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Prognostic association between the pulmonary artery-to-ascending aorta diameter ratio and outcomes in hospitalized patients with community-acquired pneumonia: A retrospective computed tomography–based study

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ABSTRACT

Aim: To investigate the prognostic value of pulmonary artery (PA) diameter, ascending aortic (Ao) diameter, and the pulmonary artery-to-aorta diameter ratio (PA/Ao ratio) measured on thoracic computed tomography (CT) in patients hospitalized with community-acquired pneumonia (CAP).

Methods: This retrospective observational study included adult patients hospitalized with CAP between January 2020 and April 2024. Demographic, clinical, laboratory, and radiological data were collected from electronic medical records. PA and Ao diameters were measured on thoracic CT scans, and the PA/Ao ratio was calculated. The primary outcome was defined as a composite of in-hospital mortality, intensive care unit admission, or requirement for mechanical ventilation. Receiver operating characteristic (ROC) analysis and logistic regression models were used to evaluate prognostic performance and identify independent predictors of poor outcomes.

Results: A total of 200 patients were included, and 107 (53.5%) experienced the primary outcome. Patients with poor prognosis were significantly older and had higher lactate levels, lower oxygen saturation, and higher frequencies of heart failure and malignancy. Although PA diameter, Ao diameter, and PA/Ao ratio were numerically higher in patients with poor outcomes, none demonstrated a significant independent association with prognosis. ROC analyses showed limited discriminatory performance for PA diameter (AUC: 0.578), Ao diameter (AUC: 0.583), and PA/Ao ratio (AUC: 0.528). Multivariable logistic regression identified elevated lactate levels (OR: 1.369, p=0.004), lower oxygen saturation (OR: 0.901, p<0.001), and heart failure (OR: 2.936, p=0.008) as independent predictors of poor prognosis.

Conclusions: PA diameter, Ao diameter, and the PA/Ao ratio measured on admission thoracic CT were not useful predictors of poor outcomes in hospitalized CAP patients. Clinical and physiological parameters, particularly oxygen saturation, lactate level, and heart failure, provided superior prognostic information and should remain the cornerstone of risk stratification in CAP.

Keywords: Community-acquired pneumonia, computed tomography, prognosis, mortality.

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1. Introduction

Community-acquired pneumonia (CAP) is an infectious and inflammatory disease involving pulmonary parenchyma and associated with considerable morbidity and

mortality rates. This medical condition recognized as the leading cause of mortality attributable to infectious diseases is among the major causes of death. Pneumonia may represent the principal cause of mortality in territories with limited socioeconomic resources [1].

Hospitalization is required in approximately 40% of patients with CAP, while nearly 5% necessitate intensive care unit (ICU) admission. Despite improvements in the management of severe pneumonia and a decline in mortality over the past several decades, the mortality rate remains substantial with an approximate rate of 20%. The patients who initially do not fulfil the criteria for severe pneumonia but subsequently experience clinical deterioration and develop severe disease may have a higher risk of mortality than patients who are already classified as having severe pneumonia at the time of presentation [2]. These findings emphasize the need for reliable prognostic parameters that can be assessed at the time of hospital admission.

Several clinical prediction models have been introduced to estimate the mortality risk in patients with CAP. The Pneumonia Severity Index (PSI) and CURB-65 are the most extensively validated and commonly implemented scoring systems. According to published meta-analyses, both tools demonstrate similar and clinically acceptable accuracy for mortality prediction. Although the PSI has a greater ability to identify patients at low risk, CURB-65 is often favored in routine clinical practice because it is straightforward and requires fewer variables [3,4]. Nevertheless, no single prognostic tool has been universally accepted as a gold-standard method for identifying patients who are likely to deteriorate clinically or experience mortality at the time of initial assessment.

The pulmonary artery (PA) transports deoxygenated blood from the right ventricle to the pulmonary circulation and represents an important vascular component of the respiratory system. Previous studies have reported that an increased PA diameter is associated with mortality in patients with chronic obstructive pulmonary disease (COPD) and coronavirus disease 2019 (COVID-19) [5,6].

In recent years, particular interest has focused on the pulmonary artery-to-aorta diameter ratio (PA/Ao ratio). An increased PA/Ao ratio has been linked to a higher risk of mortality in patients with COVID-19 [7]. Similarly, investigations involving patients with COPD have reported associations between an elevated PA/Ao ratio and adverse clinical progression; however, the association between this parameter and mortality has not been consistently demonstrated [8,9].

