Section: General Medicine

Original Research Article

CLINICAL PROFILE OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND CORRELATION OF HIGH-SENSITIVITY C-REACTIVE PROTEIN WITH INCREASING DISEASE SEVERITY: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is increasingly regarded as a systemic inflammatory disorder. High-sensitivity C-reactive protein (hs-CRP) is a marker of low-grade systemic inflammation that may reflect disease severity. The objective is to determine serum hs-CRP levels in patients with COPD and correlate them with forced expiratory volume in one second (FEV1) and Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging, and to describe the clinical, radiological, electrocardiographic and echocardiographic profile of these patients.

Materials and Methods: This prospective observational study enrolled 70 clinically stable patients aged 40-80 years with a post-bronchodilator FEV1/forced vital capacity (FVC) ratio below 0.70, attending a tertiary care hospital over 18 months. All patients underwent chest radiography, spirometry, electrocardiography, two-dimensional echocardiography and serum hs-CRP estimation by chemiluminescent immunoassay. Data were analysed using SPSS v27; a P value below 0.05 was considered significant.

Results: The mean age was 61.06 ± 9.57 years with a male predominance (58.6%); most patients were current smokers (77.1%). Breathlessness (78.6%) and tachypnoea (74.3%) were the commonest symptom and sign. Most patients were in GOLD stage 2 (47.1%). Mean hs-CRP rose progressively with severity, from 0.69 ± 0.4 mg/L in stage 1 to 4.41 ± 0.9 mg/L in stage 4. hs-CRP showed a significant negative correlation with FEV1 ($r = -0.450$, $P < 0.001$) and a strong positive correlation with GOLD staging ($r = 0.603$, $P < 0.001$).

Conclusion: Serum hs-CRP correlates significantly with airflow limitation and GOLD stage and may serve as a simple, cost-effective biomarker of disease severity and systemic inflammation in COPD.

Keywords: Chronic Obstructive Pulmonary Disease, C-Reactive Protein, Forced Expiratory Volume, Inflammation, Spirometry.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable respiratory disorder characterised by persistent airflow limitation and chronic respiratory symptoms such as dyspnoea, cough and sputum production. The disease is usually progressive and is associated with an enhanced chronic inflammatory response of the airways and lungs to noxious particles or gases.[1] COPD is

currently among the leading causes of morbidity and mortality worldwide and, in 2021, accounted for millions of deaths, with the majority of the burden borne by low- and middle-income countries.[2]

In India, COPD represents a major public health challenge, with an estimated prevalence of around 7% among adults, driven not only by tobacco smoking but also by biomass fuel combustion, occupational exposures and ambient air pollution.[3,4] Tobacco smoking remains the most important risk factor; however, non-smoking-related exposures play a crucial role, especially among individuals from rural areas and lower socioeconomic strata, and contribute a sizeable proportion of the global burden.[5,6] These exposures lead to chronic airway inflammation, airway remodelling, alveolar destruction and loss of elastic recoil, ultimately resulting in airflow obstruction and impaired pulmonary function.

Although COPD was traditionally considered a disease limited to the lungs, increasing evidence suggests that it is a systemic inflammatory disorder with several extra-pulmonary manifestations.[7,8] Persistent pulmonary inflammation may spill over into the systemic circulation, producing low-grade systemic inflammation that contributes to comorbidities including cardiovascular disease, skeletal muscle dysfunction, metabolic abnormalities, osteoporosis and anaemia, all of which adversely affect prognosis and quality of life.[9]

C-reactive protein (CRP) is a well-established acute-phase reactant synthesised by the liver in response to inflammatory cytokines, particularly interleukin-6. The development of high-sensitivity CRP (hs-CRP) assays has enabled detection of low-grade inflammation not evident with conventional CRP testing.[10] Serum hs-CRP levels are significantly higher in patients with COPD than in healthy individuals, independent of smoking status and biomass exposure,[11] and elevated levels have been associated with reduced FEV1, greater symptom burden, frequent exacerbations and higher mortality.[12]

