

Original Research Article

Association of pro-brain natriuretic peptide with degree of severity and prognostic outcome in patients with acute exacerbation of chronic obstructive pulmonary disease

Salah Uddin Anach^{1*}, Shafiul Azam Quadry¹, Mohammad Emran Hossain²,
M. Abu Rayhan³, Debashish Dhar¹

¹National Institute of Disease of Chest and Hospital (NIDCH), Dhaka

²Department of Microbiology, Noakhali Medical College, Noakhali, Bangladesh

³Chest Disease Clinic, Noakhali, Bangladesh

Received: 25 July 2026

Revised: 12 October 2026

Accepted: 17 October 2026

*Correspondence:

Salah Uddin Anach,

E-mail: anasfestival@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a major cause of morbidity and mortality. N-terminal pro-brain natriuretic peptide (NT-proBNP) is an established biomarker of heart failure, but its association with AECOPD severity in patients without overt cardiac disease remains unclear. The objective is to assess the association between NT-proBNP levels, disease severity, and prognostic outcomes in patients with AECOPD.

Methods: This cross-sectional study was conducted at the Department of Respiratory Medicine, NIDCH, Dhaka, Bangladesh, from July 2018 to June 2019. Seventy patients with established COPD admitted with acute exacerbation were enrolled through purposive sampling. Demographic and clinical data, SpO₂, arterial blood gas parameters, pulmonary artery systolic pressure (PASP), mMRC dyspnea grade, and NT-proBNP levels were recorded. Data were analyzed using chi-square tests, multivariate logistic regression, and Pearson correlation. Statistical significance was set at $p < 0.05$.

Results: The mean age was 61.77 ± 8.91 years, and 95.7% of participants were male. Elevated NT-proBNP levels were observed in 74.3% of patients. Multivariate logistic regression identified mMRC dyspnea grade as the only independent factor significantly associated with elevated NT-proBNP (OR=7.457, 95% CI: 1.772–31.370; $p=0.006$). Significant correlations were reported between NT-proBNP and SpO₂, pH, PCO₂, and PO₂. Smoking exposure of 21–30 pack-years was also significantly associated with elevated NT-proBNP ($p=0.042$).

Conclusions: Elevated NT-proBNP levels were associated with greater dyspnea severity and several respiratory parameters in patients with AECOPD, suggesting potential value in assessing disease severity. Further prospective studies are needed to establish its prognostic utility.

Keywords: Pro-BNP, NT-proBNP, AECOPD, MMRC dyspnea grade, Hypoxemia, Hypercapnia, Pulmonary hypertension, Biomarker, Prognosis

INTRODUCTION

The global initiative for chronic obstructive lung disease (GOLD) defines chronic obstructive pulmonary disease (COPD) as “a common, preventable and treatable disease characterized by persistent respiratory symptoms and

airflow limitation” due to airway or alveolar abnormalities caused by significant exposure to noxious particles.¹ The chronic airflow limitation in COPD results from a mixture of small airways disease and parenchymal destruction (emphysema).¹ Worldwide, an estimated 65 million people have moderate-to-severe COPD.² In 2005, 5% (3.2

million) of global deaths were attributable to COPD, and from 1990 to 2015, COPD mortality increased by nearly 11%, while prevalence rose by 44%.³

In Bangladesh, COPD prevalence among individuals >40 years is 13.5%.⁴ COPD is diagnosed by a post-bronchodilator FEV₁/FVC ratio <0.70 in symptomatic patients. GOLD 2019 classifies airflow limitation severity based on post-bronchodilator FEV₁.¹ Both COPD and chronic heart failure (CHF) are global epidemics affecting over 10 million patients each, incurring significant morbidity and mortality.⁵ They frequently coexist, sharing risk factors such as cigarette smoking, advanced age, and systemic inflammation.⁶ Pulmonary arterial hypertension (PAH) develops late in COPD, is associated with severe hypoxemia and core pulmonale, and carries a poor prognosis.¹ While echocardiography is useful, hyperinflation may impair sound-wave transmission; right heart catheterization remains the gold standard for pulmonary artery pressure measurement.¹ Clinically, distinguishing cardiac from pulmonary causes of dyspnea is often difficult.

