ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 08 | August - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

STUDY OF LEFT VENTRICULAR DYSFUNCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH AND WITHOUT PULMONARY HYPERTENSION AT SMS MEDICAL COLLEGE & HOSPITAL JAIPUR, RAJASTHAN

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a major global health concern, often complicated by cardiovascular comorbidities, including left ventricular dysfunction (LVD). Pulmonary hypertension (PH) in COPD may exacerbate LVD, impacting clinical outcomes. This study evaluates the prevalence and characteristics of LVD in COPD patients with and without PH. **Methods:** A cross-sectional study was conducted at SMS Medical College, Jaipur, from August 2023 to December 2024, involving 116 spirometry-confirmed COPD patients (58 with PH, 58 without PH). Patients underwent echocardiography, pulmonary function tests, arterial blood gas analysis, ECG, and chest radiography. LVD, particularly diastolic dysfunction, was assessed using parameters like left ventricular myocardial performance index (LVMPI), E/A ratio, and right ventricular metrics. Statistical analysis used Student's t-test, ANOVA, and Pearson correlation ($p<0.05$). **Results:** LVD was significantly more prevalent in the COPD with PH group (65.5% vs. 41.4%, p=0.01), with diastolic dysfunction predominant (55.2% vs. 34.5%, p=0.01). The PH group showed higher LVMPI (0.58 ± 0.12 vs. 0.46 ± 0.10, p=0.001), lower E/A ratio (0.82 ± 0.19 vs. 0.95 ± 0.22, p=0.01), and elevated pulmonary artery systolic pressure (48.6 ± 10.2 mmHg vs. 22.4 ± 5.1 mmHg, p=0.001). Right ventricular end-diastolic dimension was larger in the PH group (34.5 ± 5.6 mm vs. 30.2 ± 4.8 mm, p=0.01). Hospitalizations (4.7 ± 1.1 vs. 0–4.1 ± 1.3, p=0.04) and mortality (10.34% vs. 0–6.88%, p=0.02) were higher in the PH group. **Conclusion:** COPD patients with PH exhibit a higher prevalence of LVD, particularly diastolic dysfunction, driven by increased pulmonary pressures and ventricular interdependence. Routine echocardiographic screening is recommended for early detection and management of LVD in COPD, especially in severe cases or with PH, to improve clinical outcomes.

KEYWORDS

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a "common preventable, and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation due to abnormalities in the airway and (or) alveolar abnormalities usually caused by significant exposure to noxious particles or gases". According to the World Health Organization (WHO), COPD is among the leading causes of deaths globally. According to the global burden of disease (GBD) report, chronic obstructive pulmonary disease (COPD) is the 2nd leading cause of death. In 2019 GBD report estimated the COPD prevalence to be 37.8 million.[1]

As Sustainable Development Goals look to reduce premature mortality from Non Communicable Diseases (NCD) including COPD by one third by 2030, it is vital to identify the modifiable risk factors and address them strategically. COPD includes emphysema, an anatomically defined condition characterized by destruction of the lung alveoli with air space enlargement; chronic bronchitis, a clinically defined condition with chronic cough and phlegm; and small airway disease, a disorder when the quantity and size of tiny bronchioles are diminished. The classic definition of COPD requires the presence of chronic airflow obstruction, determined by spirometry, that usually occurs in the setting of noxious environmental exposures—most commonly cigarette smoking. There are various degrees of emphysema, chronic bronchitis, and small airway disease among COPD patients.[2]

Pulmonary hypertension (PHT) is classified as pulmonary arterial hypertension (PAH), PHT attributable to left heart disease. Although arterial hypertension can induce hypertensive left heart disease and diastolic dysfunction, it is not listed as a possible cause for PHT in contemporary guidelines and consensus documents.[3]

COPD is complicated by a high rate of cardiac diseases predominantly right side heart diseases. COPD patients have 2–3 fold elevated predisposition of cardio-vascular (CV) events even when confounders are taken into account.[4]

