Section: Medicine

Original Research Article

ROLE OF CARDIAC BIOMARKERS IN PREDICTION OF OUTCOMES IN PATIENTS HOSPITALISED FOR ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT

Background: Aim: To "analyze the mortality rate and length of hospital stay among patients admitted with acute exacerbation of COPD and to "evaluate the outcomes following hospitalization for acute exacerbation of COPD. **Materials and Methods:** This prospective observational study was conducted in the Department of Medicine, BRD Medical College, Gorakhpur. Prior "approvals were obtained from the College Research Council (CRC) and the Institutional Human Ethics Committee of BRD Medical College. The study was conducted over a period of one year. **Result:** Mortality rate and length of hospital stay among patients admitted with acute exacerbation COPD has analyzed and evaluated the outcome. **Conclusion:** This study provides valuable insights into the clinical course of a cohort of patients presenting with significant respiratory symptoms. The demographic characteristics, biochemical derangements, and the patterns of exacerbations and mortality highlight the complexity of managing these patients.

INTRODUCTION

Chronic "obstructive pulmonary disease (COPD) is defined as a common preventable and treatable disease characterized by persistent and progressive airflow limitation that is caused due to enhanced chronic inflammatory response of the airways and lungs to noxious particles and gases.[1]

COPD is a major cause of morbidity and one of the principal causes of the death worldwide. It is the fourth leading cause of death in the world and COPD is expected to be the third leading cause of death worldwide by 2020.[2,3,4]

Donalson et al.[5] (2010) showed a 2.2-fold higher possibility of MI within a brief 5-day window and a 1.3-fold higher possibility of stroke within 49 days after COPD exacerbation. Regarding the time of onset of a CV event, **Goto et al.**[6] showed that adverse cardiovascular events occur both in short-(30-day) as well as long-term periods (1 year) after AECOPD, while **Wang et al.**[7] showed that the risk of cardiovascular adverse events after hospitalization for AECOPD is high for at least 90 days and is associated with mortality."

The causal nexus between AECOPD and CV events could be explained through the abrupt rise in airway resistance during an exacerbation that substantially reduces lung emptying and expiratory flow.[8]

We "hypothesized that the prognostic value of these indicators in AECOPD patients are still uncertain. This study aimed to demonstrate the utility of circulating biomarkers including TLC, troponin I, and NT-proBNP in predicting prognosis in AECOPD patients.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Medicine, BRD Medical College, Gorakhpur. Prior "approvals were obtained from the College Research Council (CRC) and the Institutional Human Ethics Committee of BRD Medical College. The study was conducted over a period of one year.

Sample Size

The sample size was calculated using the formula:

n= (Z1-α/2)2*p*(1-p)/d2

Where:

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- "n = required sample size"
- "p = 0.627 (prevalence)"
- "α = 0.05 (level of significance)"
- "d = 0.07 (margin of error)"

Based "on the calculation, the required sample size was 183. Considering a 10% loss to follow-up, the final sample size was adjusted to **200**."

Inclusion Criteria

- "Patients aged above 18 years diagnosed with COPD as per GOLD criteria and presenting with acute exacerbation"
- "Patients who provided written informed consent"

Exclusion Criteria

- Patients aged less than 18 years
- Known cases of malignancy or immunosuppression
- Known cases of coronary artery disease
- Active cases of tuberculosis
- Diagnosed pneumonia on chest X-ray
- Patients with asthma
- Patients who did not provide consent

Methodology

A "detailed clinical history was taken from all enrolled patients, followed by a complete general and systemic examination. Patients presenting with clinical features suggestive of COPD, such as progressive dyspnoea, chronic cough, chronic sputum production, a history of exposure to risk factors, or a family history of COPD, were evaluated."

Patients "who fulfilled the criteria for Acute Exacerbation of COPD (AECOPD) were enrolled in the study. AECOPD was diagnosed based on the criteria by Anthonisen et al., which includes the presence of any two of the following symptoms:"

- "Increased cough"
- "Increased purulence and/or volume of expectorations"
- "Increased severity of dyspnoea"

These "patients were promptly treated and stabilized. Informed and written consent was obtained. All patients underwent thorough clinical history evaluation and physical examination. Investigations included complete blood count, renal and liver function tests, cardiac biomarkers (NT-proBNP and troponin I), procalcitonin, chest radiograph, and ECG."

