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Research Article

FACTORS LEADING TO ELEVATED CARDIAC BIOMARKERS IN SEVERE COMMUNITY-ACQUIRED PNEUMONIA

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ABSTRACT

Objective: Community-acquired pneumonia (CAP) is a significant public health issue with high morbidity and mortality rates. This study aims to evaluate the risk factors contributing to the increase in cardiac biomarkers in patients with severe CAP.

Materials and Methods: The study has a retrospective and cross-sectional design. A total of 70 patients diagnosed with CAP and followed in the Pulmonology Department and Intensive Care Unit of Aydın Adnan Menderes University between September 2015 and February 2020 were included in the study. Demographic data, comorbidities, vital signs, laboratory results, and cardiac biomarker levels (troponin, Pro-BNP, CK-MB) were recorded in detail, and comparisons were made between subgroups.

Results: Of the patients, 68.6% were male, with a mean age of 73.05 ± 13.77 years. At least one comorbidity was present in 92.8% of the patients, with hypertension, COPD, and diabetes mellitus being the most frequently observed. CK-MB levels showed significant positive correlations with CURB-65 and Pneumonia Severity Index (PSI) scores. Troponin levels were significantly elevated in patients with impaired consciousness, Pro-BNP levels in those who developed arrhythmias, and CK-MB levels in patients with COPD. Additionally, LDH levels correlated positively with both troponin and Pro-BNP levels.

Conclusion: This study highlights the importance of evaluating cardiac biomarkers in patients with severe CAP. Elevated levels of troponin, Pro-BNP, and CK-MB are reflective of increased cardiovascular risk and infection severity. These findings underscore the need for early and thorough monitoring of these biomarkers to identify and manage potential complications effectively, thereby improving patient outcomes.

Keywords: Cardiac biomarkers, CK-MB, Community-acquired pneumonia, CURB-65, Troponin, Pro-BNP, PSI.

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INTRODUCTION

Community-acquired pneumonia (CAP) is defined as pneumonia that develops in individuals without a known immunodeficiency during their daily life (1). Despite advancements in vaccination policies and antibiotic treatments, CAP remains a significant public health concern with high morbidity and mortality rates (2,3). Scoring systems such as the Pneumonia Severity Index (PSI) and CURB-65 are widely used to assess disease severity, guide hospitalization decisions, and predict mortality in these patients (4). While PSI evaluates 20 variables related to the patient, CURB-65 is a more practical assessment tool that includes five parameters: age, blood urea nitrogen (BUN), respiratory rate, altered mental status, and systolic blood pressure (5,6). Patients with a PSI class IV or V or a CURB-65 score $\geqslant 2$ are generally recommended for inpatient treatment. Although pneumonia-related mortality is a major concern, cardiovascular complications such as myocardial infarction, arrhythmias, and acute heart failure significantly contribute to adverse outcomes in these patients (7). Studies have shown that elevated levels of cardiac biomarkers, particularly cardiac troponins (cTn) and B-type natriuretic peptide (BNP), are associated with increased mortality in patients with CAP, independent of preexisting coronary artery disease (8–10). Elevated cardiac troponins, including high-sensitivity troponin T (cTnT) and troponin I (cTnI), have been identified as strong predictors of both short- and long-term mortality in hospitalized CAP patients, potentially reflecting acute myocardial stress or direct cardiac injury due to systemic inflammation (8,9). Similarly, BNP levels have been found to correlate with pneumonia severity, pulmonary hypertension, and cardiac dysfunction, further aiding in risk stratification (10). Given the increasing recognition of cardiovascular complications in CAP patients, understanding the factors contributing to cardiac biomarker elevation is crucial for improving risk assessment and patient management. This study aims to investigate the risk factors contributing to the elevation of cardiac biomarkers in patients with severe community-acquired pneumonia.