The data concerning the prognostic significance of the PA/Ao ratio in pneumonia remain relatively scarce, particularly with respect to clinically important outcomes such as endotracheal intubation, cardiopulmonary resuscitation, ICU admission, and mortality. Accordingly, this study was designed to evaluate whether the PA/Ao ratio determined on thoracic computed tomography (CT) is associated with clinical outcomes and mortality in hospitalized patients with community-acquired pneumonia.

2. Materials and methods

2.1. Study Design and Patient Selection: Based on the definitions reported in the current literature, adult community-acquired pneumonia (CAP) was identified by the presence of one or more newly developed pulmonary infiltrates on chest imaging together with at least one respiratory symptom and at

least one additional clinical, laboratory, or symptomatic finding compatible with an acute pulmonary infection. Respiratory manifestations included the onset or worsening of cough, sputum production, dyspnea, and pleuritic chest pain. Other supportive findings included abnormal pulmonary auscultation findings, fever, leukocytosis or otherwise unexplained bandemia, and hypoxemia.

This retrospective observational study included all adult patients who have referred to the emergency department between January 1, 2020, and April 1, 2024. Patients were eligible if they received a diagnosis of CAP based on clinical assessment and thoracic computed tomography (CT) findings and were subsequently admitted to the hospital.

Patients were excluded when any of the following were present; treatment in the outpatient setting without hospital admission; lack of thoracic CT imaging; laboratory-confirmed COVID-19 infection; age below 18 years; pregnancy; presentation associated with trauma; clinical findings indicating hospital-acquired pneumonia (HAP); or insufficient clinical information for the study analysis. HAP was defined as the pneumonia that appeared at least 48 hours after hospital admission in a patient who did not have pneumonia, or evidence of an incubating infection, at the time of admission.

2.2. Data Collection: Demographic, clinical, laboratory, and outcome data were retrospectively obtained from electronic medical records and archived patient files.

The following variables recorded at admission were collected;

- Age and gender
- Comorbid diseases
- Systolic and diastolic blood pressures
- Heart rate
- Peripheral oxygen saturation (SpO_2)
- Shock index (heart rate/systolic blood pressure)

2.3. Laboratory parameters included the following;

- White blood cell count (WBC)
- Hemoglobin (Hb)
- Hematocrit (Hct)
- Neutrophil count
- Lymphocyte count
- Monocyte count
- Neutrophil-to-lymphocyte ratio (NLR)
- Platelet count
- Blood glucose
- Blood urea nitrogen (BUN)
- Creatinine
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)
- Lactate dehydrogenase (LDH)
- Calcium
- C-reactive protein (CRP)
- Lactate
- Base excess (BE)

Clinical outcomes included hospital admission location (ward or ICU), length of hospital stay, requirement for mechanical ventilation, endotracheal intubation, cardiopulmonary resuscitation (CPR), and in-hospital mortality.

2.4. CT Scan Measurements: The pulmonary artery (PA) and ascending aortic (Ao) diameters were independently measured by two board-certified emergency physicians (MAE and KBA) through axial thoracic CT images obtained at the level of the pulmonary artery bifurcation. Measurements were performed from inner edge to inner edge, using the maximum diameter perpendicular to the longitudinal axis of the respective vessel. The measurements were subsequently reviewed jointly, and consensus values were recorded.

The pulmonary artery-to-aorta diameter ratio (PA/Ao ratio) was then calculated and entered into the study database. Thoracic CT examinations were performed through different multidetector CT scanners, including [Optima 660J/GE], with a slice thickness ranging from [2] to [5] mm.

2.5. Outcome Definition: The primary outcome was defined as poor prognosis, which was considered present if at least one of the following events occurred during hospitalization:

1. In-hospital mortality,
2. Admission to the intensive care unit (either directly from the emergency department or from the inpatient ward),
3. Requirement for mechanical ventilation (invasive or non-invasive).

Patients were categorized according to the presence or absence of the primary outcome.

2.6. Statistical Analysis: Demographic, clinical, laboratory, and radiological characteristics were summarized by using descriptive statistical methods appropriate to the nature and distribution of the data. Normally distributed continuous variables were reported as mean ± standard deviation (SD), whereas variables showing non-normal distributions were expressed as median values with interquartile ranges (IQRs). Categorical data were presented as absolute numbers and corresponding percentages.

