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# Association Between ICU Admission Delay After Vasopressor Initiation and In-Hospital Mortality in Patients With Septic Shock: A Retrospective Cohort Study

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## Abstract

**Background:** Delayed intensive care unit (ICU) transfer after the emergency department (ED) management of septic shock is associated with poor outcomes. However, most studies define delay from ED arrival, which may not accurately reflect the onset of hemodynamic instability requiring ICU-level care.

**Objective:** To investigate the association between the delay in ICU admission after vasopressor initiation and in-hospital mortality of patients with septic shock.

**Methods:** This retrospective cohort study included adults with septic shock treated at the Phrapokklao Hospital ED from January 2024 to December 2025. Index time was defined as vasopressor initiation in the ED, or ED arrival for patients already receiving vasopressors before transfer. The primary exposure was ICU admission delay, and the outcome was in-hospital mortality. Data were analyzed using multivariable models, with restricted cubic spline analysis to assess nonlinear associations.

**Results:** Among 317 patients with septic shock, in-hospital mortality was 29.0%. ICU admission delay was significantly associated with mortality, with risk increasing per hour of delay (adjusted odds ratio [aOR], 2.33 per hour; 95% CI, 1.73-3.15; $P < .001$). Patients delayed ≥ 2 hours had higher mortality than those admitted within 2 hours (45.9% vs 6.6%; aOR, 9.21; 95% CI, 3.96-21.42; $P < .001$). A dose-response relationship was observed across delay quartiles ($P$ for trend < .001), with consistent findings in walk-in and referred subgroups.

**Conclusions:** ICU admission delay after vasopressor initiation was independently associated with increased mortality, with risk increasing progressively over time. The 2-hour cutoff represents an operational, not a physiological, threshold. Potential residual confounding warrants cautious interpretation.

**Keywords:** Septic shock, ICU admission delay, Vasopressor, In-hospital mortality

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## Introduction

Septic shock has been reported as a significant contributor to morbidity and mortality around the world, and immediate resuscitation and intensive care support are required.[1,2] In real-world practice, a delay in transfer from the emergency department (ED) to the intensive care unit (ICU) is possible due to resource constraints, limited ICU bed availability, and ED overcrowding.[3,4] Existing evidence has reported that delayed ICU admission is linked to undesirable clinical outcomes, particularly among patients with

critical illness, including sepsis.[5-8] Earlier reports suggested that delays exceeding 6 hours increase mortality risk, while more recent evidence indicates that even shorter delays of about 3 hours may be clinically relevant.[9, 10] Despite these findings, the optimal timing of ICU admission in septic shock remains unclear. Most prior studies have defined ICU transfer delay using ED arrival time rather than physiological deterioration,[6, 9] which may not accurately reflect the onset of critical illness in septic shock, where hemodynamic instability evolves during early resuscitation. Vasopressor initiation is a key marker of circulatory failure in septic shock.[2] However, the recorded timing of vasopressor initiation may be influenced by ED operational factors, including overcrowding and delays between treatment decisions and medication administration. Therefore, assessing ICU admission delay relative to vasopressor initiation may provide a more physiologically relevant timing measure. This research investigated the association between delays in ICU admission after vasopressor initiation and in-hospital mortality and aimed to identify a clinically relevant delay threshold.

## Methods

A retrospective cohort study was conducted on patients with septic shock treated in the ED at Phrapokklao Hospital, Chanthaburi, Thailand, from 1 January 2024 to 31 December 2025. The *International Statistical Classification of Diseases, Tenth Revision* (*ICD-10*) codes (R57.2 and R65.1) were used to identify patients in the hospital database. This study included adult patients (age ≥ 18 years) who met Sepsis-3 criteria,[11] received vasopressors in the ED, and were subsequently admitted to the ICU. Patients with septic shock referred to Phrapokklao Hospital who were transferred directly from the referring ED were eligible for this study. Exclusion criteria were patients with 1) missing data for vasopressor initiation or ICU admission timing, 2) ED cardiac arrest requiring resuscitation, 3) management via a fast-track ICU pathway, or 4) prior admission to a ward or ICU at the referring hospital.

Index time was defined as ED vasopressor (norepinephrine) initiation for direct presentations and, for referred patients not yet receiving vasopressors. For referred patients already on vasopressors, the index time was ED arrival. In this study, vasopressor initiation meant starting norepinephrine. The primary exposure was ICU admission delay, calculated from index time to ICU admission. The outcome was in-hospital mortality. Electronic medical records served as the data source and included demographics, comorbidities, referral status, infection source, initial lactate, Sequential Organ Failure Assessment (SOFA) score, and norepinephrine dose. Documented timing variables were used to calculate ICU admission delay.

