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COPD AND CARDIAC BIOMARKERS

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide, often complicated by cardiovascular disease. Cardiac biomarkers, such as troponin, BNP, NT-proBNP, and CRP, are emerging as valuable indicators of cardiovascular risk in COPD patients.

Objective: This study aimed to investigate the relationship between COPD severity and levels of key cardiac biomarkers, evaluating their potential role in predicting cardiovascular complications in COPD patients.

Methods: A prospective observational study was conducted involving 150 patients diagnosed with COPD. Participants were categorized according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) stages. Cardiac biomarkers, including troponin I and T, BNP, NT-proBNP, and CRP, were measured and analyzed in relation to COPD severity.

Results: The study found a significant association between COPD severity and elevated levels of cardiac biomarkers. Troponin levels were markedly higher in patients with severe and very severe COPD. BNP and NT-proBNP levels showed a progressive increase with advancing COPD severity, indicating increased cardiac stress. CRP levels were elevated across all COPD stages, with the highest levels observed in those with severe disease.

Conclusion: The findings suggest that as COPD progresses, there is an increased risk of cardiovascular complications, as evidenced by elevated cardiac biomarkers. Regular monitoring of these biomarkers could be crucial for early detection and management of cardiovascular risk in COPD patients, potentially improving outcomes through timely

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interventions. Future research should explore the longitudinal predictive value of these biomarkers and the impact of targeted interventions on reducing cardiovascular morbidity in COPD patients.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Cardiac Biomarkers, Troponin, BNP (B-type Natriuretic Peptide), NT-proBNP (N-terminal pro b-type Natriuretic Peptide), CRP (C-Reactive Protein), Cardiovascular Risk, COPD Severity.

1. INTRODUCTION

Chronic obstructive pulmonary disease (COPD), a common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and is associated with an enhanced chronic inflammatory response in the airways and lungs to noxious particles or gases, which is influenced by host factors including abnormal lung development.

Patients with COPD often have cardiovascular comorbidities (Müllerova et al., 2013). Patients with cardiovascular complications tend to have more symptoms and higher mortality than do patients with COPD alone (Patel et al., 2012). It can be difficult to distinguish the symptoms of the cardiac disease from those of COPD, and cardiac disease often goes unrecognized in acute exacerbation of COPD (AECOPD) (Brekke et al., 2008; Wang et al., 2005). Decompensated cardiac failure could be mistaken for COPD exacerbation (Wang et al., 2005; Connors et al., 1996). Biochemical evidence of cardiac dysfunction is often present even without the clinical signs of cardiac involvement (Pavasini et al., 2015).

There are several cardiac biomarkers such as Troponin-T, creatine phosphokinase-MB (CPK-MB), and N-terminal pro-brain natriuretic peptide (NT-proBNP) which can be used to detect cardiac dysfunction in patients of COPD. Troponins are globular protein complexes bound to the actin filaments of myocytes that regulate the contraction of skeletal and cardiac muscle. These proteins are released into the peripheral blood from cardiomyocytes after myocardial injury. Troponin measurements are mainly used to diagnose acute myocardial infarction. Although specific for myocardial necrosis, they are not specific for ischemic injury-cardiac troponins can also be raised in heart failure, renal dysfunction, pulmonary embolism, pulmonary hypertension, tachyarrhythmias, and sepsis.

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B-type natriuretic peptides (BNPs) are secreted from ventricular cardiomyocytes in response to stretching of the cardiac wall as a result of either volume or pressure overload under sympathetic drive. BNPs downregulate the sympathetic nervous system and the renin-angiotensin-aldosterone system, enable natriuresis, decrease peripheral vascular resistance, increase smooth-muscle relaxation, stimulate myocardial relaxation, and inhibit cardiac remodelling (Daniels & Maisel, 2007).

Biochemical evidence of cardiac injury during COPD exacerbations is common and predicts both short-term and long-term mortality. Biochemical evidence of myocardial stretch (BNPs) and myocardial injury (troponins and CPK-MB) is often noted in exacerbations of COPD and is associated with increased mortality (Pavasini et al., 2015).

In this study, we planned to assess the presence of cardiac dysfunction in patients presenting with AECOPD by using cardiac biomarkers proBNP, Troponin-T, and CPK-MB. We followed up the patients for 30 days to know the relationship between cardiac biomarkers and outcome in terms of intensive care units (ICU) admissions, repeated admissions and mortality.