Patients were compared according to the presence or absence of the primary outcome. For continuous variables, comparisons were conducted using the independent-samples t-test when the data followed a normal distribution and the Mann–Whitney U test when the distribution was non-normal. Categorical variables were analyzed through either the chi-square test or Fisher's exact test, as appropriate. All statistical tests were two-tailed, and a p value of <0.05 was regarded as indicating statistical significance.

The receiver operating characteristic (ROC) curve analysis was used to assess the ability of pulmonary artery diameter, ascending aortic diameter, and the pulmonary artery-to-aorta (PA/Ao) ratio to discriminate patients with poor prognosis. For each parameter, the area under the ROC curve (AUC) and its corresponding 95% confidence interval (CI) were calculated. The optimal threshold was established according to the Youden index, defined as the cut-off providing the most favorable combination of sensitivity and specificity. At the identified thresholds, sensitivity, specificity, positive predictive value, and negative predictive value were subsequently determined.

Potential predictors of poor prognosis were first examined via univariable logistic regression analysis. A multivariable logistic regression model was used for the variables with statistical significance in the univariable analyses along with variables considered clinically relevant. The findings of the regression analyses were expressed as odds ratios (ORs) with corresponding 95% CIs and p values.

The ability of the final multivariable logistic regression model to discriminate between patients with and without poor prognosis was further evaluated by ROC curve analysis. The model AUC and its corresponding 95% CI were calculated to quantify its overall discriminative performance.

All statistical analyses were conducted on complete cases. Missing data were not subjected to imputation. The statistical analyses were performed using appropriate statistical analysis software.

3. Results

3.1. Study Population: A total of 248 patients admitted to the hospital with community-acquired pneumonia (CAP) were initially assessed for eligibility. Among these patients, 16 were excluded because of confirmed COVID-19 infection, 8 because of pregnancy, 22 because of being minor, and 2 due to unavailability of the laboratory data. After these exclusions, the final study cohort consisted of 200 patients.

Among enrolled patients, 137 (68.5%) were male and 63 (31.5%) were female. The mean age was 66.42 ± 18.43 years, with a range of 18–97 years.

The primary outcome was defined as the occurrence of at least one adverse clinical event during hospitalization, including in-hospital death, admission to the intensive care unit (ICU) either directly from the emergency department or after transfer from the inpatient ward, or the need for mechanical ventilation, whether invasive or non-invasive. Based on this composite definition, 107 patients (53.5%) experienced a poor clinical outcome.

3.2. Comparison of Clinical, Laboratory, and Imaging Characteristics According to Prognosis: The study population was subsequently categorized into two groups according to whether the predefined primary outcome occurred. Patients with poor outcomes were older than those with favourable outcomes. The median age was 74.0 years (IQR: 62.0–81.5) in the poor prognosis group when compared with 66.0 years (IQR: 56.0–77.0) in the favourable prognosis group (p = 0.003).

Several laboratory parameters differed significantly between the groups. Serum lactate concentrations were higher among patients with poor outcomes, with a median value of 2.2 mmol/L (IQR: 1.5–3.7) when compared with 1.9 mmol/L (IQR: 1.2–2.4) among patients without the primary outcome (p = 0.002). Similarly, blood urea nitrogen (BUN) levels were significantly elevated in the poor prognosis group, with median values of 28.0 (IQR: 17.0–43.0) and 20.0 (IQR: 15.5–29.0), respectively (p < 0.001). However, no statistically significant difference was found on serum creatinine levels between the two groups (0.90 [IQR: 0.60–1.60] vs. 0.90 [IQR: 0.70–1.20], respectively; p = 0.415).

Oxygenation status also differed significantly according to clinical outcome. Patients who developed the primary outcome had lower oxygen saturation values than those with favourable outcomes. The median oxygen saturation was 89.0% (IQR: 82.0–94.0) in the poor prognosis group and 94.0% (IQR: 90.0–96.0) in the favorable prognosis group (p < 0.001).

A significant association was also observed between certain comorbid conditions and poor prognosis. Heart failure was present in 34.6% of patients with poor outcomes, compared with 16.1% of those without the primary outcome (p = 0.005). Malignancy was likewise more prevalent among patients with poor prognosis than among those with favourable outcomes (24.3% vs. 11.8%, p = 0.037).