Upon "stabilization, patients underwent 2D echocardiography and pulmonary function testing (PFT). Patients were followed throughout their hospital stay, and short-term outcomes were recorded via telecommunication follow-up."

Statistical Analysis

Data were "analyzed using SPSS software version 26.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard error of the mean (SEM). T-tests and Chi-square tests were used for comparisons. A p-value of <0.05 was considered statistically significant. Fisher's exact test was applied for categorical data distribution comparisons.

RESULTS

Table 1: Distribution of the studied patients based on age group

Age group	Number of patients (n=200)	Percentage
≤40 year	9	4.5%
41-50 year	23	11.5%
51-60 year	54	27.0%
61-70 year	64	32.0%
>70 year	50	25.0%
MEAN±SD	64.0±11.9	

The majority were in the older age groups, with 32.0% (n=64) between 61-70 years, 27.0% (n=54) between 51-60 years, and 25.0% (n=50) above 70 years. The mean age was 64.0±11.9 years, indicating an older patient population. Only a small proportion of patients were in the younger age groups, with 4.5% (n=9) ≤40 years and 11.5% (n=23) between 41-50 years

Table 2: Distribution of the studied patients based on gender

Gender	Number of patients (n=200)	Percentage
Male	81	40.5%
Female	119	59.5%

A slightly higher proportion of females (59.5%, n=119) compared to males (40.5%, n=81), indicating a female predominance in the patient population.

Table 3: Distribution of the studied patients based on chief complaints

Chief complain	Number of patients (n=200)	Percentage
Shortness of breath	200	100.0%
Cough	120	60.0%
Sputum	66	33.0%
Fiver	54	27%

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All 200 patients (100.0%) presented with shortness of breath as their primary complaint. Additionally, cough was reported in 60.0% (n=120) of patients, sputum production in 33.0% (n=66), and fever in 27.0% (n=54) of patients, indicating a significant respiratory symptom burden in the study population.

Table 4: Vital Signs on Admission

Vitals on admission	MEAN	SD
Puls rate	86.26	8.15
SBP (mmHg)	118.25	18.20
DBP (mmHg)	74.68	13.12
RR	21.1	1.65
SpO2 (%)	88.2	10.6

The biochemical parameters of the studied patients on admission revealed the following values: pH (7.25 ± 0.61), Pco2 (62.15 ± 23.5), pO2 (63.4 ± 53.6), HCO3 (31.8 ± 10.3), total leukocyte count (TLC) (13800.4 ± 7114), hemoglobin (Hb) (11.6 ± 2.2), serum creatinine (1.5 ± 3.1), sodium (Na) (137.8 ± 12.5), potassium (K) (4.20 ± 1.2), serum procalcitonin (1.75 ± 6.8), troponin I (0.576 ± 3.02), and NT pro BNP (986.7 ± 1867.0).

Table 5: Biochemical Parameters on Admission

Biochemical parameters on admission	MEAN±SD	MEDIAN
Ph	7.25±0.61	7.3
Pco2	62.15±23.5	60.55
pO2	63.4±53.6	49.2
HCO3	31.8±10.3	29.7
TLC	13800.4±7114	1285.0
Hb	11.6±2.2	11.6
S. Creatinine	1.5±3.1	1.09
Na	137.8±12.5	138.8
K	4.20±1.2	4.175
S. Procalcitonin	1.75±6.8	0.075
Troponin I	0.576±3.02	0.014
NT pro BNP	986.7±1867	246.7

The biochemical parameters on admission showed a mean pH of 7.25±0.61, indicating acidosis, with a median of 7.3. The mean PCO2 was 62.15±23.5 mmHg, and mean PO2 was 63.4±53.6 mmHg. Other notable findings included elevated mean TLC (13800.4±7114 cells/μL), slightly low mean hemoglobin (11.6±2.2 g/dL), and elevated mean NT pro BNP (986.7±1867 pg/mL). The median values for S. Procalcitonin (0.075 ng/mL) and Troponin I (0.014 ng/mL) were relatively low, suggesting minimal infection and cardiac damage in most patients, although mean values were skewed by outliers.