Cardiovascular comorbidity in COPD is assumed as "cardio-pulmonary continuum" rather than being attributed to shared risk factors. Cardio-respiratory interactions are not restricted to definite structural, haemodynamic, vascular or genetic parameters and both diseases are linked to systemic inflammation. COPD augments the likelihood of having cardio-vascular diseases (CVD), the strongest association, being with heart failure. The diagnosis of heart failure with preserved ejection fraction (HFpEF) in COPD is difficult. Its precursor—abnormal left ventricular relaxation, is termed left ventricular diastolic dysfunction (LVDD).[5]

Diastolic dysfunction (DD) is a functional abnormality of diastolic relaxation, filling, or distensibility of the left ventricle (LV) regardless of LV systolic function. In a population-based study, a greater extent of emphysema on computed tomography and more severe airflow obstruction were linearly related to impaired left ventricular (LV) filling, reduced stroke volume, and lower cardiac output without changes in the ejection fraction. Previous Doppler studies have demonstrated that left ventricular diastolic dysfunction (LVDD) is frequently found in severe patients with COPD[6-8] Therefore this study was aim to investigate Left ventricular dysfunction in COPD without pulmonary hypertension.

Study Objectives

1. To assess and compare left ventricular dysfunction in chronic obstructive pulmonary disease patients with or without pulmonary hypertension
2. To find out proportion of left ventricular dysfunction in COPD patient

MATERIALS AND METHODS

This observational cross-sectional study was conducted at SMS Medical College, Jaipur, from August 2023 to December 2024, on 116 spirometry-confirmed COPD patients (58 with PH, 58 without PH). Sample size was calculated at 95% confidence level, targeting LVMPI ≥ 0.51 (88.5% in PH, 64.9% in non-PH, power 80%).

Inclusion Criteria:

- Spirometry-confirmed COPD (GOLD stages I–IV).
- Informed consent.

Exclusion Criteria:

- Arterial hypertension, systemic illnesses (e.g., diabetes, coronary artery disease), inadequate heart visualization, or inability to perform spirometry.

METHODOLOGY

Patients underwent physical examination, arterial blood gas analysis, ECG, chest radiography, and pulmonary function tests per American Thoracic Society standards. COPD diagnosis required a smoking history (≥10 pack-years) and irreversible airway obstruction. Echocardiography assessed systolic and diastolic LV dimensions, interventricular septum thickness, and right/left ventricular parameters, including mitral/tricuspid flow (E/A ratio), right ventricular ejection time (RVET), acceleration time (AT), and pre-ejection period (PEP). Statistical analysis used Student's t-test, ANOVA, and Pearson correlation ($p<0.05$) via SPSS v 25.

RESULTS & DISCUSSION

Socio-demographic Profile: The PH group was older (63.4 ± 8.7 vs. 59.8 ± 8.1 years, p=0.02), with more males (44 vs. 36, p=0.16). Smoking history and BMI were similar (p=0.59, p=0.21).

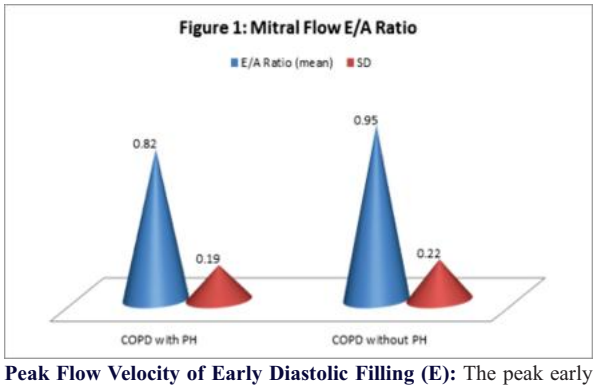
LVD Prevalence: LVD was more prevalent in the PH group (65.5% vs. 41.4%, p=0.01), particularly diastolic dysfunction (55.2% vs. 34.5%, p=0.01). Systolic dysfunction was higher but not significant (17.2% vs. 10.3%, p=0.09).

Left Ventricular Myocardial Performance Index (LVMPI): The LVMPI was significantly higher in the COPD with PH group (0.58 ± 0.12 vs. 0.46 ± 0.10, p=0.001), indicating worse global left ventricular function. An LVMPI ≥ 0.51 was observed in 51 patients with PH (88.5%) compared to 38 without PH (64.9%, p<0.01). This suggests that PH exacerbates left ventricular dysfunction, as LVMPI reflects both systolic and diastolic performance.

