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ORIGINAL ARTICLE

In-hospital mortality after concomitant pneumonia in acute coronary syndrome

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ABSTRACT

BACKGROUND
Pneumonia may complicate acute coronary syndrome (ACS), but its association with mortality during ACS hospitalization requires further characterization. This study evaluated the prevalence of concomitant pneumonia and its adjusted association with in-hospital all-cause mortality in patients with ACS.

METHODS
This retrospective cohort study included 494 adults admitted with ACS between November 2022 and November 2023. Pneumonia was identified during the index hospitalization. Univariable and multivariable logistic regression estimated odds ratios (ORs) and 95% confidence intervals (CIs) for mortality. Exploratory analyses assessed pneumonia-by-ACS-subtype interactions and adjusted associations with log-transformed length of stay. Adjustment included demographic characteristics, ACS subtype, and recorded clinical conditions. A sensitivity analysis of hospitalization duration was restricted to patients who survived to discharge.

RESULTS
Pneumonia was identified in 99 patients (20.0%); 171 patients (34.6%) died during hospitalization. Mortality was higher with pneumonia than without pneumonia (63.6% vs. 27.3%; crude OR 4.65, 95% CI 2.92–7.41; p<0.001). After adjustment, pneumonia remained associated with mortality (adjusted OR 9.69, 95% CI 5.18–18.13; p<0.001). There was no evidence of interaction with ACS subtype (p=0.787). Median hospital stay was longer with pneumonia (9 vs. 6 days; p<0.001), consistent with the adjusted analysis (geometric mean ratio 1.49, 95% CI 1.38–1.60; p<0.001).

CONCLUSIONS
Concomitant pneumonia affected one in five patients with ACS and was associated with substantially higher adjusted odds of in-hospital mortality and longer hospitalization. These findings support careful assessment for pneumonia during ACS care. Prospective studies recording infection onset and illness severity are needed to clarify the temporal relationship and evaluate management strategies.

Keywords: Acute coronary syndrome; hospital mortality; concomitant pneumonia; outcome research

ABBREVIATIONS
ACS: acute coronary syndrome
ADHF: acute decompensated heart failure
AF: atrial fibrillation

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AKI: acute kidney injury
aOR: adjusted odds ratio
CAP: community-acquired pneumonia
CAPE COD: community-acquired pneumonia: evaluation of corticosteroids
CHF: congestive heart failure
CI: confidence interval
CKD: chronic kidney disease
cOR: crude odds ratio
FORCE-ACS: future optimal research and care evaluation in patients with acute coronary syndrome
GRACE: global registry of acute coronary events
HHD: hypertensive heart disease
IQR: interquartile range
NSTEMI: non-ST-elevation myocardial infarction
OR: odds ratio
SCAI: Society for Cardiovascular Angiography and Interventions
SD: standard deviation
STEMI: ST-elevation myocardial infarction
TIMI: thrombolysis in myocardial infarction
UAP: unstable angina pectoris

INTRODUCTION

Acute coronary syndrome (ACS), comprising unstable angina pectoris (UAP), non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI), requires timely diagnosis and treatment to reduce adverse outcomes.[1] Concomitant illnesses may complicate treatment and influence prognosis. Pneumonia is relevant because respiratory compromise and systemic infection may aggravate myocardial oxygen supply–demand imbalance.[2] The 2025 American guideline likewise emphasizes prompt, evidence-based ACS care.[3]

Recent evidence also describes cardiovascular complications after pneumonia. A systematic review found higher odds of ACS, stroke, and mortality after community-acquired pneumonia, although substantial heterogeneity limited interpretation.[4] A register-based study distinguished time-dependent cardiovascular risks after inpatient and outpatient pneumonia, while a coronary artery disease cohort modeled pneumonia as a time-varying exposure.[5,6] These studies support the clinical relevance of the infection–cardiovascular relationship but do not directly estimate mortality during an ACS admission.

Previous studies have linked respiratory infections with adverse myocardial infarction outcomes. In a United States National Inpatient Sample analysis of 10,880,856 admissions, Vallabhajosyula et al.[2] identified respiratory infections in 6.9%. In-hospital mortality was higher with infection (14.5% vs. 5.5%), and the association persisted after propensity matching (adjusted OR 1.25, 95% CI 1.24–1.26). Hospitalization was also longer. In South Korea, Kim et al.[7] found increased subsequent pneumonia incidence after ACS hospitalization (adjusted hazard ratio 1.25, 95% CI 1.09–1.42). That study assessed long-term pneumonia occurrence rather than mortality during the index admission.