Table 6: Distribution of NT Pro BNP

Category	Number of patients (n=200)	Percentage
High (≥300)	90	45.0
Normal (<300)	110	55.0

Table 7: Distribution of Troponin I

Category	Number of patients (n=200)	Percentage
High (≥0.05)	64	32.0%
Normal (<0.05)	136	68.0%

Troponin I levels revealed that 136 patients (68.0%) had normal levels (<0.05), while 64 patients (32.0%) exhibited high levels (≥0.05).

Table 8: Both (Troponin I and NT Pro BNP) elevated

Category	Number of patients (n=200)	Percentage
High	34	17.0%
Normal	166	83.0%

Among the studied patients, 17.0% (n=34) exhibited elevated levels of both Troponin I and NT-proBNP, while the majority (83.0%, n=166) had normal levels for both biomarkers.

Table 9: Follow up

Follow up	Number of patients (n=200)	Percentage
Completed	179	89.5%
Loss to follow up	21	10.5%

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Out of the 200 patients, 179 (89.5%) completed the follow-up, while 21 patients (10.5%) were lost to follow-up, indicating a relatively high follow-up rate and minimal attrition bias in the study.

Table 10: Distribution of Exacerbations with in 30 and 60 Days

Number of Exacerbations			Number of patients	Percentage
30 Days (n=179)	Yes		114	63.7
	No		65	36.3
	If yes (n=114)	1	94	82.5
		2	9	7.9
		3	10	8.8
		4	1	0.9
60 Days (n=133)	Yes		20	15.0
	No		113	85.0
	If yes (n=20)	1	11	55.0
		2	6	30.0
		3	1	5.0
		5	2	10.0

*4 patients' Mortality in the hospital; 17 patients LTFU at 30 days; 4 patients LTFU at 60 days; 42 patients' mortality at 30 days follow-up period; 7 patients' mortality at 60 days follow-up period. Within 30 days, 63.7% (n=114) of patients experienced exacerbations, with 82.5% (n=94) of these patients having one exacerbation. At 60 days, the proportion of patients with exacerbations decreased to 15.0% (n=20), with 55.0% (n=11) of these patients experiencing one exacerbation. The frequency and severity of exacerbations varied between the two timeframes, with more patients experiencing multiple exacerbations within 30 days compared to 60 days.

Table 11: Distribution of Hospital Admissions Within 30 and 60 Days

Number of Hospital Admissions			Number of patients	Percentage
30 Days (n=179)	Yes		106	59.2
	No		73	40.8
	If yes (n=106)	1	92	86.8
		2	6	5.7
		3	6	5.7
		4	1	0.9
		6	1	0.9
60 Days (n=133)	Yes		12	9.0
	No		121	91.0
	If yes (n=12)	1	8	66.7
		2	3	25.0
		3	1	8.3

Within 30 days, 59.2% (n=106) of patients required hospital admissions, with 86.8% (n=92) of these patients having one admission. At 60 days, hospital admissions decreased to 9.0% (n=12), with 66.7% (n=8) of these patients having one admission. The frequency of hospital admissions varied, with a higher proportion of patients requiring multiple admissions within 30 days compared to 60 days.

Table 12: Distribution of the studied patients based on survival outcomes

Outcome	Number of patients (n=179)	Percentage
Expire	56	31.3%
Alive	123	68.7%

Among the 179 patients, 68.7% (n=123) survived, while 31.3% (n=56) expired, indicating a mortality rate of approximately one-third of the study population.