MATERIALS AND METHODS

This study has a retrospective and cross-sectional design. A total of 70 patients diagnosed with community-acquired pneumonia, who were followed in the Pulmonology Department or Intensive Care Unit of Aydın Adnan Menderes University between September 2015 and February 2020, were included in the study. Community-acquired pneumonia (CAP) was defined as pneumonia acquired outside of a healthcare setting in individuals without recent hospitalization or significant immunosuppression (1). The diagnosis of CAP was based on clinical symptoms, including fever, cough, sputum production, and dyspnea, along with radiological findings such as new pulmonary infiltrates observed on chest X-ray or computed tomography, and laboratory markers indicative of infection, in accordance with established clinical guidelines (11). Immunosuppression was defined as a condition or treatment that significantly compromises immune function. This included patients with HIV infection and a CD4 count below 200/mm^3, those who had undergone solid organ or hematopoietic stem cell transplantation, individuals with active malignancy receiving chemotherapy or radiotherapy, and those on long-term immunosuppressive therapy. Immunosuppressive treatments were considered to include corticosteroids at a dose of 20 mg per day or more for longer than two weeks, biologic agents, or disease-modifying antirheumatic drugs (12). Patients with a history of hospitalization or antibiotic use within the 14 days prior to admission, those with another focus of infection, and immunosuppressed patients were excluded.

The demographic characteristics of the patients (age, gender, smoking history), known comorbidities, vital signs at admission (temperature, level of consciousness, blood pressure, heart rate, respiratory rate), blood gas analyses [partial carbon dioxide pressure (pCO_2), partial oxygen pressure (pO_2), oxygen saturation (SaO_2), and pH], complete blood count parameters (hemoglobin, hematocrit, neutrophils, lymphocytes, platelets), and biochemistry results (sodium, albumin, lactate dehydrogenase, urea) were recorded in detail. Additionally, troponin-I, CK-MB, pro-BNP, CRP, procalcitonin, and D-dimer levels were also analyzed.

The cases included in the study were divided into various subgroups, and cardiac biomarker levels were compared among these groups. The subgroups were defined according to the following criteria: CURB-65 score (2, 3, and 4); Pneumonia Severity Index (PSI) class (Group IV and Group V); level of consciousness (conscious and unconscious); comorbidity status [presence or absence of chronic obstructive pulmonary disease (COPD)]; and cardiovascular complication status (patients who developed complications within the first 7 days of hospitalization and those who did not).

This study was approved by the Non-Interventional Clinical Research Ethics Committee of Aydın Adnan Menderes University Faculty of Medicine with the decision dated April 27, 2020, and numbered 18.

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Statistical analysis

Statistical evaluation was performed using SPSS software (SPSS 23, IBM Corp. Released 2015. IBM SPSS Statistics for Windows, Version 23.0, Armonk, NY, USA). Categorical variables were expressed as numbers and percentages. The conformity of variables to a normal distribution was determined using the Kolmogorov-Smirnov test. For continuous variables, the Student's t-test was used to compare two groups when normal distribution was observed, and the One-Way ANOVA test was applied for comparisons involving more than two groups. For variables not following a normal distribution, the Mann-Whitney U test was used for two-group comparisons, and the Kruskal-Wallis variance analysis was applied for comparisons of more than two groups. Continuous variables not following a normal distribution were presented as medians (25th–75th percentiles), while those with a normal distribution were expressed as mean ± standard deviation. The relationships between categorical variables were evaluated using Pearson's chi-square test and Fisher's exact test. Spearman and Pearson correlation analyses were used to analyze relationships between variables. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 70 patients diagnosed with community-acquired pneumonia, followed in the ward or intensive care unit between September 2015 and February 2020, were included in the study. Of the patients, 68.6% (n=48) were male, and 31.4% (n=22) were female. The age range varied from 40 to 97 years, with a mean age of 73.05 ± 13.77 years. Among the patients, 68.6% (n=48) were either active smokers or former smokers (ex-smokers), while 31.4% (n=22) had never smoked. At least one comorbidity was identified in 92.8% (n=65) of the patients. The most common comorbidities were hypertension (44.3%, n=31), COPD (35.7%, n=25), and diabetes mellitus (25.7%, n=18). Among patients with a history of cardiovascular disease, 44.3% (n=31) had hypertension, 24.3% (n=17) had coronary artery disease, 15.7% (n=11) had congestive heart failure, 8.6% (n=6) had arrhythmia, and 1.4% (n=1) had a history of myocardial infarction (MI). The demographic characteristics and comorbidity distribution of the patients are presented in Table 1.