Based on the initially planned multivariable logistic regression model with 6 predictors and the requirement of 15 events per variable, at least 90 events were needed. Given an expected 30% mortality rate,[12] the minimum required sample size was estimated at 300 patients. Continuous and categorical variables were presented as mean (SD) or median (IQR), and frequencies, respectively. Group comparisons were carried out using *t* test, Mann-Whitney *U* test, chi-square, or Fisher exact test, as appropriate. The relationship between delays in ICU admission and in-hospital mortality was investigated using univariable and multivariable logistic regression analysis. Covariates were selected for multivariable analysis based on clinical relevance and associations observed in univariable analysis. To maintain model parsimony, variables representing overlapping aspects of illness severity were not simultaneously included in the primary model.

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Nonlinear associations were evaluated using restricted cubic splines. If no inflection point was identified, delay was examined as a continuous variable and a clinically defined categorical variable. Dose-response relationships were assessed using quartiles with trend testing. Sensitivity analyses were conducted by referral status using subgroup models. A complete-case analysis was used to handle missing data. A 2-sided *P* value < .05 was considered statistically significant. Stata version 18 (StataCorp LLC) was used for analyses.

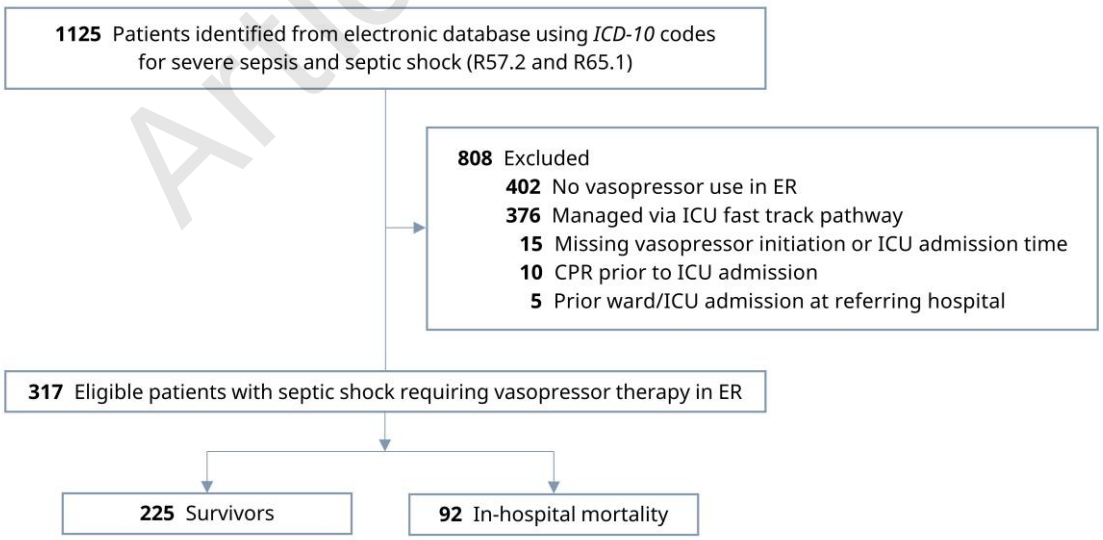
The Chanthaburi Research Ethics Committee/Region 6 (CTIREC 051/69 on 17 April 2026) approved this research. Data extraction commenced only after ethics approval and was completed on 15 May 2026.

## Results

Between 1 January 2024 and 31 December 2025, 1125 patients with severe sepsis and septic shock were assessed; the final analysis included 317 patients (Figure 1). The median age was 62 years, 53.3% were male, and pneumonia was the primary infection source (37.9%). Most patients (68.8%) were referred and had received prearrival vasopressors; for this referral subgroup, index time was defined as ED arrival. Mechanical ventilation was required in 42.0% of cases. Overall in-hospital mortality was 29.0%. Baseline characteristics by survival status are shown. Nonsurvivors were significantly older than survivors (median [IQR], 66 [53-80] vs 61 [47-72] years; *P* = .001). Nonsurvivors were more likely to have malignancy (26.1% vs 10.2%; *P* < .001). In addition, nonsurvivors had greater illness severity, reflected by higher SOFA scores (median [IQR], 10 [8-13] vs 8 [6-10]; *P* < .001), higher initial lactate levels (median [IQR], 6.11 [3.58-9.64] vs 2.75 [2.15-4.45] mmol/L; *P* < .001), and lower mean arterial pressure at index time (median [IQR], 63 [57-69] vs 72 [66-80] mmHg; *P* < .001). The delay in ICU admission was significantly longer in the nonsurvivors group (median [IQR], 3.10 [2.25-4.00] vs 1.53 [1.24-2.41] hours; *P* < .001) (Table 1).