Table 13: Comparison of Biochemical parameters with exacerbation and non exacerbation

At 30 Days	Exacerbations (n=114)	No Exacerbations (n=65)	P-value
	MEAN±SD	MEAN±SD	
Ph	7.28±0.1	7.34±0.1	**0.001**
Pco2	65.25±26.9	58.37±17.1	0.058
TLC	14310.5±8277	13369.4±6606	0.423
Troponin I	0.193±1.0	0.67±0.4	**<0.001**
NT pro BNP	1202.5±1120	709.9±830	**0.001**
PO2	65.0±22.2	61.8±26.2	0.386
HCO3	31.6±10.5	31.4±8.7	0.896
Hb	11.34±2.3	11.4±2.1	0.862
creatinine	1.72±0.3	1.14±0.4	0.001
Na	139.0±6.8	138.5±6.1	0.624
K	4.47±1.3	3.68±0.9	0.001

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Patients with exacerbations at 30 days had significantly lower pH (7.28±0.1 vs 7.34±0.1, p=0.001), higher NT pro BNP (1202.5±1120 vs 709.9±830, p=0.001), higher creatinine (1.72±0.3 vs 1.14±0.4, p=0.001), and higher potassium levels (4.47±1.3 vs 3.68±0.9, p=0.001) compared to those without exacerbations. Additionally, Troponin I levels were significantly lower in the exacerbation group (0.193±1.0 vs 0.67±0.4, p<0.001). However, there were no significant differences in TLC, PO2, HCO3, Hb, and sodium levels between the two groups.

Table 14: Comparison of Biochemical parameters with 1 Exacerbations and >1 Exacerbations

At 30 Days	1 Exacerbations (n=94)	>1 Exacerbations (n=20)	P-value
	MEAN±SD	MEAN±SD	
Ph	7.29±0.1	7.26±0.1	0.225
Pco2	63.7±24.4	72.9±35.8	0.164
TLC	14796.6±8814	12025±4363	0.164
Troponin I	0.225±0.1	0.048±0.1	**<0.001**
NT pro BNP	1286.8±1212	806.6±1121	0.106

Patients with >1 exacerbations at 30 days had lower Troponin I levels (0.048±0.1 vs 0.225±0.1, p<0.001) compared to those with 1 exacerbation. However, there were no significant differences in pH, PCO2, TLC, and NT pro BNP levels between the two groups, although patients with >1 exacerbations tended to have higher PCO2 levels (72.9±35.8 vs 63.7±24.4, p=0.164).

Table 15: Comparison of parameters with exacerbation and non exacerbation at 60 days

60 Days		Exacerbations (MEAN±SD) (n=11)	No Exacerbations (MEAN±SD) (n=50)	P-value
Age		66.2±6.8	63.4±12.7	0.82
Gender	Male	8 (72.7%)	21 (42.0%)	0.064
	Female	3 (27.3%)	29 (58.0%)	
Ph		7.32±0.1	7.35±0.1	0.234
PCO2		55.90±16.6	57.92±16.4	0.713
PO2		58.95±26.7	63.0±22.2	0.599
HCO3		25.68±8.5	33.0±8.2	0.009
TLC		14935.5±8272	13210±6444	0.448
Hb		13.5±2.0	11.0±1.9	0.001
creatinine		1.19±0.4	1.14±0.4	0.708
Na		128.9±29.0	140.0±11.3	0.038
K		3.52±1.1	3.71±0.8	0.508
TROP I		0.250±0.3	0.840±0.9	0.036
NT ProNB		1218.1±639	628.4±835	0.031

At 60 days, patients with exacerbations had significantly lower HCO3 levels (25.68±8.5 vs 33.0±8.2, p=0.009), lower sodium levels (128.9±29.0 vs 140.0±11.3, p=0.038), and lower Troponin I levels (0.250±0.3 vs 0.840±0.9, p=0.036) compared to those without exacerbations. Additionally, patients with exacerbations had higher NT pro BNP levels (1218.1±639 vs 628.4±835, p=0.031) and higher hemoglobin levels (13.5±2.0 vs 11.0±1.9, p=0.001). There were no significant differences in age, pH, PCO2, PO2, TLC, creatinine, and potassium levels between the two groups.