An acute exacerbation of COPD (AECOPD) is defined as an acute event characterized by worsening of respiratory symptoms, including dyspnea, cough, and sputum changes.¹ Although viral or bacterial infection is common, nearly one-third of AECOPD cases have no identified cause.⁷ Furthermore, AECOPD and left heart failure (LHF) often coexist, partly due to shared risk factors (aging, male sex and smoking) and complex interactions between chronic systemic inflammation and oxidative stress.^{8,9} However, diagnosing LHF in AECOPD is extremely challenging because symptoms overlap considerably.⁹ B-type natriuretic peptide (BNP) is derived from the pro-hormone pro-BNP (108 amino acids), which is cleaved into active BNP and the inactive 76-amino acid N-terminal fragment (NT-proBNP).¹⁰ Plasma NT-proBNP is widely used clinically for diagnosis, risk stratification, and management of heart failure.¹¹ Elevated NT-proBNP levels also occur in chronic pulmonary diseases, infections, critical illness, renal failure, and other conditions.¹⁰

Importantly, elevated natriuretic peptide levels have been observed in COPD patients without HF, likely originating from right heart strain due to cor pulmonale, secondary pulmonary hypertension, and hypoxemia, which increase BNP gene expression.¹⁰ However, the clinical utility of plasma NT-proBNP for detecting coexisting LHF specifically in the context of AECOPD is less well established. Previous reports indicate that NT-proBNP concentrations can rise in COPD, particularly during acute exacerbations, although not to the levels seen in overt heart failure.¹²

The general objective is to evaluate the plasma levels of Pro-BNP (N-terminal pro-brain natriuretic peptide) in patients presenting with acute exacerbation of chronic obstructive pulmonary disease (AECOPD). The specific

objectives is to assess the association between Pro-BNP levels and oxygen saturation (SpO₂) in patients with AECOPD; assess the association between Pro-BNP levels and arterial blood gas (ABG) parameters (including pH, PaO₂, PaCO₂ and HCO₃⁻) in patients with AECOPD; study the association between Pro-BNP levels and the duration of hospital stay in patients with AECOPD; and evaluate the association between Pro-BNP levels and in-hospital mortality among patients with AECOPD.

METHODS

This study was done as per the following methodology to find out the association of Pro-BNP with degree of severity and prognostic outcome in patients with AECOPD. This chapter describes the research design, description of research approach, setting and research instruments and different steps used for collecting and organizing the data.

Study design

The study employed a cross-sectional study design, which allowed the assessment of Pro-BNP levels and their association with various clinical and prognostic parameters at a single point in time during the acute exacerbation episode.

Study period

The study was conducted over a period of two years, starting from 1st July 2018 to 30th June 2019.

Study location

The study was carried out at the Department of Respiratory Medicine, National Institute of the Diseases of the Chest and Hospital (NIDCH), located in Mohakhali, Dhaka, Bangladesh. This is a tertiary care referral hospital specializing in respiratory and chest diseases, making it an ideal setting for research on COPD exacerbations.

Study population

The study population comprised adult patients who had a known pre-existing diagnosis of chronic obstructive pulmonary disease (COPD) and were subsequently admitted to the hospital with an acute exacerbation of their underlying disease.

Sampling method

Participants for this study were selected using a purposive sampling method. This non-probability sampling technique was employed to specifically enroll those patients who met all the predefined inclusion criteria and did not have any of the exclusion criteria, ensuring that the study sample was appropriate for addressing the research objectives.

Inclusion criteria

All patients admitted to the inpatient department or attending the outpatient department (OPD) of NIDCH who were diagnosed as a case of acute exacerbation of COPD based on the GOLD guidelines were included in the study. The GOLD guideline-based diagnosis required the presence of a history of smoking, consistent clinical examination findings, supportive chest X-ray findings, and a post-bronchodilator forced expiratory volume in one second (FEV₁) to forced vital capacity (FVC) ratio of less than 0.70. Additionally, only patients aged more than 40 years were included, and both male and female patients were eligible for enrollment.

Exclusion criteria

Patients were excluded from the study if they had any pulmonary disease other than COPD, such as asthma, bronchiectasis, or interstitial lung disease. Other exclusion criteria included the presence of systolic heart failure defined as an ejection fraction of less than 50%, valvular heart disease, acute myocardial infarction, grade 3 or grade 4 diastolic dysfunction, any form of malignancy, advanced renal disease, or pregnancy. These stringent exclusion criteria were applied to minimize confounding factors that could independently elevate Pro-BNP levels.

Sample size estimation

The sample size for this study was determined using the standard formula for cross-sectional studies: $n = z^2 pq / d^2$. In this formula, n represents the desired sample size, z is the value of the standard normal distribution at a given level of significance (at 95% confidence level, $Z = 1.96$), p is the expected proportion of the event or prevalence of the condition of interest, which was regarded as 6.25% or 0.0625 based on a previous study by Rajkumar and colleagues in 2017, q is calculated as $1 - p$ which equals 0.9375, and d is the margin of error set at 0.05 corresponding to a 95% confidence level. Substituting these values into the formula, the calculated sample size was approximately 90 patients, which became the targeted sample size for this study.