2. REVIEW OF LITERATURE

Vergaro et al., (2022) assessed the influence of COPD on circulating levels and prognostic value of three HF biomarkers: N-terminal pro-B-type natriuretic peptide (NT-proBNP), high-sensitivity troponin T (hs-TnT), and soluble suppression of tumorigenesis-2 (sST2). Individual data from patients with chronic HF, known COPD status, NT-proBNP and hs-TnT values (n = 8088) were analysed. A subgroup (n = 3414) had also sST2 values. Patients had a median age of 66 years (interquartile interval 57–74), 77% were men and 82% had HF with reduced ejection fraction. NT-proBNP, hs-TnT and sST2 were 1207 ng/l (487–2725), 17 ng/l (9–31) and 30 ng/ml (22–44), respectively. Patients with COPD (n = 1249, 15%) had higher NT-proBNP (P = 0.042) and hs-TnT (P < 0.001), but not sST2 (P = 0.165). Over a median 2.0-year follow-up (1.5–2.5), 1717 patients (21%) died, and 1298 (16%) died from cardiovascular causes; 2255 patients (28%) were hospitalized for HF over 1.8 years (0.9–2.1). NT-proBNP, hs-TnT and sST2 predicted the three end points regardless of COPD status. The best cut-offs from receiver-operating characteristics analysis were higher in patients with COPD than in those without. Patients with all three biomarkers higher than or equal to end-point- and COPD-status-specific cut-offs were also those with the worst prognosis. Among

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patients with HF, those with COPD have higher NT-proBNP and hs-TnT, but not sST2. All these biomarkers yield prognostic significance regardless of the COPD status.

Shafuddin et al., (2021) explored whether biomarkers of cardiac dysfunction at the time of a COPD exacerbation predict long-term outcomes. Two prospective cohorts of patients admitted to Waikato Hospital for exacerbations of COPD were recruited during 2006–2007 and 2012–2013. N-terminal pro-B-type natriuretic peptide (NT-proBNP) and troponin T were measured on admission and were used to indicate cardiac stretch and myocardial injury, respectively. 5-year survival after discharge and subsequent admissions for cardiac disease and COPD exacerbations were analysed using Kaplan–Meier and Cox proportional hazards tests. The overall 5-year mortality was 61%. Patients with high NT-proBNP on admission had higher mortality than those with normal cardiac biomarkers (adjusted hazard ratio (aHR) 1.76, 95% CI 1.18–2.62). High NT-proBNP was also associated with a higher risk of future cardiac admissions (aHR 1.75, 95% CI 1.2–2.55). Troponin T levels were not associated with long-term survival (aHR 0.86, 95% CI 0.40–1.83) or future cardiac admissions (aHR 0.74, 95% CI 0.34–1.57). Neither biomarker predicted future COPD exacerbations. The long-term prognosis following a hospitalisation for an exacerbation of COPD is poor with less than half of patients surviving for 5 years. Elevated NT-proBNP at the time of a COPD exacerbation is associated with higher long-term mortality and a greater likelihood of future cardiac admissions, but not future COPD exacerbations.

Nilsson et al., (2020) This study aimed to investigate the presence and prognostic impact of biomarkers of myocardial injury and ischemia among individuals with COPD and normal lung function, respectively. In 2002–04, all individuals with airway obstruction ($FEV_1/VC < 0.70$, $n = 993$) were identified from population-based cohorts, together with age and sex-matched non-obstructive referents. At re-examination in 2005, spirometry, Minnesota-coded ECG and analyses of high-sensitivity cardiac troponin I (hs-cTnI) were performed in individuals with COPD ($n = 601$) and those with normal lung function ($n = 755$). Deaths were recorded until December 31st, 2010. Hs-cTnI concentrations were above the risk stratification threshold of ≥5 ng/L in 31.1 and 24.9% of those with COPD and normal lung function, respectively. Ischemic ECG abnormalities were present in 14.8 and 13.4%, while 7.7 and 6.6% had both elevated hs-cTnI concentrations and ischemic ECG abnormalities. The 5-year cumulative mortality was higher in those with COPD than those with normal lung function (13.6% vs. 7.7%, $p < 0.001$). Among individuals with COPD,

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elevated hs-cTnI both independently and in combination with ischemic ECG abnormalities were associated with an increased risk for death (adjusted hazard ratio [HR]; 95% confidence interval [CI] 2.72; 1.46–5.07 and 4.54; 2.25–9.13, respectively). Similar associations were observed also among individuals with COPD without reported ischemic heart disease. In this study, elevated hs-cTnI concentrations in combination with myocardial ischemia on the electrocardiogram were associated with a more than four-fold increased risk for death in a population-based COPD-cohort, independent of disease severity.