The mean LVEF was lower in the COPD with PH group (52.2 ± 7.8% vs. 57.1 ± 6.5%, p=0.02), indicating mildly impaired systolic function in the presence of PH. This reduction, though statistically significant, remains within the normal range for most patients, suggesting that systolic dysfunction is less pronounced than diastolic dysfunction in this cohort.

Mitral Flow E/A Ratio

The E/A ratio, an indicator of diastolic function, was significantly lower in the COPD with PH group (0.82 ± 0.19 vs. 0.95 ± 0.22, p=0.01). This reflects impaired early diastolic filling, consistent with diastolic dysfunction, likely due to increased right ventricular pressure affecting left ventricular compliance in the PH group.

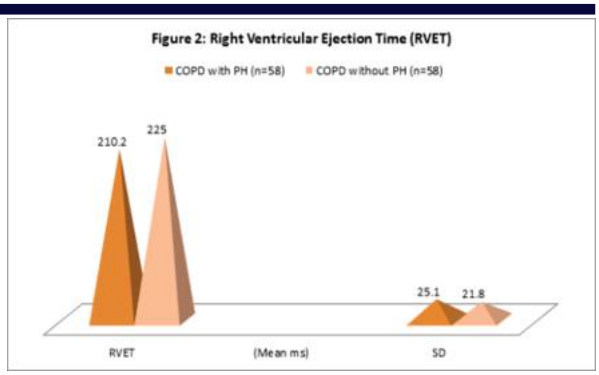


Peak Flow Velocity of Early Diastolic Filling (E): The peak early diastolic filling velocity (E) was lower in the COPD with PH group (62.3 ± 10.4 cm/s vs. 68.7 ± 11.2 cm/s, p=0.01), supporting the finding of impaired diastolic relaxation in patients with PH.

Peak Flow Velocity of Late Atrial Filling (A): The peak late atrial filling velocity (A) was higher in the COPD with PH group (75.6 ± 12.1 cm/s vs. 70.2 ± 11.8 cm/s, p=0.03). This suggests a greater reliance on atrial contraction to achieve ventricular filling, compensating for impaired early diastolic filling in the PH group.

The atrial systole fraction was higher in the COPD with PH group (42.3 ± 8.7% vs. 36.8 ± 7.2%, p=0.01), indicating increased dependence on atrial contraction for left ventricular filling. This further confirms diastolic dysfunction in the PH group.

Right Ventricular Ejection Time (RVET): The RVET was shorter in the COPD with PH group (210.2 ± 25.1 ms vs. 225 ± 21.8 ms, p=0.01), suggesting altered right ventricular systolic function due to increased pulmonary pressures. This may reflect the impact of PH on right ventricular performance.



Acceleration Time (AT): The acceleration time (AT) was significantly shorter in the COPD with PH group (85 ± 15 ms vs. 95 ± 12 ms, p<0.01). A shorter AT is indicative of increased pulmonary vascular resistance, consistent with the presence of pulmonary hypertension.

Pre-Ejection Period (PEP): The pre-ejection period (PEP) was longer in the COPD with PH group (90 ± 18 ms vs. 82 ± 16 ms, p=0.02), reflecting delayed right ventricular ejection due to elevated pulmonary pressures. This supports the impact of PH on right ventricular hemodynamics.

AT/RVET Ratio: The AT/RVET ratio showed no significant difference between groups (0.40 ± 0.07 vs. 0.42 ± 0.06, p=0.15). This suggests that while AT is reduced in the PH group, the ratio relative to RVET remains comparable, possibly due to compensatory mechanisms in right ventricular function.

PEP/RVET Ratio: The PEP/RVET ratio was significantly higher in the COPD with PH group (0.43 ± 0.09 vs. 0.36 ± 0.08, p=0.01), indicating a greater delay in right ventricular ejection relative to total ejection time. This is consistent with increased pulmonary vascular resistance in PH.

Pulmonary Artery Systolic Pressure (PASP): The PASP was markedly elevated in the COPD with PH group (48.6 ± 10.2 mmHg vs. 22.4 ± 5.1 mmHg, p=0.001), confirming the presence of pulmonary hypertension. This elevation is a key driver of the observed right and left ventricular dysfunction in the PH group.