Data collection procedure

Patients with COPD attending the outdoor department or admitted as inpatients in the Department of Respiratory Medicine at NIDCH, Dhaka, Bangladesh, were initially screened according to the predefined inclusion and exclusion criteria. Once eligibility was confirmed, each potential participant was approached and provided with a

detailed explanation of the study procedures, including the purpose of the study, the nature of their participation, the blood sample collection for Pro-BNP measurement, and the monitoring of SpO₂ and arterial blood gas analysis. After ensuring that the participant understood all aspects of the study, written informed consent was obtained from all patients prior to their enrollment. Following consent, data were collected through clinical examination, laboratory investigations, and review of medical records.

Data processing and statistical analysis

After collection data were checked thoroughly for consistency. Then collected data were uniformly coded according to standard specifications, verified, edited, reviewed to satisfy quality control standards and analyzed according to the objectives and variables. Data analysis was conducted by descriptive and analytical techniques including mean, SD, frequency distribution and computation of percentage. Means of quantitative variables of various factors were studied by using student's t -test and. Logistic regression analysis was performed to assess the independent relationship between the factors. A $p < 0.05$ was considered to be significant. Statistical Package for Social Science (SPSS) version 21 for windows was used to analyze the data

Ethical considerations

Prior to study commencement, formal ethical approval was obtained from the Institutional Review Board of NIDCH, Dhaka, ensuring adherence to national and international ethical guidelines. Written informed consent was secured from each participant after explaining the study's purpose, procedures, and potential risks. Participants were assured that participation was voluntary and they could withdraw at any time without affecting their medical care. All patient data were kept strictly confidential, anonymized using unique codes, and stored securely. The study involved only routine clinical investigations (Pro-BNP, SpO₂, and ABG) that are part of standard care for AECOPD patients. The entire study was conducted in accordance with the Declaration of Helsinki.

RESULTS

This cross-sectional study was carried out among 70 patients who are the known case of COPD who admitted in hospital with exacerbation into Department of Respiratory Medicine, National Institute of the Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka, Bangladesh to see the levels of Pro-BNP during acute exacerbation of COPD.

Table 1: Sociodemographic characteristics of the study participants (n=70).

Characteristics	Frequency (N)	Percentage (%)
Age group (years)		
44–56	19	27.1
57–68	33	47.1

Continued.

Characteristics	Frequency (N)	Percentage (%)
69–80	18	25.7
Mean age±SD (years)	61.77±8.906	-
Age range (years)	44–80	-
Sex		
Male	67	95.7
Female	3	4.3
Occupation		
Farmer	20	28.6
Businessman	17	24.3
Driver	9	12.9
Day labor	5	7.1
Rickshaw puller	5	7.1
Service holder	7	10.0
Others	7	10.0

Table 2: Distribution of the patients by SPO2 and arterial pH (n=70).

	Frequency	Percentage (%)
SPO₂ (%)		
Up to 60%	3	4.3
61-70	4	5.7
71-80	5	7.1
81-90	43	61.4
More than 90%	15	21.4
pH		
Acidosis	17	24.3
Normal	48	68.6
Alkalosis	5	7.1

Table 1 presents the sociodemographic characteristics of the 70 study participants. The largest proportion of patients (47.1%) belonged to the 57–68-year age group, followed by those aged 44–56 years (27.1%) and 69–80 years (25.7%). The mean age was 61.77±8.906 years, ranging from 44 to 80 years. Regarding sex distribution, the majority of participants were male (95.7%), while females accounted for 4.3%. In terms of occupation, farmers constituted the largest group (28.6%), followed by businessmen (24.3%) and drivers (12.9%). Service holders and individuals classified under other occupations each represented 10.0% of the participants, while day labor and rickshaw pullers each accounted for 7.1%.

Table 2 shows among total 70 patients, majority (43, 61.4%) had SPO₂ within (81-90) %, 15 (21.4%) had SPO₂ within (more than 90) %, 5 (7.1%) had SPO₂ within (71-80) %, 4 (5.7%) had SPO₂ within (61-70) %, and only 3 (4.3%) had SPO₂ up to 60%. SPO₂ ranged from 53% to 98% and mean SPO₂ was 85.86±9.878 SD. Demonstrates among total 70 patients, the greater portion of the respondents (48, 68.6%) had normal pH (7.35-7.45), 17 (24.3%) had acidosis (pH<7.35) and only 5 (7.1%) had alkalosis (>7.45). pH ranged from 7.21 to 7.49 and the mean pH was 7.3784±0.06114.

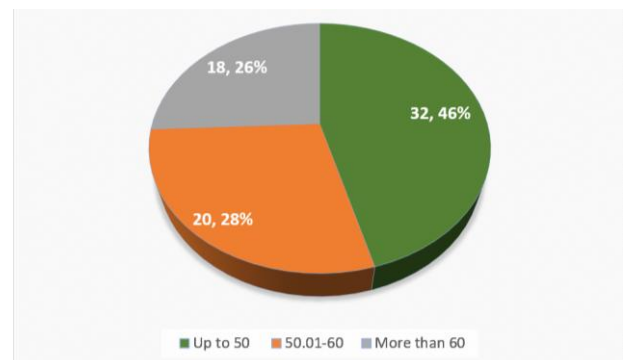


Figure 1: Distribution of the patients according to arterial PCO₂ (n=70).