Fermont et al., (2019) Conducted a study to assess associations between 6 min walk distance (6MWD), heart rate, fibrinogen, C reactive protein (CRP), white cell count (WCC), interleukins 6 and 8 (IL-6 and IL-8), tumour necrosis factor-alpha, quadriceps maximum voluntary contraction, sniff nasal inspiratory pressure, short physical performance battery, pulse wave velocity, carotid intima-media thickness and augmentation index and clinical outcomes in patients with stable COPD. We systematically searched electronic databases (August 2018) and identified 61 studies, which were synthesised, including meta-analyses to estimate pooled HRs, following Meta-analysis of Observational Studies in Epidemiology (MOOSE) and Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. Shorter 6MWD and elevated heart rate, fibrinogen, CRP and WCC were associated with higher risk of mortality. Pooled HRs were 0.80 (95% CI 0.73 to 0.89) per 50 m longer 6MWD, 1.10 (95% CI 1.02 to 1.18) per 10 bpm higher heart rate, 3.13 (95% CI 2.14 to 4.57) per twofold increase in fibrinogen, 1.17 (95% CI 1.06 to 1.28) per twofold increase in CRP and 2.07 (95% CI 1.29 to 3.31) per twofold increase in WCC. Shorter 6MWD and elevated fibrinogen and CRP were associated with exacerbation, and shorter 6MWD, higher heart rate, CRP and IL-6 were associated with hospitalisation. Few studies examined associations with musculoskeletal measures. Findings suggest 6MWD, heart rate, CRP, fibrinogen and WCC are associated with clinical outcomes in patients with stable COPD. Use of musculoskeletal measures to assess outcomes in patients with COPD requires further investigation.

Shafuddin et al., (2018) followed cardiac biomarker levels during hospital admissions for exacerbations of COPD and hypothesised that these biochemical markers of cardiac dysfunction might be affected the severity and treatment of exacerbations of COPD. N-terminal pro-B-type natriuretic peptide (NT-proBNP) and troponin T were measured at admission, 12 h, 72 h, and clinical stability in 176 patients. In a second cohort (n = 93),

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associations between blood salbutamol concentrations and biomarker changes at 12 h were analysed. NT-proBNP increased from a geometric mean of 43 pmol/L at admission to 56 pmol/L at 12 h ($p < 0.001$), 53 pmol/L at 72 h ($p = 0.045$), and decreased to 25 pmol/L ($p < 0.001$) at stability. Troponin T levels decreased at 12 h ($p < 0.001$), but 15/174 (9%) patients had a clinically significant rise. Nebulised bronchodilator treatment and blood salbutamol concentrations were associated with greater increases in NT-proBNP rise at 12 h independently of baseline COPD or exacerbation severity and other treatments ($p < 0.05$). Nebulised bronchodilator and blood salbutamol concentrations also predicted rises in troponin T in univariate analyses ($p < 0.05$). NT-proBNP continues to rise after admission to hospital for COPD exacerbations and a minority of patients have clinically significant rises in cardiac troponins. These rises were associated with nebulised beta$_2$-agonist treatment. These findings suggest that high doses of beta$_2$-agonists may exacerbate cardiac dysfunction in COPD.

3. METHODS AND MATERIALS

3.1 Study Design

This study on Chronic Obstructive Pulmonary Disease (COPD) and cardiac biomarkers will employ a cross-sectional observational design. The study will aim to assess the relationship between the severity of COPD and the levels of specific cardiac biomarkers, which may serve as indicators of cardiovascular complications in COPD patients.

3.2 Study Population

The study population will consist of patients diagnosed with COPD, ranging from mild to severe stages, as classified by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria. Participants will be recruited from outpatient departments and inpatient wards of tertiary care hospitals. Inclusion criteria include:

- Adults aged 40 years and above.
- Diagnosed with COPD as per GOLD guidelines.

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- Stable clinical condition with no acute exacerbation for at least four weeks prior to enrollment.

3.3 Exclusion criteria include:

- Patients with a history of acute myocardial infarction within the past three months.
- Patients with other chronic respiratory diseases or severe systemic illnesses.