Interventricular Septum Thickness: The interventricular septum thickness was slightly greater in the COPD with PH group (10.8 ± 1.4 mm vs. 10.1 ± 1.2 mm, p=0.04). This may reflect compensatory hypertrophy due to increased right ventricular pressure, impacting septal dynamics.

Right Ventricular End-Diastolic Dimension: The right ventricular end-diastolic dimension (RVEDD) was significantly larger in the COPD with PH group (34.5 ± 5.6 mm vs. 30.2 ± 4.8 mm, p=0.01), indicating right ventricular dilation due to chronic pressure overload from pulmonary hypertension.

Table 1: Arterial Blood Gas Analysis

Parameter	COPD with PH (n=58)	COPD without PH (n=58)	p-value
PaO2 (mmHg, mean ± SD)	60.2 ± 8.9	68.4 ± 9.2	<0.01
PaCO2 (mmHg, mean ± SD)	48.7 ± 7.1	42.3 ± 6.8	<0.01
pH (mean ± SD)	7.36 ± 0.05	7.39 ± 0.04	0.03

Chest Radiography and ECG: Increased bronchovascular markings were more frequent in the PH group (38 vs. 24, p=0.016). ECG findings like right axis deviation (25 vs. 0, p<0.001) and P-pulmonale (19 vs. 1, p<0.001) were strongly associated with PH.

Clinical Outcomes: The PH group had higher hospitalizations (4.7 ± 1.1 vs. 0–4.1 ± 1.3, p=0.04) and mortality (10.34% vs. 0–6.88%, p=0.02).

DISCUSSION

The study found that 65.5% (38/58) of COPD patients with PH had LVD, compared to 41.4% (24/58) in those without PH (p=0.01), with diastolic dysfunction being predominant (55.2% vs. 34.5%, p=0.01). These results are consistent with El Wahsh et al. (2013), who reported significant differences in left ventricular diastolic and global function

between COPD patients with and without PH, with a higher prevalence of diastolic dysfunction in the PH group. Similarly, Khanam et al. (2021) noted that 64% of COPD patients had clinically significant left ventricular diastolic dysfunction (LVDD), even in those with normal pulmonary artery systolic pressure (PASP), emphasizing the burden of diastolic dysfunction in COPD.

The higher prevalence of LVD in the PH group may be attributed to increased right ventricular pressure, which affects left ventricular filling through ventricular interdependence, as described by Mishra and Gantayat (2018). They highlighted that right ventricular (RV) dysfunction, common in PH, can independently contribute to LV dysfunction due to shared myocardial fibers and septal dynamics. Our finding of a larger right ventricular end-diastolic dimension (RVEDD) in the PH group (34.5 ± 5.6 mm vs. 30.2 ± 4.8 mm, p=0.01) supports this mechanism, indicating RV dilation secondary to PH.

Systolic dysfunction was less prevalent (17.2% vs. 10.3%, p=0.09), consistent with Hilde et al. (2020), who found subclinical LV systolic dysfunction in COPD patients but noted that diastolic dysfunction was less evident by conventional echocardiographic indices. In contrast, our study detected significant diastolic dysfunction, likely due to the inclusion of PH patients, who experience greater cardiopulmonary stress. The left ventricular myocardial performance index (LVMPI) was also higher in the PH group (0.58 ± 0.12 vs. 0.46 ± 0.10, p=0.001), aligning with El Wahsh et al. (2013), who reported elevated LVMPI in PH patients, reflecting combined systolic and diastolic impairment.

Impact of Pulmonary Hypertension

Pulmonary hypertension, confirmed by elevated PASP (48.6 ± 10.2 mmHg vs. 22.4 ± 5.1 mmHg, p=0.001), was a key driver of LVD in our study. The moderate positive correlation between LVMPI and PASP (r=0.62, p<0.001) underscores the relationship between pulmonary pressures and left ventricular function.