In Indonesia, Putri et al.[8] studied 794 patients with ACS at Dr. Sardjito General Hospital; pneumonia was recorded in 14.1%. Infectious comorbidities were associated with mortality after adjustment (OR 2.04, 95% CI 1.32–3.14). However, pneumonia, urinary tract infection, and sepsis were combined as one exposure, so the model did not isolate the pneumonia-specific association.

Previous evidence is consistent in linking infection with adverse ACS outcomes, but leaves a more specific question unresolved. The United States study evaluated respiratory infections in myocardial infarction, whereas the Indonesian study combined pneumonia, urinary tract infection, and sepsis into a composite exposure.[2,8] Neither directly provides a pneumonia-specific adjusted estimate across UAP, NSTEMI, and STEMI in the present referral setting. Our study addresses that contextual evidence gap by estimating the association of pneumonia,

considered separately from other recorded infections, with mortality during the index ACS admission. Exploratory analyses additionally assess whether this association varies by ACS subtype and examine adjusted hospitalization duration. These contributions extend local evidence rather than establish a previously unrecognized infection–mortality relationship. The distinction from studies examining myocardial infarction after pneumonia is also important because those studies use a different index population and temporal framework.[9]

Our study aimed to determine the prevalence of concomitant pneumonia and evaluate its adjusted association with in-hospital all-cause mortality. Length of hospital stay was evaluated as a secondary outcome.

METHODS

Study design

This retrospective cohort study was conducted at Hasna Medika Cardiovascular Hospital, a cardiovascular referral hospital in Cirebon, West Java, Indonesia. Medical-record review and study analyses were conducted from December 2025 to April 2026. The source records covered patients admitted with ACS between November 2022 and November 2023.

Study subjects

Eligible patients were adults aged ≥18 years with a confirmed diagnosis of UAP, NSTEMI, or STEMI based on clinical presentation, electrocardiographic findings, and cardiac biomarkers, as appropriate for each subtype. Patients with missing primary outcome data or incomplete medical records were excluded. All consecutive eligible patients within the specified period were included, yielding 494 patients.

Measurements

Pneumonia was defined as a new pulmonary infiltrate on chest radiography accompanied by at least two of the following: fever >38°C, leukocytosis >10,000/mm³, purulent sputum production, or oxygen saturation <90% on room air. These constituted the study's operational criteria. Pneumonia documented at any point during the index hospitalization was treated as the primary exposure. Community-acquired and hospital-acquired pneumonia were not analyzed separately.

Covariates included age, sex, ACS subtype, acute decompensated heart failure (ADHF)/congestive heart failure (CHF), diabetes mellitus, hypertensive heart disease (HHD), chronic kidney disease (CKD)/acute kidney injury (AKI), cardiogenic shock, stroke, atrial fibrillation (AF), and sepsis. HHD was analyzed as hypertensive heart disease, rather than hypertension alone. CKD and AKI were combined in the source dataset and analyzed as a single recorded clinical variable. Heart failure was defined according to the 2021 European Society of Cardiology (ESC) guidelines. [10] Cardiogenic shock was defined as sustained systolic blood pressure <90 mmHg with evidence of end-organ hypoperfusion despite adequate volume resuscitation.

Study outcomes

The primary outcome was in-hospital all-cause mortality. The secondary outcome was length of hospital stay in days. Patients were followed through the index hospitalization to death or discharge.

Statistical analysis

Categorical variables were summarized as frequencies and percentages. Continuous variables were expressed as mean (SD) or median (IQR). Characteristics were compared between patients who died and those who survived using Pearson's chi-square or Fisher's exact test, as appropriate. Age was compared using Welch's independent-samples t-test because it allows unequal population variances and is appropriate for groups of unequal size (323 survivors and 171 deaths), without requiring the equal-variance assumption of the pooled Student's t-test. Its use does not imply that unequal variances were demonstrated by a preliminary test. Length of stay was compared using the Mann–Whitney U test.

Univariable logistic regression estimated crude ORs and 95% CIs. Variables with Wald $p<0.25$ were considered for the multivariable model. Pneumonia was retained as the primary exposure. ACS subtype was entered as a categorical variable with UAP as the reference, retaining both subtype indicators. The multivariable model simultaneously included pneumonia, sex, age ≥60 years, ADHF/CHF, diabetes, HHD, CKD/AKI, cardiogenic shock, stroke, AF, sepsis, and ACS subtype. OR confidence intervals and p-values were based on Wald inference.