According to the vital signs assessed at the time of hospitalization, the majority of the patients were conscious (91.4%). In the evaluation of body temperature, 48.6% of the patients had a normal temperature, while 27.1% were febrile (>38°C). Blood pressure measurements revealed normal values in 62.9% of the patients, hypotension (<90/60 mmHg) in 8.6%, and hypertension (>140/90 mmHg) in 28.6%. Tachycardia (>100 bpm) was observed in 45.8% of the patients. Detailed vital signs of the patients are presented in Table 2.

Table 1. Demographic characteristics and comorbidities of patients

Age, Mean ± SD	73.05 ±13.77
Gender n (%)	
Female	22 (31.4)
Male	48 (68.6)
Smoking status n (%)	
Smoker	48 (68.6)
Non-Smoker	22 (31)
Cardiovascular Comorbidity n (%)	
Present	45 (64.2)
Absent	25 (35.7)
Hypertension n (%)	
Present	31 (44.3)
Absent	39 (55.7)
Arrhythmia n (%)	
Present	6 (8.6)
Absent	64 (91.4)
Coronary Artery Disease n (%)	
Present	17 (24.3)
Absent	53 (75.7)
History of Myocardial Infarction n (%)	
Present	1 (1.4)
Absent	69 (98.6)
Congestive Heart Failure n (%)	
Present	11 (15.7)
Absent	59 (84.2)
Non-Cardiovascular Comorbidity n (%)	
Present	58 (82.9)
Absent	12 (17.1)

Table 2. Vital signs of patients

		n	%
Consciousness	Normal	64	91.4
	Impaired	6	8.6
Fever (°C)	Normal	349	48.6
	Subfebrile (37.2°C -38°C)	17	24.3
	Febrile (>38)	19	27.1
Blood Pressure (mmHg)	Normal	44	62.9
	Hypotensive (<90/60 mmHg)	6	8.6
	Hypertensive (>140/90 mmHg)	20	28.6
Pulse (/min)	Bradycardia (<60)	2	2.9
	Normal (60-100)	36	51.4
	Tachycardia (>100)	32	45.8
Tachypnea (/min)	Present (RR>24 /min)	5	7.1
	Absent	65	92.9

RR: Respiratory Rate, mmHg: Millimeters of Mercury, /min: Per Minute

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Laboratory results showed that the median urea level was 52.5 mg/dL (25th–75th percentile: 38.0–68.0). Among inflammatory markers, the mean CRP level was 168.98 ± 91.01 mg/L, and the median procalcitonin level was 0.60 ng/mL (25th–75th percentile: 0.21–4.82). In the blood gas analysis, the median pH was 7.45 (25th–75th percentile: 7.39–7.47), and the mean oxygen saturation was 87.62 ± 5.52%. The mean PSI score, which assesses disease severity, was calculated as 116.73 ± 39.42, while the median CURB-65 score was 2.0 (25th–75th percentile: 1.0–2.0). Details of these parameters are presented in Table 3.