Table 16: Comparison of parameters with survivors and non-survivors at 30 days

Parameters		Survivors (MEAN±SD) (n=137)	Non-survivors (MEAN±SD) (n=46)	P-value
Age		63.77±12.6	62.5±10.6	0.528
Gender	Male	52 (38.0%)	18 (39.1%)	0.887
	Female	85 (62.0%)	28 (60.9%)	
Ph		7.33±0.2	7.25±0.1	<0.001
PCO2		60.8±21.6	68.16±29.5	0.072
PO2		61.4±45.0	72.6±76.3	0.230
HCO3		31.77±8.8	32.3±15.0	0.758
TLC		13982.4±8360	13876.0±5484	0.936
Hb		11.3±2.1	11.6±2.2	0.542
creatinine		1.51±3.7	1.5±1.1	0.936
Na		139.3±12.1	137.3±7.2	0.290
K		3.98±1.1	4.75±1.31	<0.001
TROP I		0.40±0.4	0.28±0.5	0.006
NT ProNB		740.0±954	1841.1±1232	<0.001

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Compared to survivors, patients who died within 30 days had significantly lower pH levels (7.33±0.2 vs 7.25±0.1, p=0.001), higher potassium levels (3.98±1.1 vs 4.75±1.31, p=0.001), and lower Troponin I levels (0.40±0.4 vs 0.28±0.5, p=0.006). Additionally, patients who died had significantly higher NT pro BNP levels (740.0±954 vs 1841.1±1232, p=0.001). There were no significant differences in age, gender, PCO2, PO2, HCO3, TLC, hemoglobin, creatinine, and sodium levels between survivors and non-survivors.

Table 17: Comparison of parameters with survivors and non-survivors at 60 days

Parameters		Survivors (MEAN±SD) (n=126)	Non-survivors (MEAN±SD) (n=49)	P-value
Age		63.8±12	62.9±10.7	0.651
Gender	Male	44 (35.8%)	25 (44.6%)	0.258
	Female	79 (64.2%)	31 (55.4%)	
Ph		7.33±0.2	7.26±0.1	0.014
PCO2		61.0±22	67.3±27	0.100
PO2		61.3±47.3	69.9±46.2	0.257
HCO3		32.2±8.7	31.4±14.2	0.643
TLC		14379.6±8587	13274.8±5601	0.379
Hb		11.2±2.9	11.8±2.4	0.127
creatinine		1.57±3.9	1.39±1.0	0.734
Na		139.3±12.6	137.5±7.3	0.312
K		4.0±1.1	4.5±1.3	0.008
TROP I		0.41±0.3	0.29±0.2	0.007
NT ProNB		719.0±980	1694.2±1120	0.001

Compared to survivors, patients who died within 60 days had significantly lower pH levels (7.26±0.1 vs 7.33±0.2, p=0.014), higher potassium levels (4.5±1.3 vs 4.0±1.1, p=0.008), and lower Troponin I levels (0.29±0.2 vs 0.41±0.3, p=0.007). Additionally, patients who died had significantly higher NT pro BNP levels (1694.2±1120 vs 719.0±980, p=0.001). There were no significant differences in age, gender, PCO2, PO2, HCO3, TLC, hemoglobin, creatinine, and sodium levels between survivors and non-survivors.

DISCUSSION

COPD "is increasingly recognized to have a profound effect on the overall health and economy globally. In 2021, the World Health Organization (WHO) declared that COPD is the third leading cause of death, and the seventh-leading cause of morbidity worldwide. Acute exacerbation of the disease affects health status and leads to a faster deterioration in lung function with associated morbidity and mortality. Additionally, exacerbations are associated with a decline in daily physical activity and the overall quality of life.[9]

The lack of effective predictive scores in the hospitals may lead to improper risk-stratification in AECOPD patients. This situation may confuse decision-making regarding treatment escalation, early discharge, and severity categorization."

This study "provides a comprehensive examination of a patient cohort, shedding light on their demographic profile, presenting symptoms, biochemical status upon admission, and their subsequent clinical course, including exacerbation rates and survival outcomes. The findings offer valuable insights into the characteristics of this patient population and highlight key factors associated with adverse events."