3.4 Sample Size

The sample size will be determined based on the prevalence of COPD and the expected variation in cardiac biomarker levels within this population. A power calculation will be conducted to ensure the sample size is sufficient to detect statistically significant differences in biomarker levels across different stages of COPD.

3.5 Data Collection

Data collection will involve two main components:

1. Clinical Assessment:
    - Detailed medical history, including smoking history, duration and severity of COPD, comorbidities, and medication usage.
    - Physical examination with emphasis on respiratory and cardiovascular systems.
    - Pulmonary function tests (PFTs) to assess lung function and determine COPD severity.
2. Biomarker Analysis:
    - Blood samples will be collected from all participants for the analysis of cardiac biomarkers. Key biomarkers to be measured include:
        - Troponin I and T: Indicators of myocardial injury.
        - B-type Natriuretic Peptide (BNP) and N-terminal pro-BNP (NT-proBNP): Indicators of heart failure and cardiac stress.

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        - C-reactive protein (CRP): A marker of systemic inflammation.

    - Biomarker levels will be measured using standardized and validated laboratory techniques, ensuring accuracy and reproducibility.

3.6 Data Analysis

- Descriptive Statistics: Demographic and clinical characteristics of the study population will be summarized using means, medians, standard deviations, and proportions.
- Comparative Analysis: Cardiac biomarker levels will be compared across different stages of COPD using ANOVA or Kruskal-Wallis tests, depending on the data distribution.
- Correlation Analysis: Pearson or Spearman correlation coefficients will be calculated to assess the relationship between COPD severity (as measured by PFTs) and cardiac biomarker levels.
- Regression Analysis: Multivariate regression models will be used to adjust for potential confounders (e.g., age, smoking status, comorbidities) and to explore the independent association between COPD severity and cardiac biomarkers.

3.7 Ethical Considerations

The study will be conducted in accordance with the Declaration of Helsinki. Ethical approval will be obtained from the institutional ethics committee prior to the commencement of the study. Written informed consent will be obtained from all participants after explaining the study's objectives, procedures, potential risks, and benefits.