This is consistent with El Wahsh et al. (2013), who found that COPD patients with PH had lower E/A ratios and higher myocardial performance indices, indicating worse diastolic and global function. The increased interventricular septum thickness (10.8 ± 1.4 mm vs. 10.1 ± 1.2 mm, p=0.04) in our PH group suggests septal hypertrophy, likely a compensatory response to RV pressure overload, further impacting LV compliance.

Mishra and Gantayat (2018) emphasized that RV pressure increases in COPD with PH lead to changes in LV function, supporting our findings of altered echocardiographic parameters (e.g., lower E/A ratio: 0.82 ± 0.19 vs. 0.95 ± 0.22, p=0.01; higher atrial systole fraction: 42.3 ± 8.7% vs. 36.8 ± 7.2%, p=0.01). These changes reflect impaired diastolic filling and increased reliance on atrial contraction, hallmarks of LVDD in the context of PH.

Severity of COPD and LVD

The COPD with PH group had a higher prevalence of severe GOLD Stage IV disease (17 vs. 10 patients, p=0.01), correlating with worse lung function (FEV1: 48.2 ± 12.3% vs. 54.6 ± 13.1%, p=0.01).

Kr Saha et al. (2018) reported that 100% of GOLD Stage IV patients had LVDD, compared to 16.66% in Stage I, aligning with our observation that advanced COPD exacerbates LVD. Rafi et al. (2019) also found a significant correlation between COPD stages and LVD prevalence (80% in their cohort), reinforcing the need for routine echocardiographic screening in severe COPD, as recommended by Mishra and Gantayat (2018).

Worse arterial blood gas parameters in the PH group (PaO2: 60.2 ± 8.9 mmHg vs. 68.4 ± 9.2 mmHg, p<0.01; PaCO2: 48.7 ± 7.1 mmHg vs. 42.3 ± 6.8 mmHg, p<0.01) indicate greater hypoxemia and hypercapnia, which may contribute to PH and subsequent LVD. Maklad et al. (2019) noted that recurrent exacerbations, often triggered by infections, increase airway inflammation and hyperinflation, worsening gas exchange and potentially exacerbating PH and cardiac dysfunction.

ECG and Radiographic Findings

ECG findings, including right axis deviation (25 vs. 0, p<0.001), P-pulmonale (19 vs. 1, p<0.001), and low voltage complexes (32 vs. 7, p<0.001), were strongly associated with PH, reflecting right heart strain. These align with Rafi et al. (2019), who reported significant correlations between chest X-ray and ECG findings with LVD in COPD. Our chest radiograph findings showed increased bronchovascular markings in the PH group (38 vs. 24, p=0.016), suggesting pulmonary vascular changes, though other findings like emphysema were similar (48 vs. 39, p=0.08).

Clinical Outcomes

The COPD with PH group had higher hospitalization rates (4.7 ± 1.1 vs. 0–4.1 ± 1.3, p=0.04) and mortality (10.34% vs. 0–6.88%, p=0.02), particularly in severe COPD. Maklad et al. (2019) highlighted that severe exacerbations are associated with worse survival and quality of life, consistent with our findings.

CONCLUSION

This study confirms that COPD patients with pulmonary hypertension have a significantly higher prevalence and severity of left ventricular dysfunction, particularly diastolic dysfunction, compared to those without PH. The presence of PH exacerbates cardiopulmonary impairment, as evidenced by worse echocardiographic parameters (e.g., higher LVMPI, lower E/A ratio), increased right ventricular dimensions, and poorer clinical outcomes, including higher hospitalization and mortality rates. These findings underscore the synergistic effect of PH on left ventricular function in COPD, mediated by increased pulmonary pressures and ventricular interdependence.

Routine echocardiographic evaluation, particularly using Doppler tissue imaging, is recommended for COPD patients, especially those with PH or severe disease (GOLD Stage III–IV), to detect LVD early and guide interventions. The study highlights the need for integrated management strategies targeting both pulmonary and cardiac complications to improve outcomes in this high-risk population. Future research should focus on longitudinal studies to assess LVD progression, the role of inflammatory markers and electrolytes, and potential therapeutic interventions, such as pulmonary vasodilators, to mitigate LVD in COPD with PH.