The imaging parameters showed a similar numerical trend, with larger main pulmonary artery (PA) and ascending aortic (Ao) diameters, as well as a higher PA/Ao ratio, observed in the poor prognosis group. However, none of these differences demonstrated statistical significance (all p > 0.05). No statistically significant difference was detected between two groups in terms of white blood cell count, C-reactive protein concentration, creatinine level, heart rate, systolic or diastolic blood pressure, diabetes

mellitus, hypertension, coronary artery disease, chronic kidney disease, asthma, chronic obstructive pulmonary disease, or gender ($p > 0.05$ for all of these parameters).

Furthermore, patients with poor clinical outcomes were characterized by older age, higher serum BUN and lactate concentrations, lower oxygen saturation, and a greater prevalence of heart failure and malignancy. These variables showed significant differences in the unadjusted comparison between patients with poor and favourable prognoses.

Table 1. Comparison of demographic, clinical, laboratory, and imaging findings according to the primary outcome.

Variable	No Primary Outcome (n=93)	Primary Outcome Present (n=107)	*p* value
Demographic characteristics			
Age, median (IQR)	66.0 (56.0–77.0)	74.0 (62.0–81.5)	0.003
Male sex, n (%)	61 (65.6)	76 (71.0)	0.501
Medical history			
Diabetes mellitus, n (%)	27 (29.0)	32 (29.9)	1.000
Hypertension, n (%)	58 (62.4)	78 (72.9)	0.150
Coronary artery disease, n (%)	38 (40.9)	50 (46.7)	0.490
Heart failure, n (%)	15 (16.1)	37 (34.6)	0.005
Chronic kidney disease, n (%)	12 (12.9)	26 (24.3)	0.062
Asthma, n (%)	9 (9.7)	3 (2.8)	0.081
COPD, n (%)	29 (31.2)	38 (35.5)	0.619
Malignancy, n (%)	11 (11.8)	26 (24.3)	0.037
Vital signs			
Heart rate, median (IQR)	98.0 (83.0–113.0)	105.0 (90.0–119.0)	0.084
Systolic blood pressure, median (IQR)	126.0 (110.0–146.0)	120.0 (100.0–142.0)	0.098
Diastolic blood pressure, median (IQR)	70.0 (65.0–80.0)	70.0 (60.0–80.0)	0.276
Oxygen saturation, median (IQR), %	94.0 (90.0–96.0)	89.0 (82.0–94.0)	<0.001
Laboratory findings			
WBC, median (IQR)	13.07 (8.87–16.93)	11.80 (8.41–15.88)	0.499
CRP, median (IQR)	9.0 (3.8–17.1)	11.6 (5.42–17.85)	0.373
BUN, median (IQR)	20.0 (15.5–29.0)	28.0 (17.0–43.0)	<0.001
Creatinine, median (IQR)	0.90 (0.70–1.20)	0.90 (0.60–1.60)	0.415
Lactate, median (IQR)	1.9 (1.2–2.4)	2.2 (1.5–3.7)	0.002
CT-derived vascular measurements			
Main pulmonary artery diameter, median (IQR), cm	2.8 (2.5–3.2)	3.0 (2.65–3.35)	0.057
Ascending aortic diameter, mean ± SD, cm	3.41 ± 0.45	3.52 ± 0.45	0.089
PA/Ao ratio, median (IQR)	0.84 (0.76–0.93)	0.85 (0.77–0.95)	0.488

Data are presented as median (interquartile range [IQR]), mean ± standard deviation (SD), or number (percentage), as appropriate.

Continuous variables were analyzed through the independent-samples t test or the Mann–Whitney U test, depending on the distribution of the data. Comparisons were performed via the chi-square test or Fisher's exact test for cetagorical variables.

Abbreviations: *Ao, ascending aorta; BUN, blood urea nitrogen; COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein; PA, pulmonary artery; PA/Ao, pulmonary artery-to-ascending aorta diameter ratio; WBC, white blood cell count.*

3.3. Predictive Performance of CT-Derived Imaging Parameters: The prognostic performance of the CT-derived vascular measurements was assessed using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC), optimal cut-off values, sensitivity, specificity, and likelihood ratios were calculated to determine the ability of each imaging parameter to discriminate between patients with and without the primary outcome.

