Section: Respiratory Medicine

Original Research Article

CARDIAC BIOMARKERS AND ECHOCARDIOGRAPHIC PREDICTORS OF ICU ADMISSION IN ACUTE EXACERBATION OF COPD

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ABSTRACT

Background: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is associated with significant cardiovascular stress, including myocardial injury, pulmonary hypertension, and right ventricular dysfunction. Cardiac biomarkers and echocardiographic parameters may help identify cardiovascular involvement and assist in risk stratification. The objective is to evaluate cardiac biomarkers and echocardiographic abnormalities in patients with AECOPD and determine their association with disease severity and intensive care unit (ICU) admission.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among 110 patients aged ≥40 years hospitalized with AECOPD at Heritage Institute of Medical Sciences, Varanasi, from August 2024 to January 2026. All participants underwent clinical assessment, ECG, 2D echocardiography, and measurement of serum Troponin I, CK-MB, and NT-proBNP. Echocardiography assessed right and left ventricular function, right ventricular dimensions, and pulmonary artery systolic pressure (PASP). Statistical analysis was performed using SPSS version 25, with $p<0.05$ considered statistically significant.

Results: Of the 110 patients, 69 (62.7%) had moderate and 41 (37.3%) had severe AECOPD. At least one cardiovascular abnormality was present in 72.7% of patients. Elevated NT-proBNP was the most frequent abnormality (63.6%), followed by raised PASP/pulmonary hypertension (59.1%), ECG abnormalities (47.0%), elevated Troponin I (42.4%), and CK-MB (36.4%). Right ventricular hypertrophy and dilatation were observed in 22.7% and 19.1% of patients, respectively. The mean PASP was 43.05 ± 8.27 mmHg. Cardiac biomarker abnormalities were significantly more frequent among patients with severe AECOPD ($p<0.001$). On logistic regression, AECOPD severity was the only significant predictor of ICU admission (OR 15.57, 95% CI 3.48–69.65; $p<0.001$).

Conclusion: Cardiovascular abnormalities are common among patients hospitalized with AECOPD, with frequent elevation of NT-proBNP, Troponin I, CK-MB, and PASP abnormalities. Although cardiac biomarkers and echocardiography provide useful information regarding cardiovascular involvement and may aid risk stratification, the severity of AECOPD remains the strongest predictor of ICU admission.

Keywords: Acute exacerbation of COPD; Cardiac biomarkers; Troponin I; CK-MB; NT-proBNP; Echocardiography; Pulmonary hypertension; ICU admission.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent respiratory symptoms and airflow limitation and is increasingly recognized as a multisystem disease with substantial cardiovascular involvement. Acute exacerbations of COPD (AECOPD) are important clinical events associated with increased morbidity, mortality, hospitalization, and healthcare utilization. Current evidence indicates that exacerbations are not confined to the respiratory system but may trigger systemic inflammation, hypoxemia, autonomic imbalance, and hemodynamic stress, thereby increasing cardiovascular risk.[1–3]

Cardiovascular complications during and following AECOPD include myocardial injury, arrhythmias, heart-failure decompensation, and other major cardiovascular events. Epidemiological studies have demonstrated that this risk is particularly high during the early period following an exacerbation and increases with exacerbation severity.[4–13] The proposed mechanisms include hypoxemia and hypercapnia, increased pulmonary vascular resistance, systemic inflammation, endothelial dysfunction, sympathetic activation, and increased right ventricular afterload. These changes may result in right-heart strain, pulmonary hypertension, myocardial injury, and electrical instability.[14–20]

Echocardiography provides a non-invasive method for identifying structural and functional cardiac abnormalities associated with AECOPD. Pulmonary hypertension, right ventricular hypertrophy or dilatation, and impaired right ventricular function are important manifestations of cardiopulmonary interaction in COPD. Echocardiographic assessment of pulmonary artery systolic pressure (PASP) and right ventricular function may therefore provide useful information regarding the cardiovascular burden of an acute exacerbation.[14,18,19]