1. Demographic Profile and Clinical Presentation:

In the present study, most patients, nearly 80% were more than 50yr of age with a mean age of 64.0±11.9 years. 51-60 and 61-70 age groups, each comprise 32.0% and 27.0% respectively. The >70 years age group accounts for 25.0%. Females constitute the majority, making up 59.5% of the patient population, while males account for 40.5%. Our findings were comparable to the findings of **Mathialagan MG et al,**[10] reported that among 200 patients, the age ranged from 31 to 82 years, with a mean age of 59 years. As seen in our study and across all other studies COPD is a disease predominant in the elderly probably because most of the cases are acquired due to long term exposure to various causative agents like smoke etc.

Regarding gender, the study observed a slight female predominance, with 59.5% (n=119) of the patients being female compared to 40.5% (n=81) male. This is in contrast to the results of the other studies mentioned above which found a male predominance. The increased female predominance could be explained by the fact that majority of the population did not have a history of smoking. The underlying cause of COPD in these patients could be exposure to non tobacco smoke, environmental or household. A female predominance suggests household smoke exposure as a common cause of COPD in this area. Ours was a mostly rural population where exposure to household smoke is common.

In the present study, all 200 patients (100.0%) presented with shortness of breath as their primary complaint. Additionally, cough was reported in 60.0% (n=120) of patients, sputum production in 33.0% (n=66), and fever in 27.0% (n=54) of patients,

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indicating a significant respiratory symptom burden in the study population.

2. Initial Physiological and Biochemical Status: Indicators of Acute Illness and Organ Strain

Biochemical findings included an elevated mean total leukocyte count (TLC) of 13800.4 ± 7114 cells/µL. Most COPD exacerbations are due to viral infections and the rise in TLC could be a marker of the inflammation in airways associated with the infection. Mean hemoglobin levels were slightly low at 11.6 ± 2.2 g/dL, suggesting a degree of anemia. Anemia is not expected in COPD as the persistent hypoxia gives rise to polycythemia. The presence of anemia in our patient population could be explained by co-existing nutritional deficiencies as majority of patients were females. We did not do Iron profile testing and could not conclude the cause of anemia seen in the patient population. Existence of anemia in COPD has also been reported by **Florian Kollert et al.**[11]

There was elevation of NT-pro BNP in 45% (n=90) patients. the mean value in these patients was 2089.64 pg/ml. NT-proBNP was significantly elevated at 986.7 ± 1867 pg/mL. Elevated BNP/NT-proBNP concentrations have been identified as prognostic markers for pulmonary hypertension and mortality in chronic lung disease. A study on midregional proatrial natriuretic peptide (MR-proANP), a related natriuretic peptide, found significantly higher levels on admission for exacerbation compared to recovery and stable states, and that MR-proANP levels were higher in non-survivors. Elevated NT-proBNP is also associated with cardiovascular mortality and ventricular dysfunction in acute exacerbations of COPD.

The mean Troponin I level was 0.576 ± 3.02 ng/mL. However, the median values for Troponin I (0.014 ng/mL) was relatively low. This discrepancy between mean and median values indicates that a few outliers with very high values skewed the mean, suggesting that while some patients might have had cardiac damage, these conditions were not widespread across the majority of the cohort. 32.0% (n=64) of patients did exhibit high Troponin I levels (≥0.05 ng/mL). 17.0% (n=34) of the patients presented with elevated levels of both Troponin I and NT-proBNP, pointing to a subset of patients with concurrent cardiac stress and potential injury. High-sensitivity cardiac troponin I (hs-cTnI) levels above the risk stratification threshold are present in a significant percentage of individuals with COPD, indicating its prognostic importance in myocardial injury and ischemia.

Other abnormalities at admission included the presence of Respiratory acidosis (partial compensated), mean PO2 levels of 63.4. Mean SBP of 118.25mmhg.

There were only 4 deaths during the initial hospitalization and most patients did not require invasive ventilatory support for management.