The ROC analysis yielded an AUC of 0.578 (95% CI: 0.499–0.657; p = 0.054) for main pulmonary artery diameter. A cut-off value of 3.0 cm provided a sensitivity of 54.2% and a specificity of 61.3%. At this threshold, the positive likelihood ratio (LR+) was 1.40, whereas the negative likelihood ratio (LR−) was 0.75.

Ascending aortic diameter demonstrated a slightly higher AUC of 0.583 (95% CI: 0.503–0.662; p = 0.041). Using 3.1 cm as the optimal cut-off, the sensitivity was 86.9%, while specificity was 28.0%. The corresponding LR+ and LR− values were 1.21 and 0.47, respectively.

The PA/Ao ratio showed the weakest discriminatory ability among the evaluated imaging parameters, with an AUC of 0.528 (95% CI: 0.447–0.608; p = 0.500). A cut-off of 0.94 resulted in a sensitivity of 27.1% and a specificity of 82.8%, with an LR+ of 1.58 and an LR− of 0.88.

Overall, all three imaging parameters demonstrated limited discriminatory performance for predicting the primary outcome. Although the AUC for ascending aortic diameter reached nominal statistical significance, the low specificity and modest overall discriminatory capacity limit its potential clinical usefulness as a standalone prognostic marker.

Visual inspection of the ROC curves demonstrated that all three parameters closely followed the diagonal reference line, indicating poor discriminative ability. The PA/Ao ratio exhibited performance close to chance, whereas PA and Ao diameters demonstrated only limited discriminatory value.

Although the AUC for ascending aortic diameter was statistically greater than 0.50, its absolute discriminatory performance remained poor.

These findings suggest that PA diameter, ascending aortic diameter, and the PA/Ao ratio are insufficient as standalone predictors of poor prognosis in patients with community-acquired pneumonia.

Table 2. ROC analysis of CT-derived vascular parameters for prediction of the primary outcome.

Parameter	AUC (95% CI)	*p* value*	Optimal cutoff	Sensitivity	Specificity	LR+	LR−
Main pulmonary artery diameter (PA)	0.578 (0.499–0.657)	0.054	3.0 cm	0.542	0.613	1.40	0.75
Ascending aortic diameter (Ao)	0.583 (0.503–0.662)	0.041	3.1 cm	0.869	0.280	1.21	0.47
PA/Ao ratio	0.528 (0.447–0.608)	0.500	0.94	0.271	0.828	1.58	0.88

AUC values are presented with asymptotic 95% confidence intervals. Optimal cut-off values were determined by using the Youden index.

**The reported p value tests the null hypothesis that the true AUC equals 0.50.*

Abbreviations: AUC, area under the receiver operating characteristic curve; CI, confidence interval; LR+, positive likelihood ratio; LR−, negative likelihood ratio; PA, pulmonary artery; Ao, ascending aorta; PA/Ao, pulmonary artery-to-ascending aorta diameter ratio.

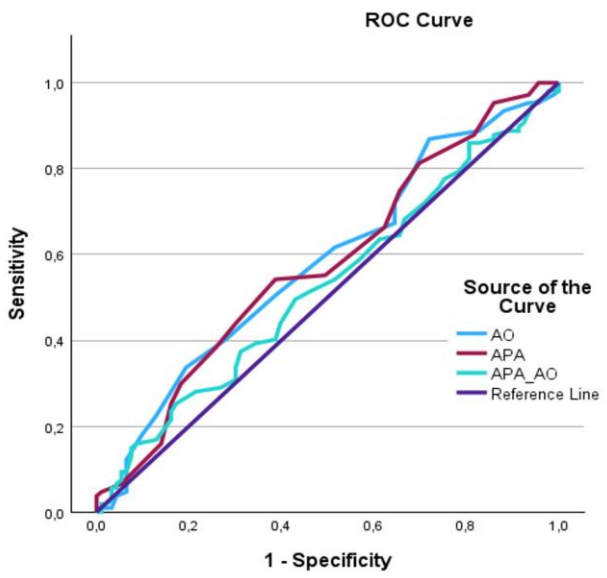

Figure 1. ROC Graph for the primary outcome.

3.4. Univariable Logistic Regression Analysis: Univariable logistic regression analysis was performed to identify variables associated with poor prognosis.