Table 3. Laboratory and clinical characteristics of patients

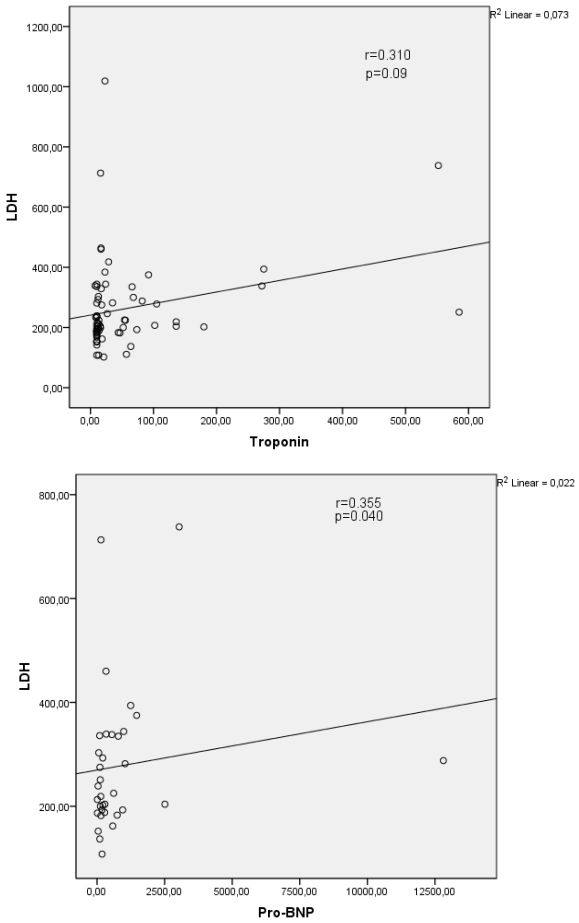
Urea, mg/dL median (25%-75%)	52.50 (38.0-68.0)
LDH, mg/dL median (25%-75%)	213.50 (186.25-300.75)
Albumin, mg/dL mean ± SD	3.10 ± 0.45
Sodium, mEq/L mean ± SD	136.54 ±5.32
CRP, mg/L mean ± SD	168.98 ± 91.01
Procalcitonin, ng/mL median (25%-75%)	0.60 (0.21- 4.82)
D-Dimer, mg/L, mg/L median (25%-75%)	1609.0 (912.50-2620.00)
Neutrophil %, median (25%-75%)	87.00 (80.37-90.00)
Neutrophil count, 10^3/cells/µL (25%-75%)	12000 (7780.0-17097.50)
Lymphocyte %, median (25%-75%)	7.25 (4.150-10.925)
Lymphocyte count, cells/µL median (25%-75%)	955.0 (725.0-1420.0)
Platelet count, platelets/µL mean ± SD	265828.57 ± 1.01
pH, median (25%-75%)	7.450 (7.390-7.470)
PaO2, mmHg median (25%-75%)	53.60 (49.00-60.00)
SpO_2, % mean ± SD	87.62 ± 5.516
PSI, mean ± SD	116.728 ±39.424
CURB-65, median (25%-75%)	2.00 (1.00-2.00)

Continuous variables are presented as mean ± standard deviation or median (25th–75th percentiles), depending on their distribution. LDH: Lactate Dehydrogenase, CRP: C-Reactive Protein, PaO_2: Partial Pressure of Oxygen, SpO_2: Peripheral Capillary Oxygen Saturation, PSI: Pneumonia Severity Index, CURB-65: Confusion, Urea, Respiratory Rate, Blood Pressure, Age ≥65

Significant differences were observed in troponin, Pro-BNP, and CK-MB levels. Troponin levels were significantly higher in patients with impaired consciousness (median: 123.00 ng/L; 25th–75th percentile: 24.00–552.00) compared to those with normal consciousness (median: 13.45 ng/L; 25th–75th percentile: 10.00–45.40) ($p<0.05$). Pro-BNP levels were significantly higher in patients who developed arrhythmias within 7 days (median: 784.00 ng/L; 25th–75th percentile: 549.00–985.00) compared to those without arrhythmias (median: 185.00 ng/L; 25th–75th percentile: 100.00–580.00) (p=0.011). Similarly, CK-MB levels were significantly higher in patients with COPD (median: 2.20 ng/mL; 25th–75th percentile: 1.05–3.95) compared to those without COPD (median: 1.05 ng/mL; 25th–75th percentile: 0.70–1.974) (p=0.031). In the PSI-5 group, CK-MB levels (median: 1.80 ng/mL; 25th–75th percentile: 0.85–3.70) were significantly higher than those in the PSI-4 group (median: 1.10 ng/mL; 25th–75th percentile: 0.90–2.80) (p=0.038). Details of these findings are presented in Table 4.