3. Outcome and Follow up

Mortality: "We had planned for a follow up of 90 days but till the time of data analysis, follow up at 90 days could be completed in only 11 patients enrolled initially and was pending in 112 patients. 60 day follow up data is available for all patients enrolled in the study. Till 60 days, a total of 21 were lost, with the majority (17) lost to follow up in the first 30 days. There was a high percentage of patients who expired within the first 30 day (n=42). The most common reason being repeat exacerbations. Comparing survivors to those who died within 60 days revealed several critical differences. Non-survivors exhibited significantly lower pH levels (7.26±0.1 vs 7.33±0.2, p=0.014), indicating persistent or worsening acidosis. They also exhibited significantly higher potassium levels (4.5±1.3 vs 4.0±1.1, p=0.008), which can be indicative of severe metabolic derangement or organ dysfunction. Similar to the exacerbation findings, Troponin I levels were significantly lower in the mortality group (0.29±0.2 vs 0.41±0.3, p=0.007), a finding that warrants careful interpretation and further research. Studies have explored the prognostic value of cardiac troponins in relation to long-term mortality in chronic obstructive pulmonary disease. Crucially, non-survivors had significantly higher NT-proBNP levels (1694.2±1120 vs 719.0±980, p=0.001), strongly suggesting that cardiac stress and dysfunction play a pivotal role in mortality. Cardiac biomarkers, including NT-proBNP and troponin T, are indeed associated with long-term outcomes of COPD exacerbations. Age, gender, PCO2, PO2, HCO3, TLC, hemoglobin, creatinine, and sodium levels did not show significant differences between survivors and non-survivors. Studies indicate that cardiac biomarkers, clinical variables, and exacerbation type all play a role in the long-term prognosis of COPD exacerbation. Many of these inflammatory and physiological mechanisms are likely to worsen during exacerbations of COPD. In a retrospective analysis of 897 patients, **Brekke et al,**[12] found that raised troponin levels were predictive of mortality at 1.9 years. Complementary information comes from further retrospective studies. **Marcun R et al,**[13] "finding that NT-proBNP on admission predicts mortality at 6 months is however not novel. Importantly, however, neither the discharge level nor a reduction in level of at least 30% (which had previously been reported to be predictive for short term outcome after hospitalisation for heart failure);[14] was associated with any of the outcomes. Diametrically different were the results for TnT, where discharge but not admission levels were predictive for 6-month rehospitalizations. These differences are relevant for clinical practice and could guide patient management. In patients with an acute exacerbation of COPD and in the absence of specific clinical indications admission natriuretic peptides and discharge troponin should be measured whilst additional measurement of the individual biomarker does not seem to improve patient characterisation and risk stratification."

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Recurrent exacerbations: "The analysis of exacerbations revealed a dynamic pattern over time. Within 30 days, a substantial proportion of patients, 63.7% (n=114), experienced exacerbations. The deaths that occurred during the first 30 days occurred in these patients who had an exacerbation. Of the surviving 69 only 9 had a repeat exacerbation within the next 30 days. Late exacerbations beyond 30 days occurred only in 11 patients. This decline in exacerbation rates over time highlights a period of stabilization or improvement in patient condition for a significant portion of the cohort. The variation in frequency and severity of exacerbations between the two timeframes indicates that short-term and medium-term clinical courses may differ.

On comparing the paramenters that could predict an early exacerbation, we found that patients who experienced exacerbations within 30 days had significantly lower pH levels (7.28±0.1 vs 7.34±0.1, p=0.001), at enrollment. They also had significantly higher NT-proBNP levels (1202.5±1120 vs 709.9±830, p=0.001), higher creatinine (1.72±0.3 vs 1.14±0.4, p=0.001), and higher potassium levels (4.47±1.3 vs 3.68±0.9, p=0.001. Troponin I levels were significantly lower in the exacerbation group at 30 days (0.193±1.0 vs 0.67±0.4, p<0.001). These findings were similar in nature to found in relation to mortality."

