ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 08 | August - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

ECHOCARDIOGRAPHIC CHANGES IN PATIENTS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND ITS CORRELATION WITH SEVERITY OF DISEASE AND HIGH SENSITIVITY SERUM CRP VALUES

Respiratory Medicine

Dr. Mahendra Kumar Bainara — Senior Professor, Department Of Respiratory Medicine, RNT Medical College, Udaipur.

Dr. Prashant Srivastava* — Junior Resident, Department Of Respiratory Medicine, RNT Medical College, Udaipur. *Corresponding Author

Dr. Neha Damor — Assistant Professor, Department Of Respiratory Medicine, RNT Medical College, Udaipur.

Dr. Mahesh Mahich — Assistant Professor, Department Of Respiratory Medicine, RNT Medical College, Udaipur.

ABSTRACT

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a major global health issue, characterized by chronic inflammation and airflow limitation. Systemic inflammation in COPD patients is commonly assessed through biomarkers like high-sensitivity C-reactive protein (hs-CRP). **Objectives:** To evaluate the 2D Echo changes and correlation with severity of disease (FEV1) & hs-CRP in COPD. **Methodology:** A cross-sectional study was conducted involving 106 COPD patients.After obtaining written informed consent, demographic details, smoking habits, environmental exposure to biomass fuels, and COPD severity were recorded. hs-CRP levels were measured as a marker of systemic inflammation. Echocardiographic evaluations were performed to assess cardiac complications. Descriptive statistics were used to analyze demographic and clinical data. **Results:** Echocardiographic abnormalities were common, with PH present in 50% of patients, increasing with disease severity ($p < 0.001$). Right ventricular dilation (RVd) was seen in 41.5% of cases, while left ventricular systolic dysfunction (LVSD) and diastolic dysfunction (LVDD) were noted in 8.49% and 13.21% of patients, respectively. A significant association was found between COPD severity and PH, LVSD, LVDD, and RVd ($p < 0.001$). hs-CRP levels were significantly higher in patients with PH (76.61 ±74.18 mg/L), LVSD (97.68 ±68.35 mg/L), LVDD (94.80 ±57.49 mg/L), and RVd (79.02 ±73.69 mg/L), indicating a strong link between systemic inflammation and cardiac complications in COPD ($p < 0.001$). **Conclusions:** Echocardiographic abnormalities are significantly associated with COPD severity and systemic inflammation. hs-CRP serves as a valuable biomarker for identifying high-risk patients. Integrating echocardiographic evaluation and inflammatory markers in COPD management can aid in early diagnosis and targeted interventions for better patient outcomes.

KEYWORDS

COPD, hs-CRP, 2D-Echo, FEV1, PH, RVd, LVSD, LVDD

INTRODUCTION

Breathing issues and persistent airflow restriction are hallmarks of chronic obstructive pulmonary disease (COPD). [1] Smoking is a leading cause, triggering lung inflammation that persists even after smoking cessation. Additionally, exposure to indoor biomass smoke—such as wood and agricultural residues—is a significant contributor, particularly in low-income settings. COPD's prevalence increases with age and is linked to reduced lung function.[2] Biomass smoke exposure contributes approximately 2.7% of global disability-adjusted life years (DALYs).[3]

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024[4] highlights the increased cardiovascular risk among COPD patients due to shared mechanisms like systemic inflammation and endothelial dysfunction.[5] Globally, the World Health Organization estimates that 65 million people suffer from COPD, with low-income countries like India bearing a disproportionate burden[6,7] India alone accounted for over 55 million cases in 2016, with the disease emerging as a leading cause of death among non-communicable diseases.[8] COPD prevalence varies significantly, ranging from 0.1% among younger populations to over 28% in older adults, and differs across regions, as seen in Delhi (10%) and Kerala (6.19%).[9]

Beyond pulmonary effects, COPD profoundly impacts cardiovascular health. Systemic inflammation, oxidative stress, and hypoxia lead to pulmonary hypertension, right ventricular remodeling, and eventual heart failure.[10] Acute exacerbations frequently result in hospitalizations and adverse cardiac outcomes.[11]Echocardiography, a non-invasive diagnostic tool, plays a vital role in assessing pulmonary pressures and cardiac function, offering insights into the cardiovascular burden of COPD.