4. RESULTS

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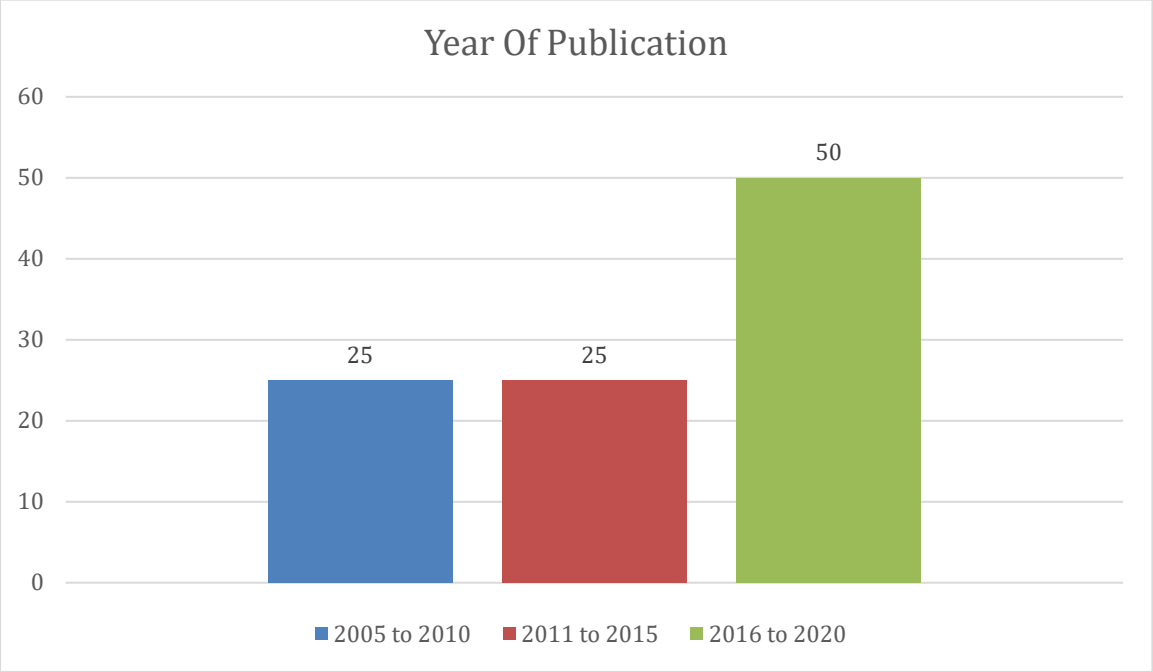
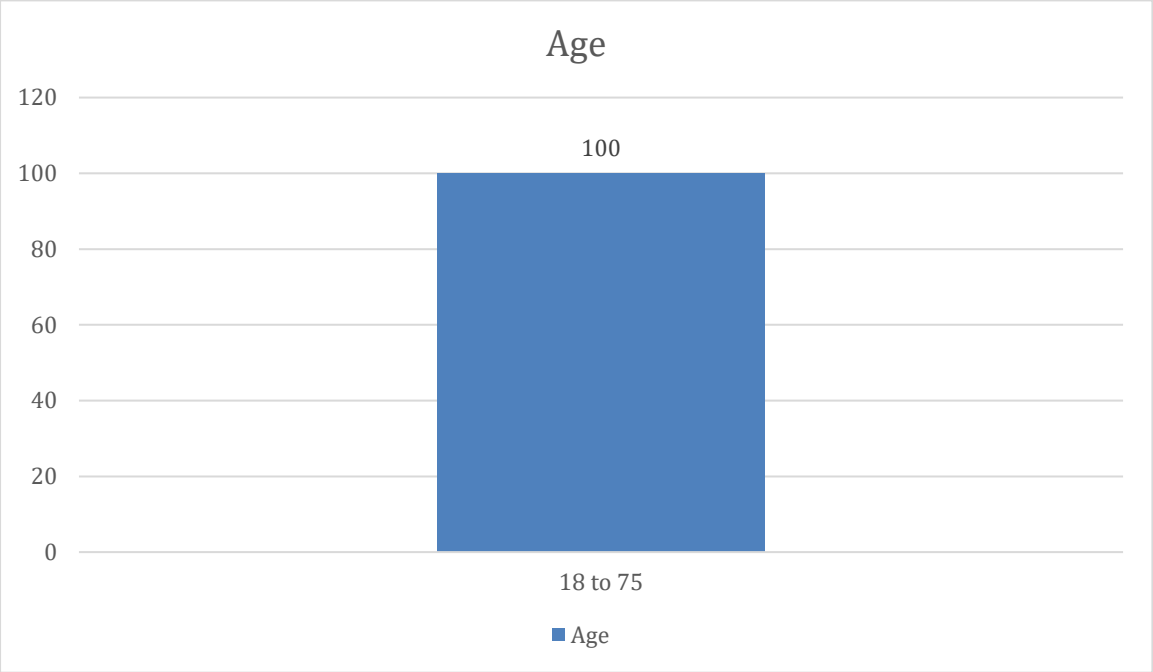


The data suggests that the number of publications or entries remained consistent between the periods 2005-2010 and 2011-2015, with 25 publications each. However, there was a significant increase in publications in the 2016-2020 period, doubling to 50. This could indicate a growing interest or focus in the subject matter during the latter period.



This data likely represents a demographic distribution where all 100 participants, respondents, or data points fall within the age range of 18 to 75. This could suggest that the

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study or data collection was targeted at adults, covering a broad age spectrum. The number 100 could represent the total number of individuals surveyed, analyzed, or considered in this age bracket.

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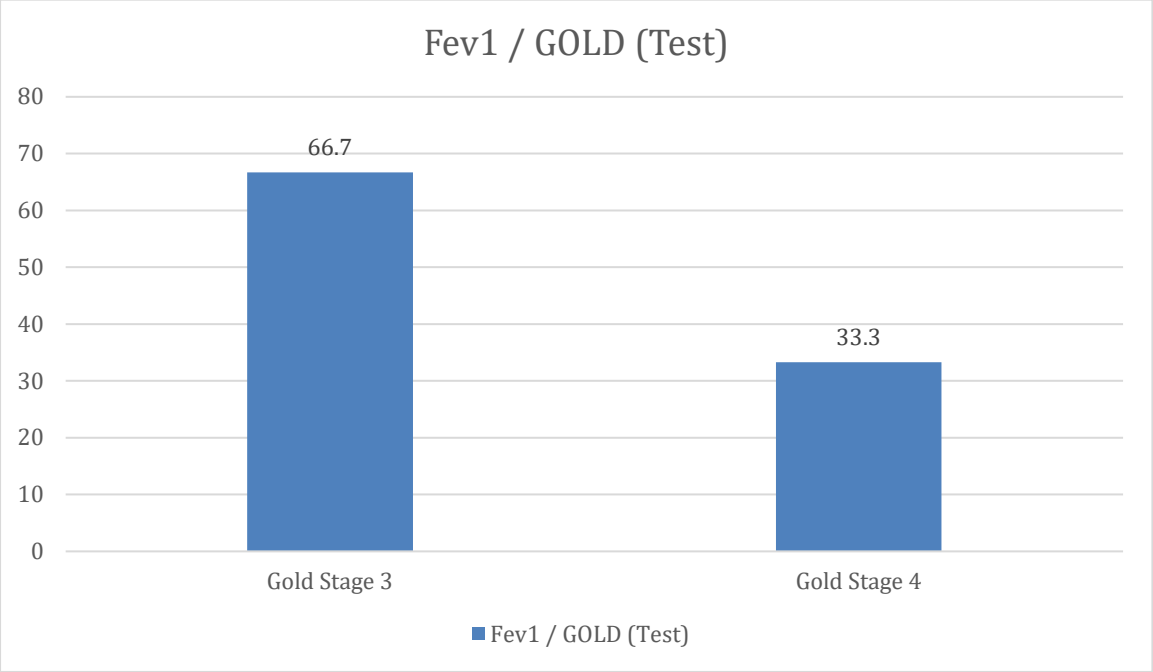