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**Figure 1. Study Flow Diagram**




Abbreviations: CPR, cardiopulmonary resuscitation; ER, emergency department; *ICD-10, International Statistical Classification of Diseases, Tenth Revision*; ICU, intensive care unit.

**Table 1. Baseline Clinical Characteristics of Patients Stratified by In-Hospital Mortality**

| Characteristic | No. (%) | | | *P* Value |
|---|---|---|---|---|
| | Total (n = 317) | Survivors (n = 225) | Nonsurvivors (n = 92) | |
| Age, median (IQR), y | 62 (50-74) | 61 (47-72) | 66 (53-80) | .001 |
| Male | 169 (53.3) | 118 (52.4) | 51 (55.4) | .628 |
| Diabetes mellitus | 90 (28.4) | 70 (31.1) | 20 (21.7) | .093 |
| Chronic kidney diseases | 60 (18.9) | 42 (18.7) | 18 (19.6) | .853 |
| Underlying malignancy | 47 (14.8) | 23 (10.2) | 24 (26.1) | < .001 |
| Initial lactate level, median (IQR), mmol/L | 3.20 (2.25-5.74) | 2.75 (2.15-4.45) | 6.11 (3.58-9.64) | < .001 |
| SOFA score, median (IQR) | 8 (6-11) | 8 (6-10) | 10 (8-13) | < .001 |
| Source of infection | | | | |
| Pneumonia | 120 (37.9) | 79 (35.1) | 41 (44.6) | .332 |
| Genitourinary tract infection | 74 (23.3) | 59 (26.2) | 15 (16.3) | |
| Intraabdominal infection | 74 (23.3) | 51 (22.7) | 23 (25.0) | |
| Skin and soft tissue infection | 15 (4.7) | 11 (4.9) | 4 (4.3) | |
| Other | 34 (10.7) | 25 (11.1) | 9 (9.8) | |
| Referral status | | | | |
| Walk-in | 99 (31.2) | 61 (27.1) | 38 (41.3) | .013 |
| Refer with vasopressor | 218 (68.8) | 164 (72.9) | 54 (58.7) | |
| Hemodynamic profile at index time | | | | |
| Heart rate, median (IQR), beats/min | 102 (86-118) | 100 (84-114) | 111 (92-124) | .001 |
| Systolic blood pressure, median (IQR), mmHg | 96 (84-108) | 99 (90-113) | 84 (77-94) | < .001 |
| Mean arterial pressure, median (IQR), mmHg | 70 (61-77) | 72 (66-80) | 63 (57-69) | < .001 |
| Diastolic blood pressure, median (IQR), mmHg | 57 (50-64) | 60 (52-67) | 50 (45-58) | < .001 |
| Initial management in ED | | | | |
| Mechanical ventilation | 133 (42.0) | 74 (32.9) | 59 (64.1) | < .001 |
| Norepinephrine dose at index time, median (IQR), μg/kg/min) | 0.11 (0.08-0.16) | 0.10 (0.07-0.14) | 0.13 (0.11-0.18) | < .001 |
| Total IV fluid used in ED, median (IQR), L | 2.00 (1.20-2.50) | 2.00 (1.50-2.50) | 2.00 (1.00-2.00) | .269 |
| Antibiotic administration ≤ 1 h | 309 (97.5) | 222 (98.7) | 87 (94.6) | .048 |
| ICU admission delay, median (IQR), h | 2.14 (1.35-3.10) | 1.53 (1.24-2.41) | 3.10 (2.25-4.00) | < .001 |
| Outcomes | | | | |
| Renal replacement therapy | 23 (7.3) | 6 (2.7) | 17 (18.5) | < .001 |
| ICU length of stay, median (IQR), d | 2 (1-4) | 3 (1-4) | 2 (1-5) | .636 |
| Hospital length of stay, median (IQR), d | 5 (3-10) | 5 (4-9) | 4 (1-11) | .006 |

Abbreviations: ED, emergency department; ICU, intensive care unit; IV, intravenous; SOFA, Sequential Organ Failure Assessment.