At 60 "days, patients with exacerbations had significantly lower HCO3 levels (25.68±8.5 vs 33.0±8.2, p=0.009) and lower sodium levels (128.9±29.0 vs 140.0±11.3, p=0.038). Lower Troponin I levels (0.250±0.3 vs 0.840±0.9, p=0.036) were again observed in the exacerbation group. Conversely, they had higher NT-proBNP (1218.1±639 vs 628.4±835, p=0.031) and higher hemoglobin levels (13.5±2.0 vs 11.0±1.9, p=0.001). The higher hemoglobin in the exacerbation group at 60 days might suggest a compensatory mechanism in response to chronic hypoxemia or even a degree of dehydration. No significant differences were noted in age, pH, PCO2, PO2, TLC, creatinine, and potassium levels at this time point.

CONCLUSION

In conclusion, "this study provides valuable insights into the clinical course of a cohort of patients presenting with significant respiratory symptoms. The demographic characteristics, biochemical derangements, and the patterns of exacerbations and mortality highlight the complexity of managing these patients.

REFERENCES

1. Global Initiative for Chronic Obstructive Lung Disease – Global Initiative for Chronic Obstructive Lung Disease – GOLD [Internet]. Global Initiative for Chronic Obstructive Lung Disease – GOLD. 2017. Available from: http://goldcopd.org.
2. Global initiative for chronic obstructive lung disease (GOLD) Global strategy for the diagnosis, management, and prevention of COPD. 2011.
3. Buenviaje MB (1989). Quantitative Sputum Culture and Gram Stain: Pulmonary Infection vs. Colonization. Phil J Microbiol Infect Dis; 18(1):28-35
4. Ziyade N, Yagci A. (2010): Improving Sputum Culture Results for Diagnosis of Lower Respiratory Tract By Saline Washing. Marmara Medical Journal 2010; 23 (1);30-36
5. Donaldson, G.C.; Hurst, J.R.; Smith, C.J.; Hubbard, R.B.; Wedzicha, J.A. Increased risk of myocardial infarction and stroke following exacerbation of COPD. Chest 2010, 137, 1091–1097.
6. Goto, T.; Shimada, Y.J.; Faridi, M.K.; Camargo, C.A., Jr.; Hasegawa, K. Incidence of Acute Cardiovascular Event After Acute Exacerbation of COPD. J. Gen. Intern. Med. 2018, 33, 1461–1468.
7. Wang, M.; Lin, E.P.; Huang, L.C.; Li, C.Y.; Shyr, Y.; Lai, C.H. Mortality of Cardiovascular Events in Patients With COPD and Preceding Hospitalization for Acute Exacerbation. Chest 2020, 158, 973–985.
8. Rabe, K.F.; Hurst, J.R.; Suissa, S. Cardiovascular disease and COPD: Dangerous liaisons. Eur. Respir. Rev. 2018, 27, 185057.
9. Almarshoodi K, Echevarria C, Kassem A, Mahboub B, Salameh L, and Ward C. An International Validation of the "DECAF Score" to Predict Disease Severity and Hospital Mortality in Acute Exacerbation of COPD in the UAE. Hospital Pharmacy 2024;59(2):234–240
10. Mathialagan MG, Preethi J, Manikandan V, Vinoth R. DECAF score at admission as a tool to predict clinical outcome in acute exacerbation of COPD. Asian Journal of Medical Sciences. 2024;5(3):156-160
11. Florian Kollert, Carolin Müller, Andrea Tippelt, Rudolf Jorres, Dominic Heidinger, et al. ANAEMIAIN CHRONIC RESPIRATORY FAILURE. International Journal of Clinical Practice, 2011, 65 (4),pp.479. ff10.1111/j.1742-1241.2011.02631.xff. ffhal-00623801ff
12. Brekke PH, Omland T, Holmedal SH, Smith P, Søyseth V. Troponin T elevation and long-term mortality after chronic obstructive pulmonary disease exacerbation. Eur Respir J 2008;31:563–70.
13. Marcun R, Sustic A, Brguljan PM, Kadivec S, Farkas J, Kosnik M, et al. Cardiac biomarkers predict outcome after hospitalisation for an acute exacerbation of chronic obstructive pulmonary disease. Int J Cardiol 2012 Nov 29;161(3):156-9.
14. Maisel A, Mueller C, Adams Jr K, et al. State of the art: using natriuretic peptide levels in clinical practice. Eur J Heart Fail 2008;10:824–39.

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