Exploratory effect modification on the multiplicative odds scale was assessed by adding two product terms, pneumonia × NSTEMI and pneumonia × STEMI, to the full multivariable logistic model, with UAP as the reference subtype. A joint two-degree-of-freedom Wald test evaluated the null hypothesis that both interaction coefficients were zero. The pneumonia-associated log OR was the pneumonia coefficient for UAP, that coefficient plus the pneumonia × NSTEMI coefficient for NSTEMI, and that coefficient plus the pneumonia × STEMI coefficient for STEMI. Subtype-specific adjusted ORs and CIs were derived from these linear combinations and their model-based covariance. This analysis tests differences in pneumonia-associated ORs between subtypes; it does not test additive interaction or differences in absolute risk. Length of stay was analyzed using linear regression of log-transformed days with HC3 heteroskedasticity-robust standard errors and the same covariates as the mortality model. Exponentiated coefficients were reported as adjusted geometric mean ratios. A sensitivity analysis was restricted to patients who survived because death can truncate hospitalization.

Analyses used all 494 observations with complete values for the modeled variables. Two-sided $p<0.05$ was considered statistically significant. Analyses were performed in Python 3.12. Logistic regression was fitted by maximum likelihood, with numerical convergence verified.

Ethical considerations

The study was approved by the ethics committee of Universitas Swadaya Gunung Jati, Cirebon, Indonesia (approval number: 060/LAIKETIK/KEPPKRSGJ/X/2024). Written informed consent was waived because the study involved retrospective analysis of de-identified records. The study was conducted in accordance with the Declaration of Helsinki.

RESULTS

Patient characteristics according to mortality status

The study included 494 patients, with a mean age of 58.2 (SD 10.3) years, among whom 380 (76.9%) were male. There were 173 patients with UAP (35.0%), 157 with NSTEMI (31.8%), and 164 with STEMI (33.2%). Pneumonia was recorded in 99 patients (20.0%), and 171 patients (34.6%) died during hospitalization. Patients who died were older and more often had pneumonia and the clinical conditions shown in Table 1. STEMI accounted for 45.6% of patients who died and 26.6% of survivors.

Table 1. Patient characteristics according to in-hospital mortality

Characteristic	Survived (n=323)	Died (n=171)	p value
Age, years, mean (SD)	57.1 (10.3)	60.2 (9.8)	<0.001
Female sex	49 (15.2)	65 (38.0)	<0.001
Pneumonia	36 (11.1)	63 (36.8)	<0.001
ADHF/CHF	250 (77.4)	146 (85.4)	0.034
Diabetes mellitus	4 (1.2)	34 (19.9)	<0.001
Hypertensive heart disease	2 (0.6)	9 (5.3)	0.002
CKD/AKI	8 (2.5)	36 (21.1)	<0.001
Cardiogenic shock	3 (0.9)	7 (4.1)	0.037
Stroke	36 (11.1)	35 (20.5)	0.005
Atrial fibrillation	11 (3.4)	25 (14.6)	<0.001
Sepsis	7 (2.2)	7 (4.1)	0.258
ACS subtype			<0.001
UAP	127 (39.3)	46 (26.9)	
NSTEMI	110 (34.1)	47 (27.5)	
STEMI	86 (26.6)	78 (45.6)	

Note: Values are n (%) unless stated otherwise; percentages use the column denominator. Age: Welch's independent-samples t-test. Categorical variables: Pearson's chi-square or Fisher's exact test when an expected cell count was <5. ACS subtype has one overall Pearson chi-square p-value. ACS, acute coronary syndrome; UAP, unstable angina pectoris; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; ADHF, acute decompensated heart failure; CHF, congestive heart failure; CKD, chronic kidney disease; AKI, acute kidney injury; SD, standard deviation.