Even mild elevations in pulmonary arterial pressure can cause RV hypertrophy, dilation, and dysfunction.[12] To assess these changes, biomarkers like brain natriuretic peptide (BNP) and troponin, as well as inflammatory markers like C-reactive protein (CRP), are frequently used. Elevated CRP levels indicate systemic inflammation and correlate with hospitalization rates and mortality in COPD patients.[13] High-sensitivity CRP (hs-CRP) has emerged as a reliable biomarker for evaluating airway obstruction, systemic inflammation, and disease severity.[14]

Echocardiographic (2D Echo) changes in COPD, such as right ventricular dysfunction and pulmonary hypertension, correlate with high-sensitivity C-reactive protein (hs-CRP) levels, reflecting systemic inflammation and endothelial dysfunction. Elevated hs-CRP is associated with worsening cardiac remodeling, emphasizing its prognostic role in COPD severity and cardiovascular complications.[15] This complexity necessitates an integrated approach to manage pulmonary-cardiovascular interplay in COPD.

MATERIALAND METHODS

Study Design And Sample Size

It was a hospital based cross sectional observational study. Study was conducted after obtaining institutional ethical committee approval on 106 patients visiting in the Department of Respiratory medicine, TB and Chest Hospital, Badi, R.N.T medical college, Udaipur.

Study Method

Patients presenting to Outpatient Department & Inpatient Department meeting inclusion and exclusion criteria. Inclusion criteria for the study included patients aged 40 years and above with a confirmed diagnosis of COPD, indicated by a post-bronchodilator Forced Expiratory Volume1 (FEV1)/Forced Vital Capacity (FVC) ratio of <0.7 in the stable stage, and those who provided written informed consent. Exclusion criteria comprised patients who did not provide consent, were unable to perform spirometry, had chronic lung diseases other than COPD, pre-existing cardiac disease, systemic hypertension, diabetes mellitus, congenital or acquired cardiac diseases leading to pulmonary hypertension (PH), systemic diseases causing PH, or were on medications known to cause PH.

Patients meeting the inclusion criteria underwent a detailed history and physical examination, followed by spirometry as per ATS/ERS guidelines, both pre- and post-bronchodilation. The COPD Assessment Test (CAT) was conducted, and severity was classified

using GOLD guidelines based on spirometry (post-bronchodilator FEV1/FVC < 70%) into mild (FEV1 ≥ 80%), moderate (50% ≤ FEV1 < 80%), severe (30% ≤ FEV1 < 50%), and very severe (FEV1 < 30%).[4] A 12-lead ECG and two-dimensional transthoracic Doppler echocardiograph were performed at R.N.T. Medical College by expert cardiologists.

Echocardiography assessed the pericardium, valvular anatomy, cardiac chambers, and function. Doppler ECHO quantified tricuspid regurgitant jet velocity, and systolic pulmonary arterial pressure (sPAP) was calculated using the modified Bernoulli equation: $sPAP = 4V^2_{max} + RAP$ with RAP estimated based on inferior vena cava (IVC) collapse. Pulmonary hypertension (PH) was defined as mmHg and classified as mild (30-45 mmHg), moderate (46-70 mmHg), or severe (>70 mmHg) using the Chemla formula.

Right ventricular (RV) dimensions were measured by M-mode echocardiography, with dilation defined as >2.6 cm. RV systolic dysfunction was diagnosed when hypokinetic function was observed. Left ventricular (LV) function was assessed using the E/A ratio and ejection fraction (EF). LV diastolic dysfunction (LVDD) was diagnosed when the E/A ratio was below the following age-specific thresholds: <1.3 (45–49 years), <1.2 (50–59 years), <1.0 (60–69 years), and <0.8 (≥70 years).[17]

Serum high-sensitivity C-reactive protein (hs-CRP) was measured, with values categorized as low risk (<1.0 mg/L), average risk (1.0–3.0 mg/L), high risk (>3.0 mg/L), or acute inflammation (>10 mg/L). All data, including demographic details, clinical findings, spirometry, echocardiography, ECG, and hs-CRP levels, were recorded systematically for analysis[18] Data were entered in Microsoft Excel and analyzed using SPSS version 16. Appropriate statistical tests, including chi-square and t-tests, were applied to interpret the data. Results were summarized in tables and figures, and a p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Distribution of study populations based on various variables-1