Assessment of COPD severity is conventionally based on spirometric parameters, particularly FEV1, together with symptom assessment and exacerbation history, as recommended by GOLD.[1] However, spirometry alone does not adequately reflect the systemic inflammatory burden characteristic of COPD, and incorporation of biomarkers such as hs-CRP may provide additional insight into disease activity and risk stratification.[11]

Despite a high disease burden in India, data correlating clinical severity with systemic inflammatory markers remain scarce. The present study was therefore conducted to assess the clinical profile of patients with COPD and to evaluate the correlation between serum hs-CRP levels and increasing disease severity, so as to explore the value of hs-CRP as a simple, cost-effective biomarker in a resource-limited setting.

MATERIALS AND METHODS

Study design, setting and population: This prospective observational study was conducted in the Department of General Medicine of a tertiary care teaching hospital (Katihar Medical College and Hospital) over 18 months, from March 2024 to August 2025. Patients with clinical evidence of COPD attending the outpatient department, emergency department, medicine wards and intensive care unit were screened for eligibility. The study conformed to the STROBE guidance for observational studies.

Sample size and selection criteria: A total of 70 patients who fulfilled the selection criteria were included. Clinically stable patients aged 40-80 years with a post-bronchodilator FEV1/FVC ratio below 0.70 (after 400 µg inhaled salbutamol) who consented to participate were enrolled.

Exclusion criteria were: bronchial asthma (post-bronchodilator reversibility greater than 12%); pulmonary tuberculosis or pneumonia; diabetes mellitus; ischaemic heart disease; hepatic or renal impairment; and any acute infection, inflammatory or autoimmune disease.

Investigations: All enrolled patients underwent a detailed clinical evaluation together with chest radiography, spirometry, electrocardiography (ECG), two-dimensional echocardiography (2D-echo) and serum hs-CRP estimation. Spirometry was performed using standardised equipment before and after bronchodilator administration (400 µg inhaled salbutamol); FEV1, FVC and the FEV1/FVC ratio were recorded. A post-bronchodilator FEV1/FVC ratio below 0.70 was considered diagnostic of persistent airflow obstruction, and patients with reversibility greater than 12% were excluded. Severity was graded using GOLD spirometric criteria based on FEV1% predicted: stage 1, mild (≥80%); stage 2, moderate (50-79%); stage 3, severe (30-49%); and stage 4, very severe (<30%).

Under aseptic precautions, 2-5 mL of venous blood was collected and hs-CRP was measured by chemiluminescent immunoassay (CLIA). In this sandwich assay, CRP in the patient's serum binds to an anti-CRP antibody coated on the solid phase, a second antibody labelled with a chemiluminescent marker (acridinium ester) forms a sandwich complex, and the emitted light, measured in relative light units, is directly proportional to the hs-CRP concentration. The assay reliably detects low circulating concentrations of CRP.

Ethical considerations: The study protocol was approved by the Institutional Ethics Committee of Katihar Medical College and Hospital. Written informed consent was obtained from all participants prior to inclusion, and confidentiality of patient data was maintained throughout.

Statistical analysis: Data on demographic variables, clinical history and laboratory investigations were collected using a semi-structured proforma. Analysis

was performed using SPSS version 27. Descriptive statistics summarised the data, and the Pearson correlation coefficient was used to assess the relationship of hs-CRP with FEV1 and GOLD staging. A P value below 0.05 was considered statistically significant.

RESULTS

Seventy patients with COPD were studied. The mean age was 61.06 ± 9.57 years; the majority (26; 37.1%) were 51-60 years of age, followed by 61-70 years (18; 25.7%) and 71-80 years (15; 21.4%), with only 11 (15.7%) aged 41-50 years [Table 1]. Males constituted the majority (41; 58.6%) with a male-to-female ratio of 1.41:1 [Table 2]. Regarding body mass index (BMI), 28 (40%) had a normal BMI, 25 (35.7%) were overweight and 17 (24.3%) were underweight [Table 3].