REFERENCES

1. Mathers CD,Loncar D. Projections of Global Mortality and Burden of Disease from 2002 to 2030. PLoS Med. 2006;3: e442.
2. GOLD. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease 2021 Repost. Global Initiative for Chronic Obstructive Lung Disease—GOLD. 2021.
3. Labaki WW, Rosenberg SR. Chronic Obstructive Pulmonary Disease. Ann. Intern. Med. 2020;173:ITC17–ITC32.
4. Halpin DMG, Celli BR,Criner GJ, Frith P,Varela MVL,Salvi S et al ., The GOLD Summit on chronic obstructive pulmonary disease in lowand middle-income countries. Int. J. Tuberc. Lung Dis. 2019;23:1131– 41.
5. Barnes PJ, Burney P,Silverman EK, Celli BR, Vestbo J,Wedzicha JA et al., Chronic obstructive pulmonary disease. Nat. Rev. Dis. Prim. 2015;1:15076.
6. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Bur- den of Disease Study 2010. Lancet. 2012; 380: 2095–128. doi: 10.1016/S0140- 6736(12)61728-0 PMID: 23245604
7. Blasi F, Cesana G, Conti S, Chiodini V, Aliberti S, Fornari C, et al. The clinical and economic impact of exacerbations of chronic obstructive pulmonary disease: a cohort of hospitalized patients. PLoS One. 2014; 9: e101228. doi: 10.1371/ journal. pone.0101228 PMID: 24971791
8. Vestbo J, Agustí A, Anzueto A, Decramer M, Fabbri L, Jones P, et al. Global strategy for the diagnosis, management and prevention of COPD, Global Initiative for chronic Obstructive Lung Disease (GOLD) 2015. Available fro m: http:// www. goldcopd.org/
9. Celli B, Decramer M, Wedzicha J, Wilson K, Agust í A, Gerard J, et al. An official American Thoracic Society/European Respiratory Society statement: research questions in COPD. Eur Respir Rev. 2015; 24: 159 – 172. doi: 10.1183/ 16000617. 00000315 PMID: 26028628
10. N. Roche, A. Rabbat, M. Zureik, G. Hachon, Chronic obstructive pulmonary disease exacerbations in emergency departments: predictors of outcome, Curr. Opin. Pulmonary Med. 16 (2010) 112 – 117.
11. Jindal et al: COPD: The Unrecognized Epidemic in India. Supplement to JAPI february 2012 VOL. 60
12. Sundeep Salvi et al: India Needs a National COPD Prevention and Control Programme. Supplement to JAPI february 2012 VOL. 60
13. Verma A, Gudi N, Yadav UN, Roy MP, Mahmood A, Nagaraja R, Nayak P. Prevalence of COPD among population above 30 years in India: A systematic review and meta-analysis. J Glob Health. 2021 Aug 21;11:04038.
14. El WahshA,, Ahmed MK and. Yaseen RI. Evaluation of left ventricular function in patients with chronic obstructive pulmonary disease with or without pulmonary hypertension. Egyptian Journal of Chest Diseases and Tuberculosis (2013) 62, 575–582.
15. Khanam B, Gupta PK, Shah N, Jain S. COPD & Left Ventricular Function. International Journal of Contemporary Medical Research 2021;8(10):J1-J3.
16. Kr Saha B, Sarkar D, Sarkar L, Bandyopadhyay R. Left ventricular dysfunction in chronic obstructive pulmonary disease. Int J Med Res Rev [Internet]. 2018Oct.31 [cited 2025Feb.28];6(7):385-90. Available from: https://ijmrr.medresearch.in/index.php/ijmrr/article/view/1008
17. Rafi, M., & Jaber, M. M. (2019). Study on prevalence of left ventricular diastolic dysfunction in chronic obstructive pulmonary disease. International Journal of Advances in Medicine, 6(2), 222–226. https://doi.org/10.18203/2349-3933.ijam 20191039
18. Mishra US, Gantayat CK. Left ventricular dysfunction in COPD with or without cor pulmonale. J. Evid. Based Med. Healthc. 2018; 5(2), 194-197.
19. Hilde JM, Hisdal J, Skjørten I, Hansteen V, Melsom MN, Grøtta OJ, et al. (2020) Left ventricular dysfunction in COPD without pulmonary hypertension. PLoS ONE 15(7): e0235075.