Figure 2 presents among total 70 patients, majority (32, 45.7%) had normal PCO₂ (up to 50 mmHg) (20, 28%) had hypercapnia (50-60 mmHg), (18, 26%) had Hypercapnia (>60 mmHg)). PCO₂ ranged from 29.3 mmHg to 127 mmHg and mean PCO₂ was 54.6414±17.43853 SD mmHg.

Figure 3 presents among total 70 patients, majority (38, 54.3%) had no hypoxia (PO₂ 60 or more mmHg) and the rest 32 (45.7%) had hypoxia (PO₂ <60 mmHg). PO₂ ranged

from 26 mmHg to 134 mmHg and mean PO_2 was 65.5886 ± 25.36856 SD mmHg.

Figure 4 shows among total 70 patients, majority (50, 71.4%) had alkalosis ($HCO_3^- > 28$), 10 (14.3%) had normal HCO_3^- (24-28) and the rest 10 (14.3%) had acidosis ($HCO_3^- < 24$). HCO_3^- ranged from 20.6 to 55 and mean HCO_3^- was 31.2757 ± 6.37241 SD.

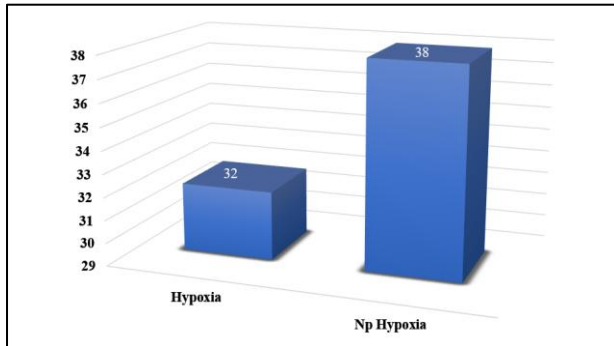


Figure 2: Distribution of the patients according to arterial PO_2 (n=70).

Table 3 illustrates the greater portion of the respondents (32, 45.7%) had PASP 24-40, 25 (35.7%) had PASP 41-60, 9 (12.9%) had PASP 61-80, 4 (5.7%) had PASP 81-110. PASP level ranged from 24 to 110 pg/ml and mean PASP level was 45.8286 ± 18.78943 SD.

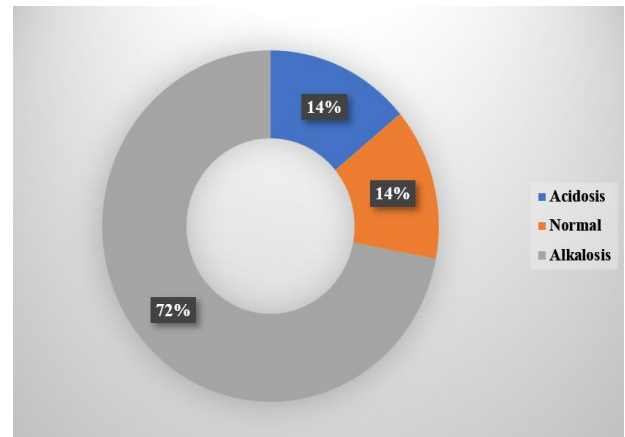


Figure 3: Distribution of the patients according to serum HCO_3^- (n=70).