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Continuous covariates were evaluated for linearity using restricted cubic spline analysis. SOFA score and initial serum lactate showed approximately linear associations with the probability of in-hospital mortality (*P* for nonlinearity = .808 and .304, respectively), whereas age showed evidence of nonlinearity (*P* for nonlinearity = .026). Therefore, age was categorized as < 60 and ≥ 60 years for further regression analyses. Univariable analysis showed significant associations between age, malignancy, referral status, SOFA score, initial lactate level, and ICU admission delay and in-hospital mortality (all *P* < .05). Antibiotic

administration within 1-hour and total intravenous fluid administered in the ED were not significantly associated with mortality. The multivariable model identified malignancy (adjusted odds ratio [aOR], 6.37; 95% CI, 2.57-15.83; *P* < .001), SOFA score (aOR, 1.29; 95% CI, 1.14-1.45; *P* < .001), and initial lactate level (aOR, 1.31; 95% CI, 1.18-1.46; *P* < .001) as independent predictors. Importantly, the findings indicated that the risk of death increased with each additional hour of delayed ICU admission (aOR, 2.33; 95% CI, 1.73-3.15; *P* < .001) (Table 2).

Restricted cubic spline analysis showed a monotonic increase in mortality risk with longer ICU admission delays. Although nonlinearity was statistically significant (*P* < .001), the inflection point remained uncertain (Figure 2). Consequently, ICU admission delay was treated as a continuous variable, and patients were categorized using a 2-hour cutoff for secondary analysis. In-hospital mortality was higher among patients with delays over 2 hours than among those admitted within 2 hours (45.9% vs 6.6%; aOR, 9.21; 95% CI, 3.96-21.42; *P* < .001) (Table 3). Quartile analysis revealed a significant dose-response relationship, with mortality increasing across groups (*P* for trend < .001) (Table 4).

Sensitivity analyses stratified by referral status showed consistent associations between ICU admission delay and in-hospital mortality across subgroups. Among walk-in patients, ICU admission delay and in-hospital mortality were independently associated (aOR, 3.00; 95% CI, 1.72-5.24; *P* < .001). Among referred patients, similar findings were observed (aOR, 2.20; 95% CI, 1.50-3.23; *P* < .001).

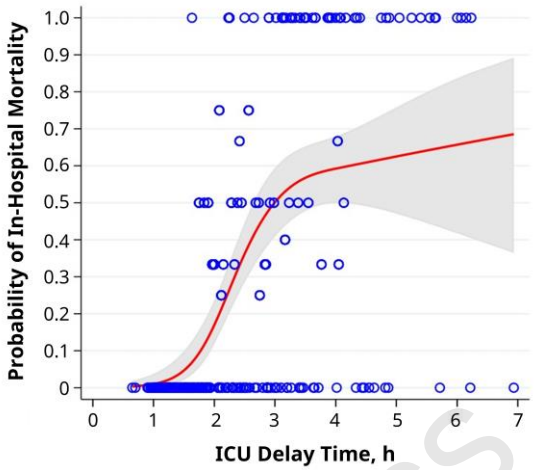
**Table 2. Association Between the Factors and Mortality of Patients With Septic Shock Using Univariable Logistic Regression and Multivariable Logistic Regression Analyses**

| Factors | Univariable Analysis Crude OR (95% CI) | *P* Value | Multivariable Analysis Adjusted OR (95% CI) | *P* Value |
|---|---|---|---|---|
| Age (≥ 60 y) | 1.83 (1.10-3.05) | .020 | 1.31 (0.67-2.58) | .429 |
| Underlying malignancy | 3.10 (1.64-5.85) | < .001 | 6.37 (2.57-15.83) | < .001 |
| Referral status | | | | |
| Walk-in | 1 [Reference] | NA | 1 [Reference] | NA |
| Refer with vasopressor | 0.53 (0.32-0.88) | .014 | 1.48 (0.69-3.20) | .318 |
| SOFA score (per 1 score increment) | 1.33 (1.22-1.46) | < .001 | 1.29 (1.14-1.45) | < .001 |
| Initial lactate level (per 1 mmol/L increment) | 1.37 (1.26-1.50) | < .001 | 1.31 (1.18-1.46) | < .001 |
| Antibiotic administration ≤ 1 h | 0.24 (0.06-1.01) | .051 | 0.33 (0.05-2.28) | .263 |
| Total IV fluid used in ED, per 1 L increment | 0.84 (0.62-1.14) | .269 | 0.80 (0.52-1.21) | .289 |
| ICU admission delay (per 1 h increment) | 2.60 (2.00-3.37) | < .001 | 2.33 (1.73-3.15) | < .001 |

Abbreviations: ED, emergency department; ICU, intensive care unit; IV, intravenous; NA, not applicable; OR, odds ratio; SOFA, Sequential Organ Failure Assessment.

