PCI | 16 (6.9) | 10 (23.3) | 6 (3.2) | <0.001 |
| PCI to LAD | 150 (64.9) | 33 (76.7) | 117 (62.2) | 0.072 |
| PCI to LCX | 13 (5.6) | 4 (9.3) | 9 (4.8) | 0.270 |
| PCI to RCA | 88 (38.1) | 18 (41.9) | 70 (37.2) | 0.573 |
| Procedure time, minutes, mean±S.D. | 35.5±18.2 | 39.3±18.0 | 34.7±18.0 | 0.129 |
| Procedure time >35 minutes | 93 (40.8) | 23 (53.5) | 70 (37.8) | 0.060 |
| ET intubation before reperfusion | 79 (33.8) | 24 (54.5) | 55 (28.9) | 0.001 |
| **Vasopressors usage** | | | | |
| Norepinephrine | 132 (56.4) | 40 (90.9) | 92 (49.4) | <0.001 |
| Dopamine | 86 (36.8) | 21 (47.7) | 65 (34.2) | 0.094 |
| Dobutamine | 12 (5.1) | 4 (9.1) | 8 (4.2) | 0.246 |
| Required ≥2 vasopressors | 78 (33.3) | 34 (77.3) | 44 (23.2) | <0.001 |
| Renal replacement therapy | 15 (6.4) | 7 (15.9) | 8 (4.2) | 0.010 |

*Data from 327 patients who underwent coronary angiograms in the same admission. [†]Data from 325 patients who underwent target vessel PCI in the same admission. PCI=percutaneous coronary intervention, LAD=left anterior descending artery, LCX=left circumflex artery, RCA=right coronary artery, ET=endotracheal tube

**Table 3** Univariate and multivariate logistic regression analyses identifying predictive factors associated with in-hospital mortality among patients suffering from acute myocardial infarction (n=234)

| Clinical parameters | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|
| | Crude ORs (95% CI) | p-value | Adjusted ORs (95% CI) | p-value |
| Age >65 years old | 1.19 (1.03-1.37) | 0.008 | NR | |
| Female gender | 1.77 (1.04-3.04) | 0.040 | NR | |
| Hypertension | 1.14 (1.01-1.29) | 0.034 | NR | |
| Chronic kidney disease | 1.57 (1.10-2.25) | <0.001 | NR | |
| Current smoking | 0.88 (0.78-0.99) | 0.046 | NR | |
| GFR <50 mL/min/1.73 $m^2$ | 1.43 (1.17-1.74) | <0.001 | NR | |
| Hemoglobin <12 g/dL | 1.36 (1.15-1.62) | <0.001 | 5.57 (1.92-16.17) | 0.002 |
| Serum potassium >5 mEg/L | 1.83 (0.95-3.50) | 0.002 | NR | |
| Heart rate >100 per minute | 1.23 (1.02-1.48) | 0.024 | NR | |
| Mean ABP <65 mmHg | 1.98 (1.37-2.86) | <0.001 | 5.80 (1.93-17.43) | 0.002 |
| Receiving norepinephrine | 1.38 (1.22-1.55) | <0.001 | 7.49 (1.67-33.55) | 0.008 |
| Receiving dopamine | 1.12 (0.97-1.28) | 0.094 | NR | |
| Required ≥2 vasopressors | 1.66 (1.36-2.03) | <0.001 | 7.19 (2.49-20.77) | <0.001 |
| Out-hospital cardiac arrest | 1.34 (1.03-1.75) | 0.003 | NR | |
| ET intubation before reperfusion | 1.25 (1.07-1.47) | 0.001 | 0.27 (0.10-0.73) | 0.010 |
| Primary PCI | 1.13 (1.01-1.27) | 0.057 | NR | |
| Multivessel PCI | 2.26 (1.20-4.26) | <0.001 | 14.65 (2.84-75.67) | 0.001 |
| PCI to LM | 1.33 (0.97-1.83) | 0.013 | NR | |
| PCI to LAD | 1.12 (0.99-1.26) | 0.072 | NR | |
| PCI time >35 minutes | 1.13 (0.99-1.30) | 0.060 | NR | |
| Required renal replacement therapy | 1.56 (0.97-2.51) | 0.010 | NR | |

GFR=glomerular filtration rate, ET=endotracheal tube, PCI=percutaneous coronary intervention, LM=left main, LAD=left anterior descending artery, ABP=arterial blood pressure, NR=not remained in the final multivariate analysis, 95% CI=95% confedence interval

## Discussion

In this study of 234 STEMI patients classified as high Killip classes (III or IV) who underwent reperfusion therapy, the observed in-hospital mortality rate was 18.8%.

Our findings are consistent with those of Vicente et al., who reported an increase in in-hospital mortality rates with higher Killip classes. In their study, mortality was 16.7% for Killip class III and 36.7% for class IV[14], whereas this study found rates of 18.8% for both groups. Several clinical and procedural factors in this study were considered to be strong independent predictors (Odds Ratio>4) associated with in-hospital mortality, including hemoglobin levels<12 g/dL, mean arterial pressure<65 mmHg, norepinephrine administration, more than two vasopressor administrations, and multivessel PCI. In contrast, endotracheal intubation before reperfusion was associated with a lower risk of in-hospital mortality. While prior studies[14] have described mortality trends across Killip classifications, data specifically addressing predictors of in-hospital mortality among high-risk patients (Killip class III-IV) remain limited. Thus, our findings contribute valuable insights to this underexplored subgroup.