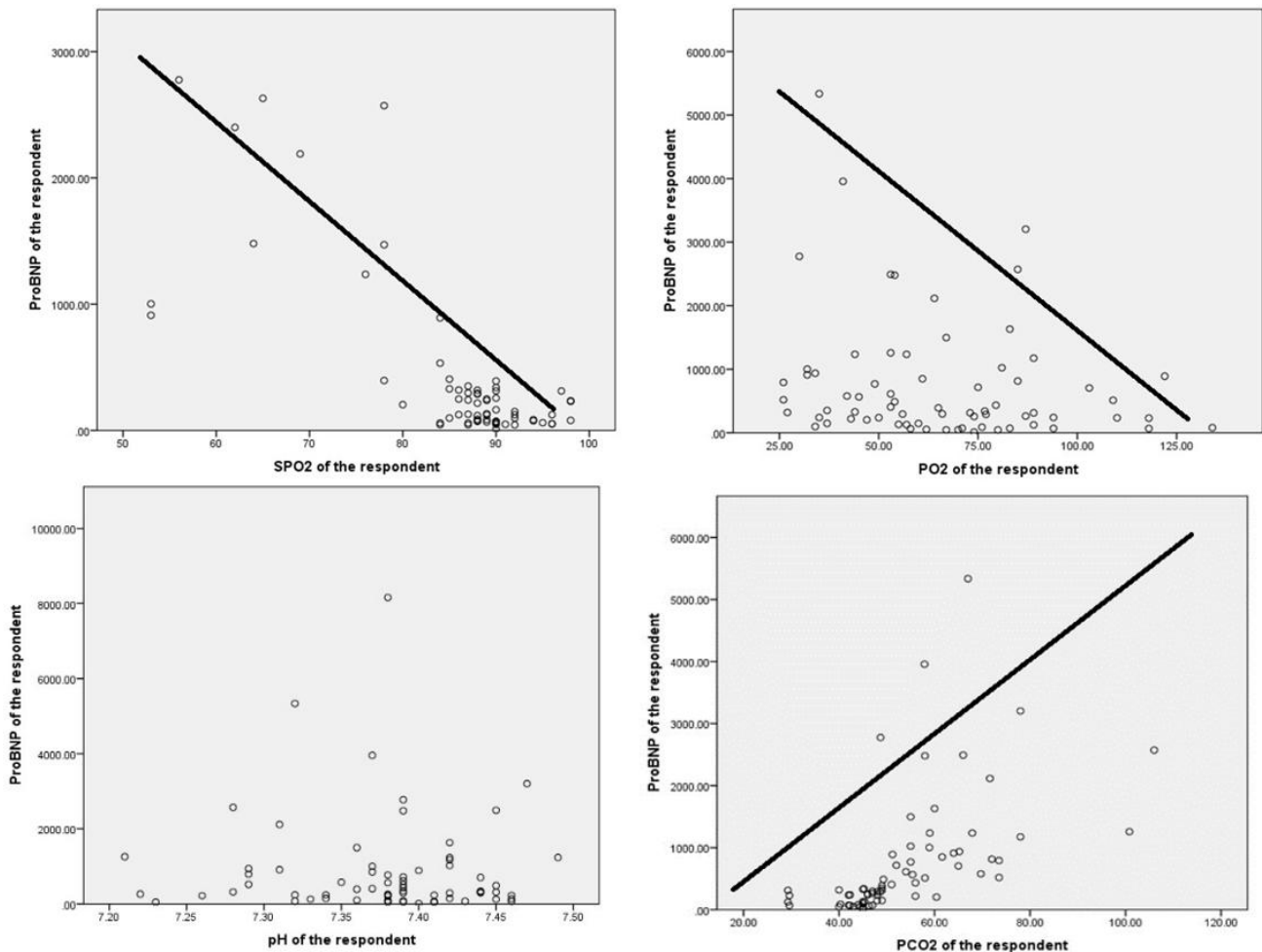


Figure 4: Hypoxemia in AECOPD results from ventilation-perfusion mismatch.

Table 3: Distribution of the patients according to PASP (n=70).

PASP	Frequency	Percent (%)
24-40	32	45.7
41-60	25	35.7
61-80	9	12.9
81-110	4	5.7

Table 4: Association between selected variables and pro BNP (n=70).

Variables	Pro BNP		Significance
Sex	Normal f (%)	Elevated f (%)	
Male	16 (23.9%)	51 (76.1%)	$\chi^2 = 2.752$, df=1, p=0.097
Female	2 (66.7%)	1 (33.3%)	
Total	18 (25.7%)	52 (74.3%)	
Smoking status			
Non- smoker	1 (14.3%)	6 (85.7%)	$\chi^2 = 0.532$, df=1, p=0.466
Smoker	17 (27%)	46 (73%)	
Total	18 (25.7%)	52 (74.3%)	
Duration of smoking in pack years	Negative f (%)	Positive f (%)	
11-20	17 (35.4%)	31 (64.6%)	$\chi^2 = 4.117$, df=1, p=0.042*
21-30	0 (0.0%)	15 (100%)	
None	1 (14.3%)	6 (85.7%)	
Total	18 (25.7%)	52 (74.3%)	

*Statistically significant.

Table 4 shows the majority of male patients (51, 76.1%) had Elevated pro BNP. The chi square test revealed that the difference was statistically not significant where χ^2 (1, 70) = 2.752, df=1, p=0.097, which is >0.05. The majority of smoker patients (46, 73%) had elevated pro BNP. The chi square test revealed that the difference was statistically not significant where χ^2 (1, 70) = 0.532, df=1, p=0.466,

which is >0.05. The majority of the respondents with smoking for 21-30 pack years (15, 100%) had elevated pro

BNP. To see the difference whether it is statistically significant or not, a chi square test is done. The chi square test revealed that the difference was statistically significant where χ^2 (1, 70) = 4.117, df=1, p=0.042, which is <0.05.

Table 5: Multivariate analysis to see the relationship between pro BNP and selected variables.