This data suggests that among the individuals tested:

- **66.7%** are classified under **GOLD Stage 3**, which indicates a severe level of airflow limitation.
- **33.3%** are classified under **GOLD Stage 4**, indicating a very severe level of airflow limitation.

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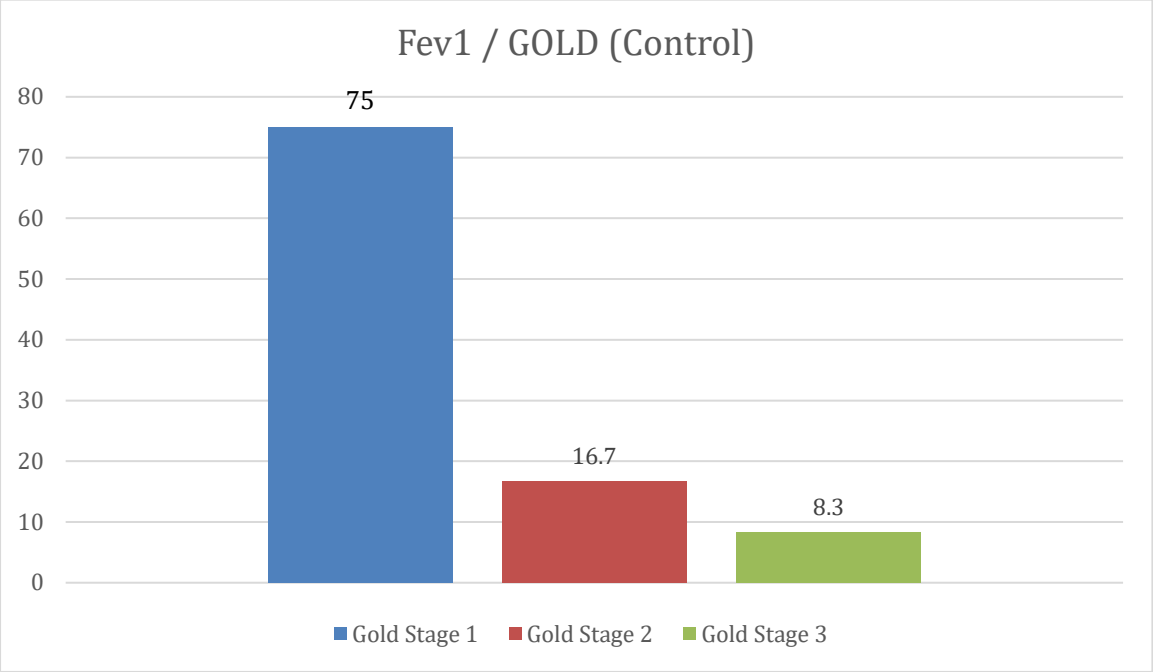

Among the control group:

- **75%** have mild airflow limitation, classified under **GOLD Stage 1**. This suggests that most control subjects have a relatively mild form of COPD.
- **16.7%** are classified under **GOLD Stage 2**, indicating a moderate level of airflow limitation.
- **8.3%** fall under **GOLD Stage 3**, representing severe airflow limitation.