In our study, LDH levels were positively correlated with troponin levels (r=0.310; p=0.09) and Pro-BNP levels (r=0.355; p=0.040) (Figure 1). Additionally, CK-MB levels showed a significant negative correlation with lymphocyte count (r=-0.303; p=0.012) and a significant positive correlation with the CURB-65 score (r=0.234; p=0.012) and the PSI score (r=0.250; p=0.039), which assess pneumonia severity (Figure 2).

Figure 1. Correlation Between LDH Levels and Troponin and Pro-BNP Levels



LDH: Lactate Dehydrogenase, Pro-BNP: Pro-Brain Natriuretic Peptide

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Table 4. Comparison of Troponin, Pro-BNP, and CK-MB levels according to clinical parameters

Groups	n (%)	Troponin (ng/ml)	p	Pro-BNP (pg/mL)	p	CK-MB (IU/L)	p
COPD present	25 (35.7%)	12.00 (10.00-48.00)	0.166	234.00 (57.225-597.00)	0.234	2.20 (1.05-3.95)	0.031
COPD absent	45 (64.3%)	16.70 (10.00-65.00)		272.00 (132.500-1089.25)		1.05 (0.70-1.974)	
Conscious	64 (91.4%)	13.45 (10.00-45.40)	0.005	234.00 (73.450-581.00)	0.423	1.20 (0.80-2.30)	0.201
Unconscious	6 (8.6%)	123.00 (24.00-552.00)		272.00 (137.00-1038.00)		2.60 (1.10-4.20)	
Arrhythmia within 7 days	10 (14.3%)	30.35 (10.00-73.50)	0.347	784.00 (549.00-985.00)	0.011	1.95 (1.10-4.40)	0.133
No arrhythmia within 7 days	60 (85.7%)	13.55 (10.00-49.40)		185.00 (100.00-580.00)		1.20 (0.80-2.30)	
PSI 4	27 (38.6)	13.00 (10.00-26.60)	0.163	186.00 (100.45-891.50)	0.908	1.10 (0.90-2.80)	0.038
PSI 5	25 (35.7)	23.00 (10.00-77.75)		289.00 (119.00-784.00)		1.80 (0.85-3.70)	
CURB-65 2	34 (48.6)	16.50 (10.00%-54.75%)	0.566	313.50 (101.475%-975.675%)	0.950	1.20 (0.80%-2.425%)	0.067
CURB-65 3	11 (15.7)	12.00 (10.00%-272.00%)		211.00 (64.50%-1793.50%)		1.70 (1.075%-8.025%)	
CURB-65 4	4 (5.7)	59.00 (20.50%-151.50%)		213.00 (121.00%-784.00%)		3.45(2.175%-12.30%)	

Comparisons between groups were performed using Student's t-test or Mann-Whitney U test for two-group comparisons and One-Way ANOVA or Kruskal-Wallis test for multiple-group comparisons. COPD: Chronic Obstructive Pulmonary Disease, PSI: Pneumonia Severity Index, CURB-65: Confusion, Urea, Respiratory Rate, Blood Pressure, Age ≥65. ***Note:*** *Patients with a CURB-65 score of 1 and a PSI score lower than 4 or 5 were hospitalized due to hypoxemia.*