Variables	B	P value	OR	95% C.I. for OR
Number of previous exacerbations	30.591	0.994	1.931	0.000
Smoking pack year	-.396	0.176	.673	0.379-1.194
MMRC grade	2.009	0.006	7.457	1.772-31.370
Location of the patient	1.373	0.748	3.947	0.001-16919.872
Ventilation	1.555	.481	4.737	0.063-358.870
pH	-1.673	0.850	0.188	0.000
PCO ₂	-1.470	0.262	.230	.018-3.003
PO ₂	0.148	0.912	1.160	0.083-16.174
SPO ₂	-0.552	0.912	.576	0.079-4.195
Constant	2.423	0.586	11.278	0.079

*Statistically significant

Table 5 presents the multivariate logistic regression analysis examining the relationship between Pro-BNP and selected variables. The overall model was significant (p=0.000) and could predict Pro-BNP levels from 23.5%

to 34.6%. Among all variables, only MMRC grade showed a statistically significant association with Pro-BNP (p=0.006, OR=7.457, 95% CI: 1.772 – 31.370), indicating that patients with higher dyspnea grades had significantly elevated Pro-BNP levels.

Table 6: Correlation analysis to see the relationship between pro BNP and selected variables.

Variables tested	Correlation coefficient	P value
Pro BNP and SPO₂	r=-0.004; 95% CI: -0.316 - 0.1	0.001*
Pro BNP and PH	r=0.103; 95% CI: -0.214 - 0.245	0.008*
Pro BNP and PCO₂	r=0.112; 95% CI: -0.260 - 0.201	0.006*
Pro BNP and PO₂	r=-0.09; 95% CI: -0.267 - 0.085	0.007*

*Statistically significant

Table 6 presents the Pearson correlation analysis, significant association was found between the log-transformed NT-pro-BNP levels and SPO₂ (r=-0.004; 95% CI: -0.316-0.1; P=0.001), PH (r=0.103; 95% CI: -0.214 - 0.245; P=0.008), PCO₂ (r=0.112; 95% CI: -0.260 - 0.201; P=0.006), PO₂ levels (r=-0.09; 95% CI: -0.267 - 0.085; P=0.007).

DISCUSSION

This cross-sectional study was conducted among 70 known COPD patients admitted with acute exacerbation to the Department of Respiratory Medicine, NIDCH, Dhaka, Bangladesh, to evaluate Pro-BNP levels and their association with severity and prognostic outcomes. Regarding age distribution, nearly half of the patients (47.1%) were in the 57–68 years age group, followed by 27.1% in the 44–56 years group and 25.7% in the 69–80 years group. The mean age was 61.77±8.906 years, ranging from 44 to 80 years (Table 1).

This is consistent with literature showing COPD exacerbations are more common in older adults.¹³ Among 70 patients, the vast majority (95.7%) were male and only 4.3% were female (Figure 1), reflecting the higher prevalence of smoking and occupational exposures among males in Bangladesh.¹⁴ Regarding occupation, most participants were farmers (28.6%), followed by businessmen (24.3%) and drivers (12.9%) (Table 2).

The high proportion of farmers is relevant, as occupational exposure to biomass fuel and agricultural chemicals is a known COPD risk factor in low-income countries.¹⁵ Assessment of SpO₂ revealed that the majority (61.4%) had SpO₂ between 81-90%, while 21.4% had SpO₂ above 90%. The mean SpO₂ was 85.86±9.878% (Table 3). Hypoxemia is a common feature of AECOPD associated with worse outcomes.¹⁶ Regarding arterial blood gas, most patients (68.6%) had normal pH, while 24.3% had acidosis and 7.1% had alkalosis.

The mean pH was 7.3784±0.06114 (Table 4). Acidosis during AECOPD indicates severe exacerbation and hypercapnic respiratory failure.¹⁷ Analysis of PCO₂ showed 45.7% had normal levels, 28.6% had hypercapnia (50-60 mmHg), and 25.7% had severe hypercapnia (>60 mmHg). The mean PCO₂ was 54.64±17.44 mmHg (Figure 2). Elevated PCO₂ is associated with increased mortality in AECOPD.¹⁸ Regarding PO₂, 54.3% had no hypoxia (PO₂ ≥60 mmHg), while 45.7% had hypoxia. The mean PO₂ was

65.59±25.37 mmHg (Figure 3). Hypoxemia in AECOPD results from ventilation-perfusion mismatch.¹⁹ Serum HCO₃⁻ showed that 71.4% had alkalosis (>28 mmol/l), with mean HCO₃⁻ of 31.28±6.37 mmol/l (Figure 4). Elevated bicarbonate is a compensatory response to chronic hypercapnia.²⁰ Assessment of PASP revealed that 45.7% had PASP between 24-40 mmHg, 35.7% between 41-60 mmHg, 12.9% between 61-80 mmHg, and 5.7% between 81-110 mmHg. The mean PASP was 45.83±18.79 mmHg (Table 5). Elevated PASP indicates pulmonary hypertension, a common complication of advanced COPD associated with poor prognosis, developing due to chronic hypoxemia-induced vasoconstriction.^{21,1} Table 6 shows that 76.1% of males had elevated Pro-BNP compared to 33.3% of females (p=0.097, not significant).