Increasing age was significantly associated with poor prognosis (OR 1.022, 95% CI 1.006–1.038, $p = 0.007$). Elevated lactate levels were also associated with an increased likelihood of poor prognosis (OR 1.326, 95% CI 1.099–1.600, $p = 0.003$).

However, higher oxygen saturation was associated with a lower risk of poor prognosis (OR 0.908, 95% CI 0.868–0.950, $p < 0.001$).

Among comorbidities, heart failure was significantly associated with poor prognosis (OR 2.466, 95% CI 1.259–4.830, $p = 0.008$), while malignancy demonstrated borderline statistical significance (OR 2.132, 95% CI 1.000–4.545, $p = 0.050$).

Higher BUN levels were significantly associated with the primary outcome (OR 1.038 per one-unit increase, 95% CI 1.017–1.059, $p < 0.001$). Creatinine also demonstrated a marginal but statistically significant association with the primary outcome in univariable logistic regression (OR 1.359 per one-unit increase, 95% CI 1.001–1.845, $p = 0.049$). Female gender (OR 0.778, 95% CI 0.428–1.414, $p = 0.409$) and COPD (OR 1.215, 95% CI 0.673–2.195, $p = 0.518$) were not significantly associated with the primary outcome.

Regarding imaging parameters, main pulmonary artery diameter showed a borderline association with poor prognosis (OR 1.866, 95% CI 0.992–3.512, $p = 0.053$). In contrast, ascending aortic diameter and PA/Ao ratio were not significantly associated with the primary outcome.

Table 3. Univariable logistic regression analysis for the primary outcome.

Predictor	OR	95% CI	*p* value
Age, per 1-year increase	1.022	1.006–1.038	0.007
Female sex	0.778	0.428–1.414	0.409
Heart failure	2.466	1.259–4.830	0.008
Malignancy	2.132	1.000–4.545	0.050
COPD	1.215	0.673–2.195	0.518
BUN, per 1-unit increase	1.038	1.017–1.059	<0.001
Creatinine, per 1-unit increase	1.359	1.001–1.845	0.049
Lactate, per 1-unit increase	1.326	1.099–1.600	0.003
Oxygen saturation, per 1-percentage-point increase	0.908	0.868–0.950	<0.001
Main pulmonary artery diameter, per 1-unit increase	1.866	0.992–3.512	0.053
Ascending aortic diameter, per 1-unit increase	1.639	0.877–3.061	0.121
PA/Ao ratio, per 1-unit increase	1.467	0.201–10.724	0.706

ORs for continuous variables represent the change in odds associated with a one-unit increase in the respective predictor. Male gender was the reference category; therefore, the reported OR represents female versus male. The absence of the respective condition was the reference category for heart failure, malignancy, and COPD. Abbreviations: Ao, ascending aorta; BUN, blood urea nitrogen; CI, confidence interval; COPD, chronic obstructive pulmonary disease; OR, odds ratio; PA, pulmonary artery; PA/Ao, pulmonary artery-to-ascending aorta diameter ratio.

3.5. Multivariable Logistic Regression Analysis: Three separate multivariable logistic regression models were utilized in order to determine whether the CT-based vascular measurements were independently related to the primary outcome, while minimizing the potential effect of collinearity between the imaging variables. The same clinically selected covariates including the age, gender, heart failure, malignancy, hypertension, chronic obstructive pulmonary disease (COPD), oxygen saturation, blood urea nitrogen (BUN), and lactate were included into each model. Main pulmonary artery (PA) diameter, ascending aortic (Ao) diameter, and the PA/Ao ratio were subsequently evaluated individually as the principal imaging variable in Models 1, 2, and 3, respectively.

After adjustment for the prespecified covariates, none of the CT-derived vascular parameters demonstrated an independent association with the primary outcome. In Model 1, main PA diameter was not significantly associated with poor prognosis (adjusted odds ratio [aOR] 1.301, 95% confidence interval [CI] 0.594–2.850, $p = 0.511$). Similarly, ascending Ao diameter showed no independent association with the primary outcome in Model 2 (aOR 0.938, 95% CI 0.372–2.366, $p = 0.892$). The PA/Ao ratio also failed to reach statistical significance in Model 3 (aOR 3.548, 95% CI 0.257–48.956, $p = 0.344$).