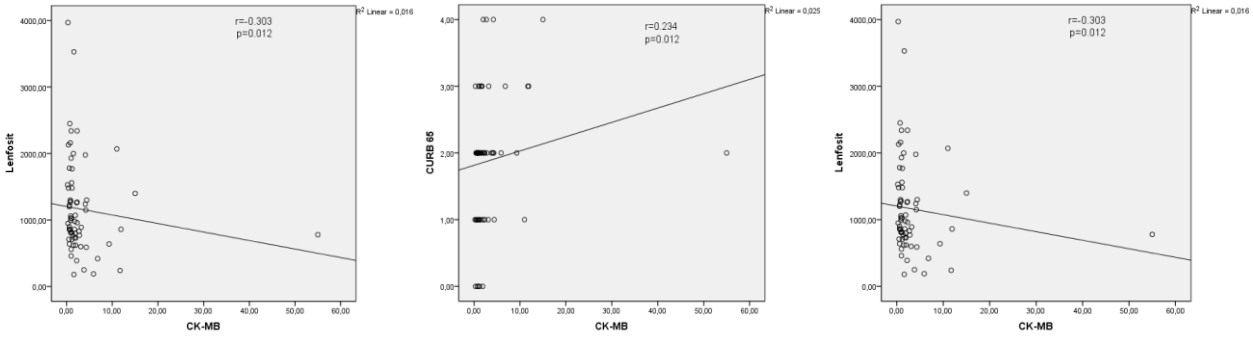

Figure 2. Correlation Between CK-MB Levels and Lymphocyte Count, CURB-65, and PSI Scores
CK-MB: Creatine Kinase-MB, CURB-65: Confusion, Urea, Respiratory Rate, Blood Pressure, Age ⩾65, PSI: Pneumonia Severity Index

DISCUSSION

CAP is a common infectious disease worldwide, with high morbidity and mortality rates. The incidence of cardiovascular complications is also increased in patients who develop pneumonia, and the development of cardiovascular complications during follow-up further elevates the risk of mortality (13-15). In our study, troponin levels were significantly elevated in patients with impaired consciousness and severe pneumonia. Impaired consciousness is considered a marker of severe infection and systemic inflammation and is often associated with multiple organ dysfunction (16,17). Proinflammatory cytokines released during infection can cause direct myocardial cell damage and increase stress on the heart (18). Additionally, hypoxia in patients with impaired consciousness and severe pneumonia may create an environment where myocardial oxygen demand cannot be met, triggering subclinical myocardial ischemia (19). These patients often exhibit hemodynamic instability and increased sympathetic nervous system activity. This condition may increase the burden on the heart, contributing to elevated troponin levels (20,21). Therefore, impaired consciousness is an important indicator not only of the infection's severity but also of the risk of cardiovascular complications. Early evaluation of cardiac biomarkers in patients with impaired consciousness is critical for the prevention and management of cardiovascular complications. Our study suggests that troponin levels may serve as a marker of cardiovascular risk in this patient group and emphasizes the need for larger-scale, prospective studies to validate these findings.

In our study, baseline Pro-BNP levels were significantly elevated in patients who developed arrhythmias within 7 days after pneumonia. This finding suggests that Pro-BNP could be an important biomarker for the early prediction

of cardiovascular complications following pneumonia. Similarly, the study by Mendenez et al. demonstrated that baseline Pro-BNP levels were elevated in patients who developed cardiovascular events in the early period following pneumonia (14). This supports the prognostic value of Pro-BNP in cardiovascular risk assessment following infection. In a study conducted by Mojón-Álvarez et al. on COVID-19 patients, Pro-BNP levels were reported to be higher, particularly in those who developed myocardial damage or hemodynamic instability (22). This finding illustrates that increased systemic inflammation and hemodynamic stress during infection trigger Pro-BNP release. Our study further highlights that Pro-BNP is a valuable biomarker for detecting cardiovascular complications in the early period following infection. However, it should be noted that Pro-BNP levels vary across different patient populations and types of infections in the existing literature. Therefore, caution should be exercised in the clinical use and interpretation of Pro-BNP, and further studies are needed in diverse patient groups.

Although CK-MB is commonly used as a biomarker for myocardial damage, its sensitivity and specificity are known to be limited. The literature shows that CK-MB levels can also be elevated in conditions such as trauma, rhabdomyolysis, renal failure, and pulmonary infections (23-25). In the study by Fan et al., CK-MB levels were significantly elevated in patients with Mycoplasma pneumoniae pneumonia, and this was associated with the increased inflammatory response and myocardial stress during infection (26). Similarly, in Seedat et al.'s study, CK-MB levels were reported to increase due to inflammation and hypoxia in patients with severe pneumonia, and this increase was linked to cardiovascular complications (27). In our study, CK-MB levels were significantly higher in CAP patients with COPD compared to those without COPD. Additionally, a positive correlation was identified between CK-MB levels and CURB-65 and PSI scores. These findings suggest that CK-MB levels in severe pneumonia cases reflect not only myocardial damage but also the severity of the infection and the inflammatory response. The literature supports the results obtained in our study, indicating that CK-MB may be an important parameter for cardiovascular risk assessment in patients with severe pneumonia. However, it should be noted that the specificity of CK-MB is limited in clinical practice, and it should be evaluated in conjunction with other biomarkers.