Variables		Number (N=106)	%
Age Group (years)	40-50	7	6.6
	51-60	27	25.5
	61-70	40	37.7
	>70	32	30.2
Gender	Male	92	86.8
	Female	14	13.2
Domicile	Urban	9	8.5
	Rural	97	91.5
Smoking Status	Non-smoker	15	14.2
	Ex-smoker	66	62.3
	Smoker	25	23.6
Biomass Fuel Exposure	Absent	92	86.8
	Present	14	13.2
Smoking Index	Non-smoker (0)	15	14.2
	Mild smoker (1-100)	6	5.7
	Moderate smoker (101-300)	39	36.8
	Heavy smoker (>301)	46	43.4
COPD Severity	Mild (FEV1>80%predicted)	19	17.9
	Moderate (FEV150-79%predicted)	43	40.6
	Severe (FEV1 30-49%predicted)	28	26.4
	Very severe (FEV1<30%predicted)	16	15.1
GOLD Group	A	19	17.9
	B	49	46.2
	E	38	35.9
HS-CRP	Low risk (<1.0mg/dl)	14	13.2
	Average risk (1.0-3.0mg/dl)	23	21.7
	High risk (3-10mg/dl)	17	16
	Acute inflammation (>10.0mg/dl)	52	49.1

The study included 106 COPD patients, predominantly male (86.8%), with a rural domicile (91.5%). The majority (67.9%) were aged 61 years or older, with 37.7% in the 61–70 age group and 30.2% above 70 years. Smoking exposure was significant, with 62.3% being ex-smokers and 23.6% active smokers, while 43.4% were heavy smokers (smoking index >301). Biomass fuel exposure was noted in 13.2% of participants. COPD severity was classified as mild in 17.9%, moderate in 40.6%, severe in 26.4%, and very severe in 15.1%, with GOLD group B (46.2%) being the most common. High-sensitivity C-reactive protein (hs-CRP) levels indicated acute inflammation (>10 mg/dL) in 49.1%, high risk (3–10 mg/dL) in 16%, average risk (1.0–3.0 mg/dL) in 21.7%, and low risk (<1.0 mg/dL) in 13.2%.(Table 1)

The mean hs-CRP levels significantly increased with the severity of COPD ($p < 0.001$). Patients with mild COPD had the lowest mean hs-CRP level (2.28 ± 5.09), followed by moderate COPD (13.83 ± 25.67), severe COPD (68.18 ± 55.80), and very severe COPD, which showed the highest levels (124.35 ± 93.18). This indicates a strong correlation between COPD severity and systemic inflammation.

The study found a significant association ($p = 0.001$) between smoking intensity and HS-CRP levels. Non-smokers had an HS-CRP mean of 20.48 ± 53.96 mg/L, while mild smokers had the lowest levels at 2.73 ± 2.31 mg/L. Moderate smokers showed increased levels at 36.94 ± 71.02 mg/L, and heavy smokers had the highest HS-CRP levels at 60.28 ± 61.32 mg/L. This demonstrates a progressive rise in systemic inflammation with higher smoking intensity.

Table 2: Correlation of COPD severity with echocardiographic findings (N=106)

ECHOCARDIOGRAPHIC FINDINGS	COPD SEVERITY				p value
	Mild	Moderate	Severe	Very Severe	
	N (%)	N (%)	N (%)	N (%)	
PH	0 (0)	16 (30.2)	24(45.3)	13(24.5)	<0.001
TR	19(17.9)	43 (40.6)	28(26.4)	16(15.1)	NA
LVSD	0 (0)	0 (0)	3 (33.3)	6 (66.7)	<0.001
LVDD	0 (0)	0 (0)	7 (50)	7 (50)	<0.001
RVd increased	0 (0)	12 (27.3)	22 (50)	10(22.7)	<0.001
TAPSE abnormality	0 (0)	0 (0)	0 (0)	0 (0)	NA
IVC abnormality	0 (0)	0 (0)	0 (0)	0 (0)	NA
LA/LV DILATED	0 (0)	1 (12.5)	2 (25)	5(62.5)	0.001
RA/RV DILATED	0 (0)	5 (13.5)	22(59.5)	10 (27)	<0.001

The echocardiographic findings showed no significant gender differences in abnormalities. Pulmonary hypertension (PH) was present in 50% of cases, predominantly in males (90.6%, $p = 0.251$). Tricuspid regurgitation (TR) was observed in all cases (100%), with 86.8% in males. Left ventricular systolic dysfunction (LVSD) was noted in 8.49% (77.8% in males, $p = 0.404$), and left ventricular diastolic dysfunction (LVDD) in 13.21% (85.7% in males, $p = 0.898$). Right ventricular dilation (RVd) occurred in 41.51% (90.9% in males, $p = 0.292$). No abnormalities were detected in TAPSE, IVC, or LA/LV dilation, except for 8 cases of LA/LV dilation (all in males, $p = 0.251$).