Among the clinical covariates, heart failure consistently demonstrated an independent association with poor prognosis across all three models, with aORs ranging from 2.645 to 2.803 (all $p < 0.05$). In contrast, higher oxygen saturation was associated with a lower likelihood of experiencing the primary outcome in each model, with an aOR of 0.896–0.897 for each one-percentage-point increase (all $p < 0.001$).

BUN remained independently associated with the primary outcome in all models, with aORs ranging from 1.027 to 1.028 for each one-unit increase ($p = 0.017$–0.018). Lactate showed a similar independent association, with aORs ranging from 1.326 to 1.354 per one-unit increase ($p = 0.006$–0.012).

However, age, gender, malignancy, hypertension, and COPD did not demonstrate statistically significant independent associations with poor prognosis in any of the adjusted models.

Table 4. Multivariable logistic regression analyses for the primary outcome using separate models for CT-derived vascular measurements.

4. Discussion

In the present study, we investigated whether thoracic computed tomography (CT)-derived measurements of the main pulmonary artery (PA) diameter, ascending aortic (Ao) diameter, and PA-to-Ao diameter ratio could predict poor clinical outcomes in patients hospitalized due to community-acquired pneumonia (CAP). The principal finding was that none of these vascular parameters demonstrated a statistically significant independent association with the composite adverse outcome. This result contrasts with observations from several disease-specific populations in which pulmonary vascular measurements have shown prognostic value. In our cohort, clinical and physiological indicators of disease severity at presentation, particularly impaired oxygenation, evidence of tissue hypoperfusion, and cardiac comorbidity, appeared to be more relevant determinants of an unfavourable clinical course.

Previous studies have reported associations between pulmonary artery enlargement or an increased PA/Ao ratio and adverse outcomes,

Table 4. Multivariable logistic regression analyses for the primary outcome using separate models for CT-derived vascular measurements.

Predictor	Model 1: PA diameter aOR (95% CI)	*p* value	Model 2: Ao diameter aOR (95% CI)	*p* value	Model 3: PA/Ao ratio aOR (95% CI)	*p* value
Age, per 1-year increase	0.991 (0.967–1.015)	0.442	0.992 (0.966–1.020)	0.580	0.995 (0.970–1.020)	0.679
Female sex	0.807 (0.382–1.704)	0.574	0.828 (0.392–1.750)	0.622	0.769 (0.359–1.648)	0.500
Heart failure	2.645 (1.113–6.284)	0.028	2.803 (1.192–6.590)	0.018	2.651 (1.123–6.257)	0.026
Malignancy	2.156 (0.885–5.255)	0.091	2.157 (0.885–5.259)	0.091	2.148 (0.882–5.232)	0.092
Hypertension	1.236 (0.551–2.776)	0.607	1.230 (0.549–2.756)	0.614	1.249 (0.554–2.816)	0.591
COPD	0.826 (0.372–1.834)	0.639	0.865 (0.394–1.899)	0.717	0.831 (0.376–1.835)	0.647
Oxygen saturation, per 1-percentage-point increase	0.897 (0.852–0.944)	<0.001	0.896 (0.851–0.943)	<0.001	0.897 (0.852–0.944)	<0.001
BUN, per 1-unit increase	1.028 (1.005–1.051)	0.017	1.027 (1.005–1.051)	0.018	1.027 (1.005–1.051)	0.018
Lactate, per 1-unit increase	1.338 (1.075–1.664)	0.009	1.354 (1.089–1.683)	0.006	1.326 (1.063–1.653)	0.012
Main PA diameter, per 1-unit increase	1.301 (0.594–2.850)	0.511	—	—	—	—
Ascending Ao diameter, per 1-unit increase	—	—	0.938 (0.372–2.366)	0.892	—	—
PA/Ao ratio, per 1-unit increase	—	—	—	—	3.548 (0.257–48.956)	0.344

Each model used an identical clinically defined adjustment set comprising age, gender, heart failure, malignancy, hypertension, COPD, oxygen saturation, BUN, and lactate, with only the CT-derived vascular parameter varying between models.

Male gender was the reference category for gender, while absence of the respective condition was the reference category for categorical comorbidities.