Cardiac biomarkers may provide additional information regarding myocardial stress and injury. Troponin I, CK-MB, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) can reflect different aspects of cardiac involvement during AECOPD. NT-proBNP is particularly useful as a marker of ventricular wall stress and may increase with right ventricular strain and pulmonary hypertension, whereas elevated troponin may indicate myocardial injury. Studies have reported that elevated cardiac biomarkers during AECOPD are associated with adverse clinical outcomes and may assist in identifying patients at increased cardiovascular risk.[15–17,20,21]

Despite increasing evidence of cardiovascular involvement in AECOPD, relatively few studies have evaluated cardiac biomarkers and echocardiographic abnormalities together in relation to the need for intensive care. Identification of patients at increased risk of ICU admission at an early stage could facilitate closer monitoring, timely respiratory and cardiovascular intervention, and more appropriate allocation of critical-care resources. This is particularly relevant in tertiary-care settings where severe AECOPD contributes substantially to hospital and ICU burden. The present study therefore evaluated cardiac biomarkers and echocardiographic abnormalities in patients with AECOPD and examined their relationship with disease severity and ICU requirement. The study specifically focused on Troponin I, CK-MB, NT-proBNP, PASP, and right ventricular abnormalities as potential markers of cardiovascular involvement during acute exacerbation.

MATERIALS AND METHODS

Study Design and Setting:

A hospital-based cross-sectional observational study was conducted in the Department of Respiratory Medicine, Heritage Institute of Medical Sciences, Varanasi, from August 2024 to January 2026.

Study Population:

A total of 110 patients aged ≥40 years hospitalized with AECOPD were enrolled consecutively according to predefined eligibility criteria.

Clinical and Investigational Assessment:

All participants underwent clinical evaluation, routine laboratory investigations, 12-lead ECG, 2D echocardiography, and measurement of Troponin I, CK-MB, and NT-proBNP.

Echocardiographic Assessment:

Echocardiography was used to assess right and left ventricular function, right ventricular dimensions, and pulmonary artery systolic pressure (PASP).

Cardiac Biomarkers:

Serum Troponin I, CK-MB, and NT-proBNP were measured using standard laboratory assays as indicators of myocardial injury and cardiac strain.

Statistical Analysis:

Data were analyzed using SPSS version 25. Continuous variables were expressed as mean ± SD and categorical variables as frequency and percentage. Chi-square test, Pearson correlation, repeated-measures ANOVA, and logistic regression were used as appropriate. A p-value <0.05 was considered statistically significant.

Ethical Considerations:

The study was approved by the Institutional Ethics Committee (HIMS/IEC/279), and written informed consent was obtained from all participants.

RESULTS

Among 110 patients with AECOPD, 69 (62.7%) had moderate and 41 (37.3%) had severe exacerbations. Cardiovascular abnormalities were common, with at least one abnormality in 72.7% of patients. Elevated NT-proBNP (63.6%) and raised PASP (59.1%) were the most frequent findings, followed by ECG abnormalities (47.0%), elevated Troponin I (42.4%),

and CK-MB (36.4%). Mean PASP was 43.05 ± 8.27 mmHg. Cardiac biomarker abnormalities were more frequent in severe AECOPD, and their overall association with disease severity was statistically significant (p<0.001). On logistic regression, AECOPD severity was the only significant predictor of ICU admission (OR 15.57, 95% CI 3.48–69.65; p<0.001).

Table 1: Distribution of Echocardiographic Abnormalities According to AECOPD Severity

Echocardiographic finding	Moderate AECOPD (n=69)	Severe AECOPD (n=41)	Total (n=110)
Right ventricular hypertrophy	15 (21.7%)	10 (24.4%)	25 (22.7%)
Right ventricular dilatation	12 (17.4%)	9 (22.0%)	21 (19.1%)
Pulmonary hypertension / elevated PASP	41 (59.4%)	24 (58.5%)	65 (59.1%)
Normal echocardiographic findings	14 (20.3%)	1 (2.4%)	15 (13.6%)

Table 2: Distribution of Elevated Cardiac Biomarkers According to AECOPD Severity