Among participants, Pulmonary hypertension (PH) increased with disease severity ($p < 0.001$), being most common in severe and very severe cases. Left ventricular systolic dysfunction (LVSD) and diastolic dysfunction (LVDD) were present only in severe and very severe cases ($p < 0.001$). Right ventricular dilation (RVd) and RA/RV dilation also showed a rising trend with increasing severity ($p < 0.001$). LA/LV dilation was rare but more frequent in very severe cases ($p = 0.001$). No abnormalities were observed in TAPSE or IVC across all severity levels. (Table 2)

The data shows a significant association ($p < 0.001$) between GOLD severity and the degree of pulmonary hypertension (PH). Mild PH was predominantly observed in moderate COPD cases (71.4%), while severe PH was most common in severe (41.2%) and very severe (58.8%) cases. Moderate PH was primarily present in severe cases (86.7%). Overall, PH increased in prevalence and severity with worsening COPD severity.

HS-CRP levels were significantly higher in patients with PH, TR, LVSD, LVDD, RVd, and RA/RV dilation & LA/LV dilation also showed a significant association statistically. (Table 3) Echocardiographic findings reveal significant differences in CAT scores among patients with cor pulmonale (mean 24.70 ± 8.18, $p < 0.001$), pulmonary hypertension (mean 23.83 ± 7.92, $p < 0.001$), left ventricular systolic dysfunction (mean 30.33 ± 9.22, $p = 0.001$), left

ventricular diastolic dysfunction (mean 27.21 ± 8.78, $p < 0.001$), and right atrial/ventricular dilation (mean 25.05 ± 7.79, $p < 0.001$).

Table 3: Correlation of HS-CRP with echocardiographic findings. (N=106)

ECHOCARDIOGRAPHIC FINDINGS	HS-CRP (mg/L)		p value
	Mean	SD	
PH	76.61	74.18	<0.001
TR	42.80	64.30	<0.001
LVSD	97.68	68.35	0.002
LVDD	94.80	57.49	<0.001
RVd increased	79.02	73.69	<0.001
TAPSE abnormality	0	0	NA
IVC Collapsibility abnormality	0	0	NA
LA/LV DILATED	93.99	67.54	0.013
RA/RV DILATED	86.15	57.23	<0.001

DISCUSSION

The study's findings on demographics, smoking habits, BMI, and environmental exposures among COPD patients align with existing literature, reinforcing the multifactorial nature of COPD risk. The mean age of 65.24 years and male predominance (86.8%) mirror reports by Twinamasiko et al. (2023)[18], Hochhausen et al. (2024)[19], and Thalla et al. (2020) [20] , emphasizing COPD's chronic nature in older males. Smoking remains a major risk factor, with 62.3% ex-smokers and 23.6% current smokers, echoing similar studies. Additionally, 13.2% biomass fuel exposure highlights environmental contributions to COPD, consistent with Twinamasiko et al.[18] These findings underscore the need for targeted smoking cessation and environmental interventions.

In this study, COPD severity distribution showed 40.6% moderate, 26.4% severe, 17.9% mild, and 15.1% very severe cases, aligning with studies by Cheng et al. (2023)[21] and Flynn et al. (2018)[22] linking advanced COPD stages to increased mortality and hospitalizations. Additionally, 49.1% of participants had hsCRP levels >10 mg/dL, indicating acute inflammation, while 21.7% had average risk, 16% high risk, and 13.2% low risk levels. These findings align with Moayyedkazemi et al. (2018)[23]and Zheng et al. (2022)[24], highlighting the correlation of elevated hsCRP with advanced disease, inflammation, and poor outcomes, reinforcing its role as a predictive marker in COPD.

This study found the mean age of COPD patients with pulmonary hypertension (PH) to be 65 years, with males predominantly affected (90.6% PH, 86.8% tricuspid regurgitation), though gender differences were not statistically significant ($p = 0.251$). Cardiac complications, including PH, left ventricular systolic dysfunction (LVSD), diastolic dysfunction (LVDD), and right ventricular dilation (RVd), were strongly associated with COPD severity ($p < 0.001$). PH was present in 45.3% of moderate and 24.5% of severe cases. These findings align with studies by Kabir et al. (2019)[25] and Shadani et al. (2023)[26], emphasizing the need for routine cardiovascular assessments in moderate-to-severe COPD patients.