Abbreviations: Ao, ascending aorta; aOR, adjusted odds ratio; BUN, blood urea nitrogen; CI, confidence interval; COPD, chronic obstructive pulmonary disease; PA, pulmonary artery; PA/Ao, pulmonary artery-to-ascending aorta diameter ratio.

including mortality and intensive care unit admission, in patients with acute exacerbations of chronic obstructive pulmonary disease (COPD), pulmonary embolism, idiopathic pulmonary fibrosis, and COVID-19 pneumonia [6,10,11]. Particularly, an elevated PA/Ao ratio has been proposed as a marker of unfavourable prognosis for patients with COVID-19. The absence of a similar relationship in our CAP population may be attributable, at least in part, to differences in the underlying pathophysiology and temporal characteristics of these conditions.

One possible explanation for our findings is the distinction between acute and chronic pulmonary vascular changes. Enlargement of the pulmonary artery is generally associated with structural vascular remodelling caused by persistent elevations in pulmonary vascular resistance and pulmonary arterial pressure, as observed in chronic pulmonary diseases and pulmonary hypertension [12]. However, CAP is an acute infectious and inflammatory condition. Although acute hypoxemia may trigger hypoxic pulmonary vasoconstriction, this physiological response is generally transient. The limited duration of pulmonary vascular stress during acute pneumonia may therefore be insufficient to induce structural remodelling that would produce readily measurable changes in vascular dimensions on CT. Consequently, short-term physiological disturbances may not necessarily

result in anatomical alterations with sufficient magnitude to serve as reliable prognostic markers.

The findings concerning heart failure and oxygen saturation provide further context for the lack of prognostic significance of the CT-derived vascular parameters. Heart failure is a well-established contributor to secondary pulmonary hypertension and elevated pulmonary arterial pressure [13]. Therefore, an increased pulmonary artery diameter in patients with CAP may reflect underlying cardiovascular disease or pre-existing pulmonary hemodynamic abnormalities rather than changes directly attributable to the acute infectious process. In this setting, the clinical course may be more strongly influenced by impaired oxygenation, inadequate tissue oxygen delivery, systemic hypoperfusion, and subsequent organ dysfunction than by relatively subtle changes in pulmonary vascular anatomy. Our findings also support the current approach to severity assessment in CAP. Contemporary recommendations emphasize a multidimensional evaluation of disease severity using validated clinical prediction instruments, such as CURB-65 and the Pneumonia Severity Index (PSI), rather than relying on a single radiological or laboratory parameter [14,15]. Furthermore, the vascular measurements assessed in our study did not provide sufficient additional independent prognostic information to justify their use as isolated markers of disease severity. Therefore, the PA diameter, Ao diameter, and PA/Ao ratio should not be regarded as alternatives to established clinical severity scores or used independently for triage and risk stratification in hospitalized patients with CAP.

Several clinical variables identified in our analysis may be more directly related to the mechanisms leading to adverse outcomes. Reduced oxygen saturation may indicate significant impairment of pulmonary gas exchange and consequently inadequate tissue oxygenation. Similarly, elevated lactate and BUN levels may reflect systemic hypoperfusion, impaired physiological reserve, or the development of organ dysfunction. The persistent association of heart failure with poor prognosis further emphasizes the contribution of underlying cardiovascular reserve to outcomes in patients hospitalized with CAP.

Consequently, our findings suggest that the PA diameter, ascending Ao diameter, and PA/Ao ratio measured on admission thoracic CT have limited prognostic utility in hospitalized patients with CAP. Unlike chronic pulmonary vascular disorders, the acute nature of pneumonia may not provide sufficient time for structural pulmonary vascular remodelling to develop to a degree that can be reliably detected by CT. Accordingly, these anatomical measurements appear to have limited value as standalone prognostic biomarkers in this clinical setting.

A clinical perspective reveals that prognostic assessment in hospitalized patients with CAP should continue to rely primarily on an integrated evaluation of clinical presentation, physiological abnormalities, and relevant comorbidities. Parameters reflecting oxygenation, tissue perfusion, and cardiovascular reserve may provide more clinically meaningful prognostic information than isolated measurements of pulmonary vascular anatomy. Further prospective studies involving larger and more diverse CAP populations may help determine whether specific CT-derived vascular measurements have prognostic value in selected patient subgroups or under clinical circumstances.

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All procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Due to the retrospective design of the study, the requirement for informed consent was waived by the ethics committee. All patient data were anonymized prior to analysis.

Data Availability Statement: *The datasets generated and/or analyzed during the current study are available from the corresponding author, Muhammet Ali Erinmez, upon reasonable request.*

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