Table 2. Univariate and multivariate logistic regression analyses of in-hospital mortality

Variable	Crude OR (95% CI)	p value	Adjusted OR (95% CI)	p value
Pneumonia	4.65 (2.92–7.41)	<0.001	9.69 (5.18–18.13)	<0.001
Female sex	3.43 (2.22–5.29)	<0.001	4.95 (2.74–8.94)	<0.001
Age ≥60 years	1.48 (1.02–2.14)	0.040	1.37 (0.81–2.31)	0.241
ADHF/CHF	1.71 (1.04–2.81)	0.036	2.76 (1.35–5.62)	0.005
Diabetes mellitus	19.79 (6.89–56.85)	<0.001	29.48 (8.70–99.91)	<0.001
Hypertensive heart disease	8.92 (1.90–41.75)	0.005	9.72 (1.59–59.45)	0.014
CKD/AKI	10.50 (4.75–23.19)	<0.001	21.22 (8.10–55.60)	<0.001
Cardiogenic shock	4.55 (1.16–17.84)	0.030	10.33 (1.93–55.18)	0.006
Stroke	2.05 (1.23–3.41)	0.006	4.53 (2.18–9.41)	<0.001
Atrial fibrillation	4.86 (2.33–10.14)	<0.001	6.91 (2.68–17.83)	<0.001
Sepsis	1.93 (0.66–5.59)	0.227	5.38 (1.55–18.72)	0.008
NSTEMI vs. UAP	1.18 (0.73–1.91)	0.500	1.15 (0.60–2.22)	0.667
STEMI vs. UAP	2.50 (1.59–3.95)	<0.001	3.20 (1.68–6.08)	<0.001

Pneumonia and in-hospital mortality

Mortality was higher with pneumonia than without pneumonia [63/99 [63.6%] vs. 108/395 [27.3%]; crude OR 4.65, 95% CI 2.92–7.41; p<0.001]. The association persisted after adjustment (aOR 9.69, 95% CI 5.18–18.13; p<0.001). Female sex, ADHF/CHF, diabetes, HHD, CKD/AKI, cardiogenic shock, stroke, AF, sepsis, and STEMI presentation were also associated with mortality after adjustment. Age ≥60 years and NSTEMI presentation were not independently associated (Table 2).

Exploratory analysis by ACS subtype

In the interaction model, pneumonia was associated with higher adjusted odds of death within UAP (aOR 9.80, 95% CI 3.39–28.34), NSTEMI (aOR 12.34, 95% CI 4.43–34.41), and STEMI (aOR 7.56, 95% CI 2.77–20.63). The joint test of the two pneumonia-by-subtype interaction terms was not significant (p=0.787; Table 3). Thus, there was no statistical evidence that the pneumonia-associated OR differed by ACS subtype on the multiplicative odds scale. The significant within-subtype associations do not themselves establish interaction. Wide CIs limit precision, and a nonsignificant interaction test does not establish equivalent associations across subtypes.

Length of hospital stay

Median hospital stay was longer with pneumonia (9 days [IQR 7–12]) than without pneumonia (6 days [IQR 5–7]; p<0.001). Pneumonia was also associated with longer hospitalization in the adjusted log-linear analysis (geometric mean ratio 1.49, 95% CI 1.38–1.60; p<0.001). Among the 323 survivors, the sensitivity analysis yielded a geometric mean ratio of 1.59 (95% CI 1.42–1.77; p<0.001).

Table 3. Exploratory adjusted association between pneumonia and mortality by ACS subtype

ACS subtype	Adjusted OR (95% CI)	p-value
UAP	9.80 (3.39–28.34)	<0.001
NSTEMI	12.34 (4.43–34.41)	<0.001
STEMI	7.56 (2.77–20.63)	<0.001
Pneumonia × ACS subtype interaction	Joint Wald test (2 df)	0.787

Note: n=494. Subtype-specific adjusted ORs compare pneumonia with no pneumonia within each ACS subtype and were derived from one full multivariable logistic model containing pneumonia × NSTEMI and pneumonia × STEMI product terms, with UAP as the reference subtype. Adjustment included sex, age ≥60 years, ADHF/CHF, diabetes, HHD, CKD/AKI, cardiogenic shock, stroke, AF, sepsis, and ACS subtype. The joint Wald test (2 degrees of freedom) tests whether both interaction coefficients equal zero; p=0.787 indicates no evidence of effect modification on the multiplicative odds scale. The within-subtype p-values test each pneumonia-associated OR against 1 and are distinct from the interaction p-value. ACS, acute coronary syndrome; UAP, unstable angina pectoris; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; OR, odds ratio; CI, confidence interval; df, degrees of freedom.