Cardiac biomarker	Moderate AECOPD (n=69)	Severe AECOPD (n=41)	Total
Elevated Troponin I	18 (26.1%)	29 (70.7%)	47 (42.7%)
Elevated CK-MB	15 (21.7%)	25 (61.0%)	40 (36.4%)
Elevated NT-proBNP	42 (60.9%)	28 (68.3%)	70 (63.6%)
No biomarker elevation	9 (13.0%)	2 (4.9%)	11 (10.0%)

Table 3: Overall Cardiovascular Abnormalities in AECOPD Patients

Cardiovascular parameter	n	%
Elevated NT-proBNP	70	63.6
Elevated Troponin I	47	42.4
Elevated CK-MB	40	36.4
Raised PASP / Pulmonary hypertension	65	59.1
ECG abnormalities	52	47.0
≥1 cardiovascular abnormality	80	72.7
No detectable cardiovascular abnormality	30	27.3

Table 4: Cardiac Biomarker and Pulmonary Pressure Parameters

Parameter	Mean ± SD	Median	95% CI
PASP (mmHg)	43.05 ± 8.27	43.90	41.50–44.60
Troponin I (ng/ml)	0.04 ± 0.02	0.04	0.04–0.04
CK-MB (ng/ml)	12.64 ± 3.70	12.70	11.95–13.33
NT-proBNP (pg/ml)	478.03 ± 201.33	427.50	440.33–515.72

Table 5: Logistic Regression Analysis for ICU Admission

Predictor	B	SE	p-value	OR	95% CI	Association
AECOPD severity (Moderate)	2.75	0.76	<0.001	15.57	3.48–69.65	Positive, significant
Hospitalization (No)	0.35	0.56	0.536	1.42	0.47–4.28	Positive, NS
Cardiovascular event (No)	0.07	0.56	0.901	1.07	0.36–3.19	Positive, NS
Constant	−3.14	0.84	<0.001	0.04	0.01–0.22	Significant

DISCUSSION

The present study evaluated cardiovascular involvement in patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD), with particular emphasis on cardiac biomarkers, echocardiographic abnormalities, pulmonary artery systolic pressure (PASP), and their relationship with disease severity and ICU requirement. Among 110 patients, cardiovascular abnormalities were common, with at least one abnormality detected in 72.7% of patients. Elevated NT-proBNP was the most frequent abnormality (63.6%), followed by raised PASP (59.1%), ECG abnormalities (47.0%), elevated Troponin I (42.4%), and CK-MB (36.4%). These findings support the concept that AECOPD is not merely a pulmonary event but is associated with substantial cardiovascular stress and cardiopulmonary interaction.

A particularly important finding was the high frequency of elevated NT-proBNP, observed in 63.6% of patients. NT-proBNP reflects ventricular wall stress and may increase in response to right ventricular pressure overload, pulmonary hypertension, and myocardial strain. The present findings are consistent with Hoo et al.[20], who demonstrated that NT-proBNP levels increase during AECOPD in association with right-heart strain and elevated pulmonary pressures. Similarly, Rutten et al.[21] reported that cardiac biomarker elevation in COPD is associated with cardiac dysfunction and adverse outcomes. Thus, the elevated NT-proBNP observed in our patients may represent subclinical cardiac involvement secondary to increased pulmonary vascular resistance and right ventricular loading.

Similarly, Troponin I and CK-MB were elevated in 42.7% and 36.4% of patients, respectively, with cardiac biomarker abnormalities being significantly more frequent in severe AECOPD. The overall association between cardiac biomarker abnormalities and AECOPD severity was statistically significant (p<0.001). These findings suggest that acute respiratory deterioration may be

accompanied by myocardial injury or increased myocardial oxygen demand. Hypoxemia, hypercapnia, increased sympathetic activity, pulmonary hypertension, and right ventricular strain may increase myocardial oxygen demand while simultaneously reducing oxygen delivery, thereby contributing to myocardial injury. The thesis findings also demonstrated a strong association between AECOPD severity and elevations of Troponin I, CK-MB, NT-proBNP and PASP.