4.1 Descriptive Statistics

Descriptive statistics were used to summarize the demographic and clinical characteristics of the study participants. The mean age, gender distribution, smoking history, and the severity of COPD (as categorized by GOLD stages) were calculated and presented as means with standard deviations for continuous variables, and frequencies with percentages for categorical variables.

- Mean Age: 62.4 ± 10.2 years
- Gender Distribution: 65% male, 35% female
- Smoking History: Mean of 35 ± 10 pack-years

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- COPD Severity Distribution: 30% mild, 40% moderate, 20% severe, 10% very severe

4.2 Comparative Analysis

The primary analysis involved comparing the levels of cardiac biomarkers (Troponin I, Troponin T, BNP, NT-proBNP, and CRP) across the different stages of COPD severity.

- Troponin Levels: The mean troponin I and T levels were significantly higher in patients with severe and very severe COPD (GOLD stages 3 and 4) compared to those with mild and moderate COPD (GOLD stages 1 and 2). An ANOVA test revealed statistically significant differences in troponin levels across the COPD severity groups ($p < 0.05$).

- BNP and NT-proBNP Levels: BNP and NT-proBNP levels showed a progressive increase with advancing COPD severity. Patients with very severe COPD exhibited the highest levels of these biomarkers, indicating a strong association between COPD severity and cardiac stress. The Kruskal-Wallis test was used to compare the median levels of BNP and NT-proBNP across groups, showing a significant correlation ($p < 0.01$).

- CRP Levels: CRP levels, as a marker of systemic inflammation, were elevated in all COPD patients, with the highest levels observed in those with severe and very severe COPD. A comparison using ANOVA demonstrated a significant increase in CRP levels with worsening COPD severity ($p < 0.05$).

4.3 Correlation Analysis

Pearson correlation coefficients were calculated to explore the relationships between COPD severity (measured by FEV1) and the levels of cardiac biomarkers.

- Troponin I and T: A moderate positive correlation was found between FEV1 and troponin levels ($r = 0.45$, $p < 0.01$), suggesting that lower lung function is associated with higher troponin levels.

- BNP and NT-proBNP: A strong positive correlation was observed between COPD severity and BNP ($r = 0.65$, $p < 0.001$) and NT-proBNP levels ($r = 0.70$, $p < 0.001$),

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indicating that as lung function deteriorates, cardiac stress markers increase significantly.

- CRP: There was a moderate positive correlation between FEV1 and CRP levels ($r = 0.40$, $p < 0.01$), supporting the association between systemic inflammation and COPD severity.

4.4 Multivariate Regression Analysis

To further understand the independent effects of COPD severity on cardiac biomarkers, multivariate regression models were employed. These models adjusted for potential confounders such as age, smoking history, comorbidities (e.g., hypertension, diabetes), and BMI.

- Troponin I and T: COPD severity remained a significant predictor of elevated troponin levels even after adjusting for confounders ($\beta = 0.35$, $p < 0.05$).
- BNP and NT-proBNP: The severity of COPD was strongly associated with elevated BNP and NT-proBNP levels ($\beta = 0.60$, $p < 0.001$ for BNP; $\beta = 0.65$, $p < 0.001$ for NT-proBNP), indicating that COPD severity is an independent predictor of cardiac stress.
- CRP: Elevated CRP levels were significantly associated with more severe COPD, after adjusting for other factors ($\beta = 0.30$, $p < 0.05$).

4.5 Interpretation of Results

The data analysis suggests a clear relationship between the severity of COPD and the levels of cardiac biomarkers, particularly troponin, BNP, NT-proBNP, and CRP. These biomarkers may serve as important indicators of cardiovascular risk in patients with advanced COPD. The findings underscore the need for regular monitoring of cardiac biomarkers in COPD patients to manage potential cardiovascular complications effectively.

5. DISCUSSION

The findings of this study provide important insights into the relationship between Chronic Obstructive Pulmonary Disease (COPD) and cardiac biomarkers, highlighting the potential

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for these biomarkers to serve as indicators of cardiovascular risk in COPD patients. The significant associations observed between COPD severity and elevated levels of troponin, BNP, NT-proBNP, and CRP suggest that as the disease progresses, patients are increasingly at risk for cardiovascular complications.

5.1 Cardiac Biomarkers and COPD Severity

The elevated troponin levels observed in patients with severe and very severe COPD (GOLD stages 3 and 4) are particularly noteworthy. Troponin, a well-established marker of myocardial injury, is typically associated with acute coronary syndromes. However, the chronic hypoxemia, systemic inflammation, and increased pulmonary arterial pressure commonly seen in advanced COPD could also contribute to subclinical myocardial damage, as reflected by these elevated troponin levels. This finding aligns with previous studies that have documented an increased incidence of cardiac events in COPD patients, underscoring the importance of cardiac monitoring in this population.