This study found significantly elevated HS CRP levels in participants with cardiac complications, including PH (76.61 mg/dL), LVSD (97.68 mg/dL), LVDD (94.80 mg/dL), and RV dysfunction (79.02 mg/dL), with all associations being statistically significant ($p < 0.001$). These findings align with studies by Jatav et al. (2017)[16] and Chaudhari et al. (2018)[27], highlighting the link between systemic inflammation and cardiac dysfunction in COPD. This study found significantly elevated CAT scores in COPD patients with cor pulmonale (24.70), PH (23.83), LVSD (30.33), LVDD (27.21), and RA/RV dilation (25.05) ($p < 0.001$). These findings align with studies by Selem et al. (2017) [28] and Zhang et al. (2018),[29] showing worse symptom burden in patients with these cardiac abnormalities. The results emphasize the importance of comprehensive cardiac evaluation to manage cardiovascular complications in advanced COPD.

CONCLUSION

This study highlights the significant correlation between COPD severity, systemic inflammation (hs-CRP levels), and echocardiographic abnormalities. Higher hs-CRP levels were associated with worsening COPD severity, reflecting increased systemic inflammation and cardiovascular risk. Pulmonary hypertension (PH), right ventricular dysfunction, and left ventricular dysfunction were significantly more prevalent in severe and very severe COPD cases. Smoking intensity showed a strong association with both COPD severity and systemic inflammation, emphasizing the need for aggressive smoking cessation strategies. Given the high prevalence of cardiac complications in advanced COPD, routine echocardiographic screening and hs-CRP measurement can aid in early detection and management of cardiovascular risks. Integrating systemic inflammation markers and cardiac assessments into COPD management may improve patient outcomes and reduce morbidity.