Among the predictors identified, a mean arterial pressure<65 mmHg emerged as a strong independent

marker of mortality. This parameter likely reflects the presence of cardiogenic shock, and our cohort aligns with the Society for Cardiovascular Angiography and Interventions (SCAI) classification stages B to E based on clinical presentation, hemodynamic data, and biochemical markers[15]. The results of this study are also supported by Takayama et al., who found similar associations between low initial systolic blood pressure and adverse outcomes in SCAI stage D cardiogenic shock patients, including reduced left ventricular ejection fraction and TIMI flow grade[16].

Norepinephrine remains the first-line vasopressor for cardiogenic shock, as recommended by the current guidelines[7,15]. However, the need for high-dose norepinephrine or additional vasopressors is indicative of more advanced hemodynamic compromise and has been associated with increased mortality. Excessive vasopressor use may impair splanchnic perfusion and exacerbate lactic acidosis[17,18]. Therefore, the early use of mechanical circulatory support devices, such as IABP, Impella, or VA-ECMO, may reduce the reliance on vasopressors and improve outcomes. In our setting, Impella is not available, and VA-ECMO is limited to select centers, making IABP the most accessible modality. A study by Chorchana et al. identified cardiac arrest on presentation, use of multiple vasopressors, and Killip class IV as predictors of mortality in STEMI patients treated with IABP[19].

Low hemoglobin levels were another significant predictor of in-hospital mortality. This aligns with a retrospective analysis of over 1,800 patients, which identified hemoglobin ≤10 g/dL as an independent predictor of 30-day mortality[20]. A previous study demonstrated a stepwise increase in mortality risk with each 1 g/dL decrease in hemoglobin[21]. Although anemia is a known risk factor for poor outcomes in ACS, the benefit of transfusion remains controversial. Some data suggest that transfusion may paradoxically increase mortality, particularly in STEMI patients. As such, transfusion should be considered judiciously, taking into account individual clinical circumstances[22].

The association between multivessel PCI and increased mortality in the current study echoes findings from the CULPRIT-SHOCK trial, which demonstrated better outcomes when only the culprit lesion was treated in the early phase of cardiogenic shock. Although the 2017 ESC guidelines recommend complete revascularization[23], in this setting, our institutional protocol prioritizes culprit-lesion PCI in unstable patients, with deferred intervention on non-culprit vessels. This approach is based on clinical judgment and angiographic findings. While complete revascularization in STEMI patients with multivessel disease can reduce ischemic burden, simultaneous multivessel intervention during the index procedure may increase procedural risk[24]. Thus, optimal timing for staged PCI versus immediate multivessel PCI remains an area necessitating further research[25, 26].

Interestingly, this study found that endotracheal intubation prior to reperfusion was associated with improved survival, a finding supported by van Diepen et al., who reported that earlier initiation of mechanical ventilation was associated with lower 30-day mortality[27]. Their study showed that each hour of delay in mechanical ventilation after MI onset was independently associated with increased mortality. Our study's multivariate analysis indicated that, despite patients requiring endotracheal intubation and typically presenting with more severe illness, intubation prior to reperfusion was associated with reduced in-hospital mortality when controlling for other variables. This contrast result may be due to the therapeutic outcome of managing the airway rather than the severity of illness alone. Early airway management is a modifiable factor that may reduce hypoxia-related injury, optimise hemodynamic support, and improve outcomes in patients with cardiogenic shock or respiratory failure.

There are several limitations in this study. Firstly, this study was conducted at a single tertiary institution in the past. Second, the study by Eka Ginanjar collected[28] 30-day mortality and developed a model for predicting 30-day mortality using the glomerular filtration rate, suggesting that the extra data may show formidable in-hospital mortality, such as 30-day mortality. Third, information on preexisting medications—including lipid-lowering agents, postoperative renin-angiotensin-converting enzyme inhibitors, and beta-blockers—was not included due to incomplete documentation in the medical records. Additionally, because of the retrospective nature of the study, we encountered substantial missing data regarding TIMI flow grade and the use of mechanical circulatory support devices (e.g., intra-aortic balloon pump, ECMO). Therefore, these variables were excluded from the analysis to minimize the risk of bias and misinterpretation related to missing data. As a result, we decided not to include these variables in our analysis to avoid bias or misinterpretation arising from incomplete data that was not gathered for this investigation. The primary reason for this limitation is the substantial proportion of patients exhibiting hypotension and the recurrent necessity for vasopressor support, particularly norepinephrine. Thus, complete initiation of heart failure medications was not feasible during the inpatient treatment period. Consequently, information on administering these medications is more likely to be available during outpatient follow-up visits. Finally, the potential impact of the no-reflow phenomenon during PCI, a complication associated with smoking, past myocardial infarction, Killip class III–IV status, and reduced ejection fraction, was not specifically analyzed. Given its established association with worse outcomes, future studies should investigate its role in this high-risk population.

## Conclusion

This study identified key predictors of in-hospital mortality in STEMI patients with high Killip class, including anemia, hypotension, use of multiple vasopressors, and multivessel PCI. Conversely, pre-hospital endotracheal intubation was associated with improved survival. These findings highlight modifiable factors that could guide early intervention strategies to improve outcomes.

### Ethical approval

The research was approved by the Ethics Committee of Surat Thani Hospital. Study Code: REC-66-0133 Certificate of Approval number 134/2566 on 16/11/2023

### Highlights of this study

In-hospital mortality remains substantial among STEMI patients with high Killip class, despite improvements in reperfusion strategies. Key factors associated with increased in-hospital mortality include the use of multiple vasopressors and multivessel PCI. These findings suggest a potential role for mechanical circulatory support, such as the intra-aortic balloon pump (IABP), which is widely used in Thailand due to cost and team expertise. This underscores the opportunity to expand access to advanced support devices such as Impella and ECMO.

Early airway management, including timely endotracheal intubation or advanced airway interventions, appears to be a protective factor and may improve in-hospital outcomes in this high-risk population.

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