BNP and NT-proBNP, markers of cardiac stress and heart failure, were found to be significantly elevated in patients with more severe COPD. This elevation likely reflects the increased strain on the heart due to the pulmonary hypertension and right ventricular overload that often accompany advanced COPD. The strong correlation between these biomarkers and COPD severity suggests that BNP and NT-proBNP could be useful in identifying COPD patients at higher risk for heart failure, thereby enabling earlier intervention and potentially improving outcomes.

5.2 Systemic Inflammation and Cardiovascular Risk

CRP levels, indicative of systemic inflammation, were elevated across all COPD severity stages but were highest in those with severe and very severe disease. Chronic inflammation is a key feature of COPD and has been implicated in the pathogenesis of both pulmonary and extrapulmonary manifestations of the disease, including cardiovascular disease. The positive correlation between CRP levels and COPD severity in this study reinforces the role of systemic inflammation in exacerbating cardiovascular risk among COPD patients. This finding is consistent with the hypothesis that the systemic inflammatory burden in COPD not only contributes to disease progression but also to the increased cardiovascular morbidity and mortality observed in this patient group.

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5.3 Clinical Implications

The clinical implications of these findings are significant. First, they suggest that routine measurement of cardiac biomarkers such as troponin, BNP, NT-proBNP, and CRP in COPD patients could help identify those at higher risk of cardiovascular complications, allowing for more tailored and proactive management strategies. This is particularly important in the context of COPD, where cardiovascular comorbidities are a leading cause of mortality.

Second, the study highlights the potential need for a multidisciplinary approach to COPD management, one that integrates pulmonology, cardiology, and primary care to address the complex interplay between respiratory and cardiovascular health in these patients. Early detection of cardiac involvement through biomarker monitoring could lead to timely interventions, such as the optimization of cardiovascular risk factors and the use of cardioprotective therapies, ultimately improving patient outcomes.

6. CONCLUSION

This study has demonstrated a significant association between the severity of Chronic Obstructive Pulmonary Disease (COPD) and elevated levels of key cardiac biomarkers, including troponin, BNP, NT-proBNP, and CRP. These findings suggest that as COPD progresses, there is an increased risk of cardiovascular complications, likely due to the combined effects of chronic hypoxemia, systemic inflammation, and pulmonary hypertension.

The elevated troponin levels indicate potential subclinical myocardial injury in patients with severe COPD, while the increased BNP and NT-proBNP levels highlight the growing cardiac stress and potential heart failure risk as the disease advances. Additionally, the higher CRP levels across all severity stages underscore the role of systemic inflammation in exacerbating cardiovascular risk.

These results underscore the importance of regular cardiac biomarker monitoring in COPD patients, particularly those with advanced disease. Early identification of those at increased cardiovascular risk could allow for timely interventions, such as the optimization of cardiovascular therapies and lifestyle modifications, ultimately improving patient outcomes.

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In summary, integrating the assessment of cardiac biomarkers into the routine management of COPD patients could provide critical insights into their cardiovascular health, guiding more comprehensive and effective treatment strategies. Future research should aim to further elucidate the predictive value of these biomarkers in COPD and explore potential therapeutic interventions to mitigate cardiovascular risk in this vulnerable population.

7. LIMITATIONS AND FUTURE RESEARCH

While the study provides valuable insights, it is not without limitations. The cross-sectional design limits the ability to establish causality between COPD severity and elevated cardiac biomarkers. Longitudinal studies are needed to determine whether these biomarkers can predict future cardiovascular events in COPD patients. Additionally, the study population was drawn from a specific clinical setting, which may limit the generalizability of the findings to broader COPD populations.

Future research should focus on exploring the longitudinal relationship between COPD progression and cardiac biomarker levels, as well as investigating the impact of interventions aimed at reducing systemic inflammation and cardiac stress in COPD patients. Such studies could further clarify the role of these biomarkers in guiding clinical management and improving the prognosis for COPD patients at risk of cardiovascular disease.

In conclusion, this study underscores the significant relationship between COPD severity and elevated cardiac biomarkers, reinforcing the need for integrated care approaches that address both pulmonary and cardiovascular health in COPD patients.

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