REFERENCES

1. Decramer M, Janssens W, Miravitlles M. Chronic obstructive pulmonary disease. Lancet. 2012 Mar 31;379(9823):1341-51.
2. Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, Calverley P, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2007 Sep 15;176(6):532-55.
3. Barnes PJ. Chronic obstructive pulmonary disease. N Engl J Med. 2000 Jul 27;343(4):269-80.
4. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for Prevention, Diagnosis and Management of COPD: 2024 Report. Bethesda: GOLD; 2024 [cited 2024 Aug 10]. Available from: https://goldcopd.org/2024-gold-report.
5. Miller MR, Quanjer PH, Swanney MP, Ruppel G, Enright PL. Interpreting lung function data using 80% predicted and fixed thresholds misclassifies more than 20% of patients. Chest. 2011 Mar;139(3):734-40.
6. Perez-Padilla R, Montes de Oca M, Thirion-Romero I, Wehrmeister FC, Lopez MV, Valdivia G, et al. Trajectories of Spirometric Patterns, Obstructive and PRISm, in a Population-Based Cohort in Latin America. Int J Chron Obstruct Pulmon Dis. 2023;18:1277-85.
7. Wan ES, Balte P, Schwartz JE, Bhatt SP, Cassano PA, Couper D, et al. Association Between Preserved Ratio Impaired Spirometry and Clinical Outcomes in US Adults. JAMA. 2022 Jan 18;327(3):286-7.
8. India State-Level Disease Burden Initiative CRD Collaborators. The burden of chronic respiratory diseases and their heterogeneity across the states of India: the Global Burden of Disease Study 1990–2016. Lancet Glob Health. 2018 Dec;6(12)
9. Campo G, Pavasini R, Malagù M, Mascetti S, Biscaglia S, Ceconi C, et al. Chronic obstructive pulmonary disease and ischemic heart disease comorbidity: Overview of mechanisms and clinical management. Cardiovasc Drugs Ther. 2015 Apr;29(2):147-57.
10. Mahmood T, Singh RK, Kant S, Das Shukla A, Chandra A, Srivastava RK. Prevalence and etiological profile of chronic obstructive pulmonary disease in nonsmokers. Lung India. 2017 Jan-Feb;34(1):122-6.
11. Panigrahi A, Padhi BK. Chronic bronchitis and airflow obstruction is associated with household cooking fuel use among never-smoking women: a community-based cross-sectional study in Odisha, India. BMC Public Health. 2018 Aug 9;18:924.
12. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. Circulation. 1984 Sep;70(4):657-62.
13. Thompson D, Pepys MB, Wood SP. The physiological structure of human C-reactive protein and its complex with phospho choline. Structure. 1999 Feb 15;7(2):169-77.
14. Buhr RG, Barjaktarevic IZ, Quibrera PM, Bateman LA, Bleecker ER, Couper DJ. Reversible Airflow Obstruction Predicts Future Chronic Obstructive Pulmonary Disease Development in the SPIROMICS Cohort An Observational Cohort Study. Am J Respir Crit Care Med. 2022 Sep 1;206(5):554-62.
15. Sin DD, Man SP. Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular diseases? The potential role of systemic inflammation in chronic obstructive pulmonary disease. Circulation. 2003 Mar 25;107(11):1514-9
16. Jatav VS, Meena SR, Jelia S, Jain P, Ajmera D, Agarwal V, et al. Echocardiographic findings in chronic obstructive pulmonary disease and correlation of right ventricular dysfunction with disease severity. Int J Adv Med. 2017 Aug;4(4):476-80.
17. Kamath DY, Xavier D, Sigamani A, Pais P. High sensitivity C-reactive protein (hsCRP) & cardiovascular disease: An Indian perspective. Indian Journal of Medical Research. 2015 Sep 1;142(3):261-8
18. Twinamasiko W, Byakika S, Ssenyonga R, Kigonya A, Tumusiime D. The impact of smoking and BMI on the severity of COPD in rural populations: A cross-sectional study. Int J Chron Obstruct Pulmon Dis. 2023 Mar 21;18:327-35.
19. Hochhausen N, Muller S, Gupta N, Singh R, Choudhury R, Das S, et al. Demographic factors influencing the prevalence and progression of COPD: Insights from a global study. Respir Med. 2024 Mar;201:105312.
20. Thalla M, Reddy KP, Sharma R, Kumar P. Smoking and chronic obstructive pulmonary disease: A population-based study in South India. Indian J Public Health. 2020 Apr-Jun;64(2):128-34.
21. Cheng W, Zhou A, Zeng Y, Yuan D, Chen R, Chen Y, et al. Prediction of hospitalization and mortality in patients with chronic obstructive pulmonary disease with the new Global Initiative for Chronic Obstructive Lung Disease 2023 group classification: A prospective cohort and a retrospective analysis. Int J Chron Obstruct Pulmon Dis. 2023 Jul 31;18:2341-52.
22. Flynn RW, MacDonald TM, Chalmers JD, Schembri S, Winter JH, Lee KK, et al. The effect of changes to GOLD severity stage on long term morbidity and mortality in COPD. Respir Res. 2018 Nov 20;19(1):249.
23. Moayyedkazemi A, Rahimirad MH. Evaluating serum C-reactive protein level in patients with chronic obstructive pulmonary disease and its correlation with disease severity. Biomed Res Ther. 2018 Nov 30;5(11):2784-8.
24. Zheng Y, Qi Y, Seery S, Wu Y, Yang L, Zhang B, et al. Elevated HsCRP in chronic obstructive pulmonary disease: A prospective study of long-term outcomes after percutaneous coronary intervention. Int J Chron Obstruct Pulmon Dis. 2022 Sep 23;17:2517-28.
25. Kabir MA, Haque SD, Baker A, Alam MK, Rahman SA. Association between echocardiographic findings in COPD patients with severity of COPD. Mediscope. 2019 Jul;7(1):20-7.
26. Shadani AK, Dhanlakshmi P, Gupta R. An observational study to assess the echocardiographic changes in chronic obstructive pulmonary disease. Int J Sci Res. 2023 Nov;12(11):1376-80.
27. Chaudhari R, Shrimali L. Study of clinical, electrocardiographic and echocardiographic profile in patients with chronic obstructive pulmonary disease. Int J Res Med Sci. 2018 May;6(5):1716-20.
28. Selem AH, Sayed SI, Sayed MA. The impact of pulmonary hypertension on COPD assessment test score and dyspnea scale in patients with COPD. Egypt J Chest Dis Tuberc. 2017 Apr;66(2):273-8.
29. Zhang M, Shi J, Yang W, Li C, Zhang W, Yin H. The relationship between left ventricular dysfunction and COPD severity assessed by CAT score. J Thorac Dis. 2018 Dec;10(12): 6470-7.