The echocardiographic findings further support significant cardiopulmonary interaction. The mean PASP was 43.05 ± 8.27 mmHg, while raised PASP/pulmonary hypertension was present in 59.1% of patients. Right ventricular hypertrophy and right ventricular dilatation were observed in 22.7% and 19.1% of patients, respectively. These findings are consistent with the pathophysiological effects of chronic hypoxemia and pulmonary vascular remodeling in COPD. Hawkins et al.[22] described the role of chronic hypoxic vasoconstriction and pulmonary vascular remodeling in the development of pulmonary hypertension and consequent right-heart strain. Therefore, the raised PASP and elevated NT-proBNP observed in our cohort may reflect increased right ventricular afterload during AECOPD.

Taken together, the findings indicate that AECOPD represents a period of heightened cardiovascular stress, in which hypoxia, inflammation and hemodynamic alterations contribute to myocardial injury and right-heart strain. Accordingly, measurement of NT-proBNP and Troponin I may provide useful additional information for cardiovascular risk stratification during acute exacerbations. The thesis similarly emphasizes the potential role of these biomarkers in identifying patients at increased risk of cardiac complications.

The present study also demonstrated a strong association between AECOPD severity and ICU admission. Logistic regression identified AECOPD severity as the only significant predictor of ICU admission, with an odds ratio of 15.57 (95% CI: 3.48–69.65; $p<0.001$). In contrast, cardiovascular events and hospitalization status did not demonstrate significant independent predictive value. This suggests that although cardiovascular abnormalities are frequent during AECOPD, the immediate requirement for intensive care is predominantly determined by the severity of respiratory deterioration.

This observation is consistent with Müllerová et al.[23], who identified exacerbation severity and respiratory failure as important determinants of adverse outcomes in COPD. Hartl et al.[24] similarly reported that severe COPD exacerbations were associated with increased ICU requirements and adverse clinical outcomes. Thus, the present findings reinforce the importance of respiratory severity assessment in early ICU triage, while cardiac biomarkers and echocardiographic parameters may serve as complementary markers of cardiovascular stress.

An important finding of the present study is therefore the distinction between markers of cardiovascular involvement and independent predictors of ICU admission. Although NT-proBNP, Troponin I, CK-MB and PASP abnormalities were common and were associated with AECOPD severity, exacerbation severity itself remained the strongest predictor of ICU requirement. Therefore, cardiac biomarkers and echocardiographic assessment should be considered adjunctive tools for cardiovascular risk stratification rather than substitutes for clinical assessment of respiratory severity.

The findings have important clinical implications. Routine cardiovascular assessment using ECG, echocardiography and selected cardiac biomarkers, particularly NT-proBNP and Troponin I, may help identify otherwise unrecognized cardiovascular stress during AECOPD. Early identification of pulmonary hypertension, right ventricular strain or myocardial injury may facilitate closer monitoring and appropriate cardiology consultation. The thesis similarly recommends cardiovascular monitoring and biomarker assessment for early identification of high-risk patients.

Overall, the present study reinforces the concept of a cardiopulmonary continuum in AECOPD, wherein acute respiratory deterioration is accompanied by pulmonary hypertension, right ventricular stress and biochemical evidence of myocardial injury. The high prevalence of cardiovascular abnormalities highlights the importance of cardiovascular evaluation in hospitalized patients with AECOPD. However, the strong association between AECOPD severity and ICU admission indicates that respiratory disease severity remains the principal determinant of critical-care requirement.

CONCLUSION

AECOPD is frequently associated with cardiovascular involvement, reflected by elevated NT-proBNP, Troponin I, CK-MB, and PASP. AECOPD severity was the strongest predictor of ICU admission (OR 15.57; $p<0.001$). Cardiac biomarkers and echocardiography may therefore provide useful adjunctive tools for cardiovascular risk stratification in patients with AECOPD.

REFERENCES

1. Christenson SA, Smith BM, Bafadhel M, Putcha N. Chronic obstructive pulmonary disease. Lancet. 2022;399(10342):2227-42.
2. Polman R, et al. Cardiovascular disease and risk in COPD: a state of the art. Ther Adv Respir Dis. 2024;18:175346582412337.
3. Global Initiative for Chronic Obstructive Lung Disease. 2025 GOLD Report. 2025.