Among smokers, 73% had elevated Pro-BNP (p=0.466, not significant). However, when analyzed by pack-years, all patients (100%) with 21-30 pack-years had elevated Pro-BNP, a statistically significant association ($\chi^2=4.117$, p=0.042). Cumulative smoking exposure, rather than smoking status alone, is associated with elevated Pro-BNP, possibly due to smoking-induced myocardial stress.²² Table 7 presents multivariate logistic regression. The overall model was significant (p=0.000) and could predict Pro-BNP levels from 23.5% to 34.6%.

Among all variables, only MMRC dyspnea grade showed a statistically significant independent association with Pro-BNP (p=0.006, OR=7.457, 95% CI: 1.772-31.370), indicating that patients with higher MMRC grades had significantly elevated Pro-BNP levels. This is clinically relevant, as dyspnea severity reflects both respiratory and cardiac compromise in AECOPD.²³ Table 8 presents Pearson correlation analysis. Significant associations were found between Pro-BNP and SpO₂ (r=-0.004, p=0.001), pH (r=0.103, p=0.008), PCO₂ (r=0.112, p=0.006), and PO₂ (r=-0.09, p=0.007).

The negative correlation with SpO₂ and PO₂ suggests lower oxygen levels are associated with higher Pro-BNP. The positive correlation with PCO₂ indicates hypercapnia is associated with elevated Pro-BNP. These findings are consistent with previous reports that hypoxemia and hypercapnia stimulate natriuretic peptide release from the right heart due to increased right ventricular afterload from hypoxic pulmonary vasoconstriction.^{10,24} Elevated NT-proBNP in AECOPD patients without overt heart failure correlates with severity of hypoxemia and pulmonary hypertension.¹²

Limitations

Despite all efforts, this study had several limitations. First, this was a single-center observational study with a small sample size, and with any study of this design there is the potential for confounding variables. Second, the study was conducted on purposively selected non-random samples; therefore, the results might not necessarily have external validity. Third, follow-up of patients was not possible in this study. Fourth, as only a cross-sectional study was done, causal association could not be established; to prove associations, a prospective follow-up cohort study or case-control study is recommended. Fifth and finally, as this was a single-center study, the results may not reflect the true burden prevalent in the community as a whole.

CONCLUSION

In light of the findings of this study, measurement of serum NT-proBNP may serve as an available, valuable, low-cost, and noninvasive predictor for assessing the severity of acute exacerbation of COPD (AECOPD). Pro-BNP levels were elevated in the majority (74.3%) of hospitalized patients with AECOPD. Multivariate analysis revealed that higher MMRC dyspnea grade was significantly and independently associated with elevated Pro-BNP ($p=0.006$, $OR=7.457$). Correlation analysis also showed significant associations between Pro-BNP and SpO_2 , pH, PCO_2 , and PO_2 . Therefore, Pro-BNP may be considered a significant predictor for ICU admission, need for invasive mechanical ventilation, and prolonged hospital stay in patients with AECOPD.

Recommendations

During acute exacerbation of COPD, spirometry is often not feasible and ABG analysis requires special equipment and expert personnel; therefore, simple measurement of Pro-BNP may be used as a practical indicator of severity and short-term prognosis. We suggest that such studies should be repeated with a larger sample size for all practical purposes. More multicenter studies are needed to clarify the findings. To prove causal associations, a prospective follow-up cohort study is recommended. Our study is small, and a more extended study is needed to settle this issue. We hope the results presented here will serve as a pilot study prompting further research with larger patient samples to validate the results.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: 2019 report. Global Initiative for