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**Figure 2. Restricted Cubic Spline Showing the Correlation Between ICU Admission Delay and In-Hospital Mortality**




Abbreviation: ICU, intensive care unit.

Adjustments were made for age (≥ 60 years), underlying malignancy, SOFA score, initial lactate level, antibiotic administration within 1-hour, total intravenous (IV) fluid administered in ED, and referral status. The red line shows the adjusted predicted probability of in-hospital mortality as a function of intensive care unit (ICU) admission delay time. The gray shaded area indicates the 95% CI. Blue circles represent observed in-hospital mortality outcomes. *P* for nonlinearity < .001.

**Table 3. Correlation Between ICU Admission Delay Threshold and In-Hospital Mortality**

| ICU Admission Delay, h | Nonsurvivors, No. (%) | Univariable Analysis Crude OR (95% CI) | *P* Value | Multivariable Analysis Adjusted OR (95% CI) | *P* Value |
|---|---|---|---|---|---|
| ≤ 2 | 9 (6.6) | 1 [Reference] | NA | 1 [Reference] | NA |
| > 2 | 83 (45.9) | 11.95 (5.72-24.96) | < .001 | 9.21 (3.96-21.42) | < .001 |

Abbreviations: ICU, intensive care unit; NA, not applicable; OR, odds ratio.

**Table 4. Quartile Analysis of ICU Admission Delay and In-Hospital Mortality**

| Quartile of ICU Admission Delay | Delay Time Range, h | No. (%) | | *P* Value |
|---|---|---|---|---|
| | | Survivors (n = 225) | Nonsurvivors (n = 92) | |
| Quartile 1 | < 1.35 | 80 (100.0) | 0 | < .001 |
| Quartile 2 | 1.35-2.15 | 64 (81.0) | 15 (19.0) | |
| Quartile 3 | 2.15-3.10 | 51 (62.2) | 31 (37.8) | |
| Quartile 4 | > 3.10 | 30 (39.5) | 46 (60.5) | |

Abbreviations: ICU, intensive care unit.

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## Discussion

This study demonstrates that delays in ICU admission after vasopressor initiation are independently associated with higher in-hospital mortality. This association persisted after adjusting for clinical confounders and was supported by a dose–response relationship. Notably, restricted cubic spline analysis showed a monotonic increase in mortality risk without a clear inflection point, suggesting that even incremental delays in ICU admission may worsen outcomes.

The findings in this study align with prior studies linking delayed ICU admission with worse outcomes in sepsis patients.[6, 9] For instance, Zhang et al[9] found that ED stays exceeding 6-12 hours resulted in higher mortality than shorter stays (< 6 hours) (OR, 1.82; 95% CI, 1.28-2.58). Although previous studies generally measured delays from ED arrival, this research used vasopressor initiation, a more physiologically relevant marker of septic shock onset. Beyond ICU admission delay, multivariable analysis identified several independent predictors of in-hospital mortality. Underlying malignancy increased mortality risk, aligning with Li et al,[6] while higher SOFA scores and initial lactate levels reflected disease severity and predicted fatal outcomes, consistent with Zhang et al[9] and Cha et al.[10] Crucially, after adjusting for confounders such as age, malignancy, SOFA score, initial lactate level, referral status, and ED resuscitation metrics (fluid volume and 1-hour antibiotics), ICU admission delay remained independently associated with mortality. This underscores the clinical necessity of timely ICU transfer in septic shock care, while acknowledging potential residual confounding by illness severity and resuscitation intensity.

Recent research has focused on early transfer. Cha et al[10] reported that ICU admission within 3 hours of triage significantly improves survival. In addition, the results further suggest the significance of timing after vasopressor initiation. Although spline analysis demonstrated a continuous increase in mortality risk without a distinct inflection point, a 2-hour window was selected as a pragmatic and actionable operational benchmark for quality improvement and ICU transfer coordination. This cutoff was intended to give clinicians and hospital administrators a clear time target for evaluating and optimizing transfer workflows rather than to represent a fixed physiological threshold.