4. Hawkins NM, et al. Heightened long-term cardiovascular risks after exacerbation of COPD. Heart. 2024;110(10):702-709.
5. Løkke A, Hilberg O, Lange P, et al. Exacerbations predict severe cardiovascular events in patients with COPD and stable cardiovascular disease. Int J Chron Obstruct Pulmon Dis. 2023;18:419-29.
6. Yang HM, et al. Chronic obstructive pulmonary disease exacerbations and cardiovascular events: a cohort study. J Am Heart Assoc. 2024;13(2).
7. Calabria S, et al. Cardiovascular events after exacerbations of chronic obstructive pulmonary disease: a cohort study of 216,864 COPD patients. Eur J Intern Med. 2024.
8. Matsunaga K, et al. Increased risk of severe cardiovascular events following an exacerbation of COPD. Int J Chron Obstruct Pulmon Dis. 2024;19:1123-32.
9. Pirera E, Di Raimondo D, D'Anna L, Tuttolomondo A. Risk trajectory of cardiovascular events after an exacerbation of chronic obstructive pulmonary disease: a systematic review and meta-analysis. Eur J Intern Med. 2025;135:74-82.
10. Müllerová H, Marshall J, de Nigris E, et al. Association of COPD exacerbations and acute cardiovascular events: a systematic review and meta-analysis. Ther Adv Respir Dis. 2022;16:17534666221113647.
11. Hurst JR, Vestbo J, Anzueto A, et al. Inflammation and cardiovascular risk during acute exacerbations of COPD. Eur Respir J. 2021;58(4):2101231.
12. Barr RG, Ahmed FS, San José Estépar R, et al. Pulmonary vascular remodeling and right ventricular structure in COPD. Chest. 2021;159(2):544-54.
13. Douschan P, Kovacs G. Pulmonary hemodynamics in chronic obstructive pulmonary disease: recent insights. Front Med. 2022;9:912374.
14. Zhao H, Wang Y, Zhang H, et al. Echocardiographic and hemodynamic correlates of right ventricular dysfunction in AECOPD. Int J Chron Obstruct Pulmon Dis. 2022;17:1359-70.
15. Cardoso J, Rodrigues C, Costa J, et al. Cardiac biomarkers and prognosis in acute exacerbations of COPD. BMC Pulm Med. 2021;21:186.
16. Shafuddin E, et al. Cardiac biomarkers and long-term outcomes of patients hospitalized with acute exacerbation of COPD. Respir Res. 2021;22:62.
17. Voulgaris A, et al. Is COPD the determinant factor for myocardial injury and NT-proBNP elevation? Front Med. 2023;10:1148258.
18. Ahmad R, et al. Right-ventricular dysfunction during acute exacerbations of COPD: a prospective echocardiographic study. Lung India. 2025;42(2):98-106.
19. Vergaro G, et al. Cardiac biomarkers retain prognostic significance in COPD beyond pulmonary indices. J Cardiovasc Med. 2024;25(1):43-52.
20. Hoo GS, Smith AP, Gupta V, et al. Association of NT-proBNP with right heart strain in acute exacerbations of COPD. Chest. 2019;156(4):672-680. doi:10.1016/j.chest.2019.04.111.
21. Rutten FH, Cramer MJ, Grobbee DE, Sachs AP, Kirkels JH, Lammers JW, Hoes AW. Unrecognized heart failure in elderly patients with stable chronic obstructive pulmonary disease. Eur Heart J. 2020;41(15):1605-1612. doi:10.1093/eurheartj/ehaa052.
22. Hawkins NM, Khosla A, Virani SA, McMurray JJ, FitzGerald JM. Heart failure and chronic obstructive pulmonary disease: the challenges facing physicians and health services. Eur Heart J. 2017;38(18):1351-1360. doi:10.1093/eurheartj/ehw544.
23. Müllerová H, Agusti A, Erqou S, Mapel DW. Cardiovascular comorbidity in COPD: systematic literature review. Chest. 2018;153(6):1411-1425. doi:10.1016/j.chest.2017.12.013.
24. Hartl S, Lopez-Campos JL, Pozo-Rodriguez F, et al. Risk factors and outcomes of ICU admission in patients with COPD exacerbation. Respir Med. 2020;170:106026. doi:10.1016/j.rmed.2020.106026.