- Chronic Obstructive Lung Disease; 2019. Available from: <https://goldcopd.org/wp-content/uploads/2018/11/GOLD-2019-v1.7-FINAL-14Nov2018-WMS.pdf> Accessed on 10 April 2026.
2. World Health Organization. Chronic obstructive pulmonary disease (COPD). Geneva: World Health Organization; 2026. Available from: [https://www.who.int/news-room/fact-sheets/detail/chronic-obstructive-pulmonary-disease-\(copd\)](https://www.who.int/news-room/fact-sheets/detail/chronic-obstructive-pulmonary-disease-(copd)). Accessed on 10 April 2026.
3. Soriano JB, Abajobir AA, Abate KH, Abera SF, Agrawal A, Ahmed MB, et al. Global, regional, and national deaths, prevalence, disability-adjusted life years, and years lived with disability for chronic obstructive pulmonary disease and asthma, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet Respir Med*. 2017;5(9):691-706.
4. Sutradhar I, Das Gupta R, Hasan M, Wazib A, Sarker M. Prevalence and risk factors of chronic obstructive pulmonary disease in Bangladesh: a systematic review. *Cureus*. 2019;11(1).
5. Hawkins NM, Petrie MC, Jhund PS, Chalmers GW, Dunn FG, McMurray JJ. Heart failure and chronic obstructive pulmonary disease: diagnostic pitfalls and epidemiology. *Eur J Heart Fail*. 2009;11(2):130-9.
6. Suskovic S, Kosnik M, Lainscak M. COPD and heart failure: a two-way street. *Eur J Heart Fail*. 2010;12(5):427-9.
7. Sapey E, Stockley RA. COPD exacerbations. 2: aetiology. *Thorax*. 2006;61(3):250-8.
8. Bhatt SP, Dransfield MT. Chronic obstructive pulmonary disease and cardiovascular disease. *Transl Res*. 2013;162(4):237-51.
9. Zeng Q, Jiang S. Update in diagnosis and therapy of coexistent chronic obstructive pulmonary disease and chronic heart failure. *J Thorac Dis*. 2012;4(3):310-5.
10. Tsai SH, Lin YY, Chu SJ, Hsu CW, Cheng SM. Interpretation and use of natriuretic peptides in non-congestive heart failure settings. *Yonsei Med J*. 2010;51(2):151-63.
11. Don-Wauchope AC, McKelvie RS. Evidence based application of BNP/NT-proBNP testing in heart failure. *Clin Biochem*. 2015;48(4-5):236-46.
12. Pavasini R, Tavazzi G, Biscaglia S, Guerra F, Pecoraro A, Zaraket F, et al. Amino terminal pro brain natriuretic peptide predicts all-cause mortality in patients with chronic obstructive pulmonary disease: systematic review and meta-analysis. *Chron Respir Dis*. 2017;14(2):117-26.
13. Celli BR, Wedzicha JA. Update on clinical aspects of chronic obstructive pulmonary disease. *N Engl J Med*. 2019;381(13):1257-66.
14. Ntritsos G, Franek J, Belbasis L, Christou MA, Markozannes G, Altman P, et al. Gender-specific estimates of COPD prevalence: a systematic review and meta-analysis. *Int J Chron Obstruct Pulmon Dis*. 2018;13:1507-14.
15. Siddharthan T, Grigsby MR, Goodman D, Chowdhury M, Rubinstein A, Irazola V, et al.

- Association between household air pollution exposure and chronic obstructive pulmonary disease outcomes in 13 low- and middle-income country settings. *Am J Respir Crit Care Med.* 2018;197(5):611-20.
16. Kim V, Aaron SD. Hypoxemia in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 2019;199(12):1460-1.
 17. Roussos C, Koutsoukou A. Respiratory failure. *Eur Respir J Suppl.* 2003;22(Suppl 47):3s-14s.
 18. O'Brien A, O'Donnell DE, Webb KA. Hypercapnia in COPD: a clinical review. *Chest.* 2020;157(5):1168-78. doi:10.1016/j.chest.2019.12.042.
 19. Kent BD, Mitchell PD, McNicholas WT. Hypoxemia in patients with COPD: cause, effects, and disease progression. *Int J Chron Obstruct Pulmon Dis.* 2011;6:199-208.
 20. Bruton A, Garrod R, Thomas M. Respiratory disease: a case study approach to patient care. 3rd ed. Edinburgh: Elsevier; 2017.
 21. Hurdman J, Condliffe R, Elliot CA, Swift A, Rajaram S, Davies C, et al. Pulmonary hypertension in COPD: results from the ASPIRE registry. *Eur Respir J.* 2013;41(6):1292-301.
 22. Nadar SK, Al Nooryani A. Association of smoking with natriuretic peptides and cardiovascular risk factors. *J Clin Med Res.* 2020;12(6):356-62.
 23. Pavasini R, Tavazzi G, Biscaglia S, Guerra F, Pecoraro A, Zaraket F, et al. Amino terminal pro brain natriuretic peptide predicts all-cause mortality in patients with chronic obstructive pulmonary disease: systematic review and meta-analysis. *Chron Respir Dis.* 2017;14(2):117-26.
 24. Hopkins WE, Chen Z, Fukagawa NK, Hall C, Knot HJ, LeWinter MM. Increased atrial and brain natriuretic peptides in adults with cyanotic congenital heart disease: enhanced understanding of the relationship between hypoxia and natriuretic peptide secretion. *Circulation.* 2004;109(23):2872-7.

Cite this article as: Anach SU, Quadry SA, Hossain ME, Rayhan MA, Dhar D. Association of pro-brain natriuretic peptide with degree of severity and prognostic outcome in patients with acute exacerbation of chronic obstructive pulmonary disease. *Int J Res Med Sci* 2026;14:4995-5003.