DISCUSSION

In this study cohort, pneumonia was recorded in one fifth of patients with ACS and was associated with substantially higher adjusted odds of in-hospital death. The association remained after inclusion of demographic characteristics, ACS subtype, and recorded clinical conditions. Pneumonia was also associated with longer hospitalization. These findings identify pneumonia as a clinically relevant marker of adverse outcomes, without establishing a causal effect or showing that treatment of pneumonia reduces mortality. The direction of the mortality association agrees with the United States analysis of myocardial infarction with respiratory infection and the Indonesian study of composite infectious comorbidities.[2,8] The larger adjusted OR in the present cohort should not be interpreted as evidence of a stronger causal effect, because the populations, exposure definitions, covariate sets, and analytic approaches differed. Likewise, the 20.0% pneumonia frequency was higher than the 14.1% recorded in the Sardjito cohort, but differences in ascertainment and patient selection limit direct comparison.[8] The overall mortality of 34.6% warrants careful consideration of referral patterns and illness severity. The available data do not establish why mortality was high. A separate Indonesian ACS cohort at Hasan Sadikin Hospital reported in-hospital mortality of 10.6%, further illustrating variation across settings and case mix.[11] That difference cannot be attributed to pneumonia alone.

Several mechanisms could plausibly contribute to adverse outcomes when pneumonia and ACS coexist. Hypoxemia and infection-related metabolic demands may worsen myocardial oxygen supply–demand imbalance, while systemic inflammation may aggravate cardiovascular dysfunction.[2,7] These are biological explanations rather than mechanisms measured in this study. In patients with heart failure, respiratory infection and congestion may coexist, and pulmonary edema may resemble pneumonia on imaging. The high prevalence of ADHF/CHF in this cohort reinforces the importance of considering diagnostic overlap. [10] In a prospective community-acquired pneumonia (CAP) cohort, higher circulating lipopolysaccharide levels were associated with short- and long-term cardiovascular events, providing supporting observational evidence for an inflammatory pathway.[12] Among patients admitted with acute heart failure, hospital-acquired pneumonia was associated with higher in-hospital mortality and longer hospitalization, although the population and the infection definition differ from ours.[13] Diabetes and CKD/AKI had large adjusted associations with mortality, but their wide confidence intervals indicate limited precision. Several conditions were uncommon, including HHD and cardiogenic shock, and sparse cells may inflate estimates. The combined CKD/AKI variable also mixes chronic disease with an acute complication and cannot distinguish their separate contributions. Contemporary guidance emphasizes cardiovascular risk and coordinated care in kidney disease and diabetes, but does not validate the magnitude of these cohort-specific estimates.[14,15] Female sex was associated with mortality, although treatment differences and presentation characteristics were not available to explain this finding. The 2023 ESC diabetes guideline also addresses the overlapping risks of coronary disease, heart failure, arrhythmia, and kidney disease.[16]

The association with female sex requires cautious interpretation. Contemporary registries from Japan, Egypt, and Australia describe differences in age, comorbidities, presentation, and early outcomes between women and men with ACS.[17-19] In the Future Optimal Research and Care Evaluation in Patients with Acute Coronary Syndrome (FORCE-ACS) registry, women received less revascularization and pharmacotherapy, but the higher unadjusted mortality at 36 months did not persist after adjustment.[20] Differences in follow-up and adjustment preclude direct comparison with our in-hospital estimate; our data cannot determine whether sex-related treatment disparities contributed to mortality.

Sepsis and shock may represent overlapping pathways of severe illness. A national analysis of myocardial infarction with cardiogenic shock found that concomitant sepsis was associated with higher mortality, more organ failure, and longer hospitalization.[21] The updated Society for Cardiovascular Angiography and Interventions (SCAI) shock classification emphasizes graded clinical severity rather than a single binary diagnosis.[22] Because shock stages and onset times were not available here, residual confounding by hemodynamic severity remains

possible. The classification is discussed as context and was not retrospectively applied to this dataset.

Arrhythmia deserves attention when pneumonia coexists with cardiovascular disease. A Danish cohort found increased thromboembolic risk and recurrent AF after pneumonia-associated new-onset AF.[23] The 2024 ESC AF guideline emphasizes individualized assessment of comorbidities, stroke risk, and rhythm or rate management.[24] Our binary AF variable did not distinguish pre-existing from incident arrhythmia, so its adjusted association cannot establish an infection-triggered mechanism.

The exploratory interaction analysis did not show evidence of variation in the pneumonia–mortality association across UAP, NSTEMI, and STEMI. This finding does not establish equivalent effects: the wide subgroup confidence intervals and limited sample size reduce the ability to detect interaction. The main adjusted estimate should therefore remain the primary result.