LDH is an enzyme widely present in tissues and is considered a systemic marker of inflammation. In the study by Yamaguchi et al., LDH was found to represent myocardial damage and has prognostic value in acute decompensated heart failure (28). In our study, a positive correlation was also identified between LDH and troponin and Pro-BNP levels. This finding suggests that LDH may assist in determining the risk of cardiac complications in pneumonia patients.

COPD is an independent risk factor for cardiovascular diseases, and elevated levels of Pro-BNP and troponin have been reported in these patients (29,30). In our study, CK-MB levels were found to be significantly higher in CAP patients with COPD; however, no significant differences were observed in troponin and Pro-BNP levels. This finding suggests that cardiac biomarkers should be carefully monitored in pneumonia patients with COPD and highlights the need for further studies in this patient group.

The retrospective design of our study and the limited number of patients are the primary limitations affecting the generalizability of these findings. Nevertheless, these results highlight the critical importance of carefully evaluating cardiac biomarkers in patients with severe pneumonia for the early detection and management of cardiovascular complications. It should be noted, however, that the specificity of biomarkers is limited in clinical practice, and they should be evaluated in conjunction with other clinical parameters. Validation of these findings through larger-scale, prospective studies will enhance the understanding and management of cardiovascular risks following infection.

CONCLUSION

Our study demonstrated that the increase in cardiac biomarker levels in patients with severe community-acquired pneumonia reflects both the severity of the infection and the risk of cardiovascular complications. The significantly elevated troponin levels in patients with impaired consciousness highlight the adverse cardiac effects of the systemic impacts of the infection. Furthermore, the prognostic value of Pro-BNP in predicting cardiovascular complications, such as arrhythmias, in the early stages has been emphasized. The positive correlation between CK-MB levels and both COPD patients and pneumonia severity scores (CURB-65 and PSI) suggests that CK-MB could serve as an indicator of infection severity and inflammation. The positive correlation between LDH levels and troponin and Pro-BNP underscores the need to consider LDH in understanding the cardiac effects of systemic inflammation.

Based on these findings, a structured approach for monitoring cardiac biomarkers in CAP patients may improve early risk stratification and guide clinical management.

- Patients with severe pneumonia (CURB-65 ≥2, PSI class IV-V) and those with impaired consciousness or hemodynamic instability should undergo baseline troponin measurement at hospital admission.
- For patients with elevated troponin levels, follow-up measurements may be considered to assess trends and evaluate myocardial stress in collaboration with cardiology specialists.
- In patients at high risk for cardiovascular complications, Pro-BNP levels may serve as an adjunctive biomarker, particularly for predicting arrhythmias and hemodynamic instability.
- CK-MB and LDH levels, although less specific, could be considered complementary markers to further refine risk stratification.

Incorporating a biomarker-driven monitoring strategy into CAP management could facilitate early identification of high-risk patients and allow for timely cardiovascular interventions, potentially improving clinical outcomes. Future prospective studies should focus on validating this approach and assessing its impact on patient prognosis.

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Authorship contributions
Concept: B.D, O.Y, F.K., Design: O.Y, F.K.,Data Collection or Processing: B.D., O.Y., Analysis or Interpretation: B.D., O.Y., Literature Search: B.D., O.Y., F.K., Writing: B.D., O.Y., F.K.

Data availibity statement
The data that support the findings of this study are available from the corresponding author upon reasonable reques.

Declaration of competing interest
The authors deny any conflicts of interest related to this study.

Ethics
The study was approved by the local Ethics Committee (approval no: 2022/18).

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