Many mechanisms explain this relationship. While EDs focus on stabilization on arrival, continued monitoring, prompt vasopressor titration, and effective advanced organ support are vital to managing septic shock.[13] Furthermore, Carlbom et al[3] reported that key limitations to protocol-based sepsis resuscitation in the ED included resource and workflow constraints. In addition, Lasater et al[14] reported that higher nurse-to-patient ratios in the ICU were associated with better outcomes. Consequently, ICU admission delays may reflect a mismatch between patient severity and resource availability, leading to inadequate resuscitation.

From a clinical and operational perspective, these findings suggest that delaying ICU admission after the onset of vasopressor-dependent shock is an important and potentially modifiable component of time-sensitive sepsis care. However, delayed ICU admission is inherently multifactorial and may be influenced by ICU bed availability,[3] ED overcrowding,[4] ongoing stabilization,[7] coordination between ED and ICU teams, and interhospital transfer processes.[6] Greater illness severity may necessitate more intensive and time-consuming ED resuscitation, thereby prolonging ICU transfer.[5, 8] In particular, patients with high initial lactate levels may require more intensive resuscitation and repeated clinical reassessment before transfer, potentially contributing to longer ED stays.[7] Although the association between ICU admission delay and mortality persisted after adjustment for initial lactate

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level, SOFA score, and ED resuscitation-related variables, residual confounding by illness severity, treatment intensity, and response to resuscitation cannot be entirely excluded.

Caution is required when interpreting this study's definition of delay given the differing starting points among referred patients. Notably, 68.8% of patients had already received norepinephrine before transfer; however, because documentation from the referring hospitals was limited, their index time was defined as their arrival at the ED. While this methodology provides a consistent benchmark for assessing intrahospital transfer timelines, it omits the duration of prearrival shock and may be underestimating the true physiological delay. To account for this diversity, patient referral status was included as a covariate in the multivariable regression analysis. Delayed ICU admission was consistently associated with mortality. These results effectively capture the operational window between the institutional index time and ICU transfer. However, clinicians must acknowledge that recorded times for ED vasopressor initiation may be subject to measurement errors. In a highly demanding ED environment, a pragmatic gap often exists between a physician's decision and the nursing intervention during time-sensitive resuscitations.[7]

This study has several limitations. First, the initial sample size estimate followed the event-per-variable rule of thumb rather than an a priori power calculation for comparing independent proportions. Nevertheless, a post hoc analysis based on the observed mortality rates indicated sufficient statistical power to detect between-group differences. Second, granular data on prehospital care and ED management workflows were scarce, particularly for referred patients. These included prearrival fluid volumes, the precise timing of norepinephrine initiation, and the duration of shock or vasopressor exposure before arrival. Serial hemodynamic measurements, cumulative vasopressor requirements, and lactate clearance during the ED stay were also unavailable. Consequently, resuscitation adequacy and dynamic clinical responses before ICU admission could not be fully characterized. Third, despite multivariable adjustment, residual confounding related to illness severity, treatment intensity, and resuscitation response cannot be entirely ruled out. Patients with greater illness severity may require more intensive and time-consuming ED interventions or procedures, which could prolong ICU transfer and independently increase mortality. Therefore, the observed association between ICU admission delay and mortality should be interpreted cautiously, as it may reflect a complex interplay of clinical factors rather than a simple causal link. Finally, these findings are based on a single-center cohort and may not generalize to settings with different healthcare systems, institutional resources, or ICU admission protocols.

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## Conclusions

ICU admission delay after vasopressor initiation was independently associated with increased in-hospital mortality in patients with septic shock, with mortality risk rising as the delay lengthened. Although this risk became more pronounced beyond a 2-hour delay, this timeframe should be viewed as an operational cutoff rather than a fixed physiological threshold. Given the retrospective design and potential residual confounding, these findings warrant cautious interpretation and highlight the need for prospective studies to confirm whether expedited ICU transfer directly improves patient outcomes.

## Additional Information

**Acknowledgments:** The author thanks all participants who were involved in this study.

**Ethics Approval:** This study was approved by the Chanthaburi Research Ethics Committee/ Region 6 (CTIREC 051/69 on 17 April 2026).

**Clinical Trial Consideration:** This study does not report on a clinical trial.

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