The longer hospitalization associated with pneumonia was supported by both the unadjusted comparison and the exploratory log-linear model. The geometric mean ratio describes proportional differences in hospitalization duration and should not be interpreted as a difference in days or a ratio of medians. The survivor-only analysis showed a consistent direction, but conditioning on survival creates a selected population. Moreover, prolonged hospitalization may increase the opportunity to develop or diagnose pneumonia. Without infection-onset dates, the temporal direction of this association remains uncertain.

For hospitalized patients with ACS, these findings support careful evaluation of new respiratory symptoms, hypoxemia, fever, and pulmonary infiltrates, together with assessment for congestion and other alternative diagnoses. Recognition of concomitant infection should prompt coordinated cardiovascular and respiratory care, with treatment guided by the clinical diagnosis, severity, and applicable recommendations.[1,10,25,26] This study does not support routine prophylactic antibiotics or prove that a pneumonia-screening bundle reduces mortality. Prospective evaluation of diagnostic and management pathways is needed before their outcome benefits can be claimed. International severe-CAP guidance further supports assessment of infection severity, appropriate antimicrobial treatment, and organ support when indicated.[27]

Therapeutic evidence must be interpreted within its own population. The Community-Acquired Pneumonia: Evaluation of Corticosteroids (CAPE COD) trial demonstrated a mortality benefit from hydrocortisone in selected ICU patients with severe community-acquired pneumonia; this does not establish an indication for all patients with ACS and pneumonia.[28] A recent multicenter comparison of clarithromycin and azithromycin did not show a consistent difference in cardiovascular events across settings, and its observational design left uncertainty about confounding.[29] Our dataset contains no antimicrobial regimen or corticosteroid information and therefore cannot compare treatment efficacy or cardiovascular safety.

Future studies should capture frailty, cardiac biomarkers, pneumonia severity, and the timing of complications. In a prospective cohort of older adults with CAP, frailty and cardiac biomarkers added prognostic information beyond the Pneumonia Severity Index for secondary cardiovascular events.[30] That result requires external validation and cannot be assumed to validate a prediction model in our younger ACS population.

The retrospective single-center design limits generalizability and causal inference. Pneumonia was defined clinically and radiologically without microbiological confirmation, and community-acquired and hospital-acquired infections were not distinguished. Infection-onset dates were unavailable in the analysis dataset. Classifying pneumonia detected at any time during admission as a fixed exposure may introduce time-dependent exposure bias and does not demonstrate that pneumonia preceded clinical deterioration. Longer admission also increases the opportunity for pneumonia ascertainment. The timing of clinical covariates was not established; heart failure, shock, kidney injury, and sepsis could precede or follow pneumonia. Adjustment may therefore include mediators or complications rather than baseline confounders. Global Registry of Acute Coronary Events (GRACE) and Thrombolysis in Myocardial Infarction (TIMI) scores, serial troponin, cardiac function, pneumonia severity, and time to revascularization were not uniformly available for adjustment. CKD and AKI were grouped, and HHD does not capture all patients with hypertension. Sparse clinical categories and wide confidence intervals limit precision. With mortality occurring in 34.6% of patients, ORs should not be interpreted as risk ratios. The subgroup and length-of-stay analyses were exploratory; the survivor-only sensitivity

analysis is additionally subject to selection on survival. Prospective multicenter studies with standardized diagnoses, exposure timing, treatment information, and independent validation are warranted.

CONCLUSION

Concomitant pneumonia was documented in 20.0% of patients with ACS and was associated with higher adjusted odds of in-hospital all-cause mortality. Pneumonia was also associated with longer hospitalization in an exploratory adjusted analysis. These findings support careful assessment for pneumonia during ACS care, while prospective studies are needed to clarify temporal relationships and evaluate whether specific management strategies improve outcomes.

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Conflict of Interest

All authors declare no conflicts of interest related to this submission.

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Authors' Contributions

YPR: Conceptualization; Data Curation; Formal Analysis; Investigation; Methodology; Project Administration; Writing – Original Draft; Writing – Review & Editing. WP: Supervision; Validation; Writing – Review & Editing. All authors are responsible for reviewing and approving the final version before submission.

Data Availability Statement

The data presented in this study are available upon reasonable request from the corresponding author.

Declaration of AI Usage in Scientific Writing

During manuscript revision, DeepL was used to assist with English-language editing. No new patient-level analyses were performed for this revision; the statistical estimates reported in the supplied manuscript were retained. The authors are responsible for verifying the analyses, interpretation, references, and final manuscript and for approving the submitted version.

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