Most patients were current smokers (54; 77.1%), while 7 (10%) were ex-smokers and 9 (12.9%) were non-smokers [Table 4]. According to GOLD staging, 33 (47.1%) were in stage 2, 23 (32.9%) in stage 3, 9 (12.9%) in stage 4 and 5 (7.1%) in stage 1 [Table 5].

Breathlessness was the commonest symptom (55; 78.6%), followed by wheezing (36; 51.4%), cough with sputum (21; 30%) and fever (8; 11.4%) [Table 6]. On examination, tachypnoea was the commonest sign (52; 74.3%), followed by raised jugular venous pressure (JVP) (37; 52.9%), pedal oedema (26; 37.1%), a loud P2 (17; 24.3%), parasternal heave (16; 22.9%) and ascites (8; 11.4%) [Table 7].

On chest radiography, hyperinflation and prominent bronchovascular markings were each seen in 35 (50.0%) patients, cardiomegaly in 34 (48.57%) and a low flat diaphragm in 32 (45.71%) [Table 8]. ECG showed right ventricular hypertrophy in 34 (48.57%), right axis deviation and P pulmonale in 26 (37.1%) each, poor R-wave progression in 15 (21.42%) and low-voltage QRS complexes in 4 (5.7%) [Table 9].

On 2D-echo, pulmonary artery hypertension was the commonest finding (36; 51.40%), followed by right ventricular hypertrophy (34; 48.57%), left ventricular diastolic dysfunction (21; 30.0%), right ventricular dysfunction (18; 25.71%) and left ventricular systolic dysfunction (7; 10%) [Table 10].

Association of hs-CRP with disease severity: Overall, 47 (67.1%) patients had an hs-CRP above 3 mg/L, 16 (22.9%) between 1 and 3 mg/L and 7 (10%) below 1 mg/L. The proportion of patients with hs-CRP above 3 mg/L increased progressively with GOLD stage, from 0% in stage 1 to 63.6% in stage 2, 78.3% in stage 3 and 88.9% in stage 4 [Table 11]. Correspondingly, the mean hs-CRP rose from 0.69 ± 0.4 mg/L in stage 1 to 2.95 ± 1.1 mg/L in stage 2, 4.06 ± 1.1 mg/L in stage 3 and 4.41 ± 0.9 mg/L in stage 4 [Table 12].

On correlation analysis, hs-CRP showed a significant negative correlation with FEV1 ($r = -0.450$, $P < 0.001$) and a strong positive correlation with GOLD staging ($r = 0.603$, $P < 0.001$) [Table 13].

Table 1: Age distribution of study participants

Age group (years)	Number of patients	Percentage (%)
41-50	11	15.7
51-60	26	37.1
61-70	18	25.7
71-80	15	21.4
Total	70	100
Mean age	61.06 ± 9.57 years	

Table 2: Sex distribution of study participants

Sex	Number of patients	Percentage (%)
Male	41	58.6
Female	29	41.4
Total	70	100.0
Male:female ratio	1.41:1	

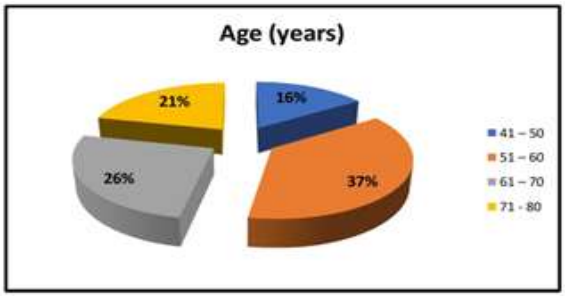

Figure 1: Age distribution of study participants

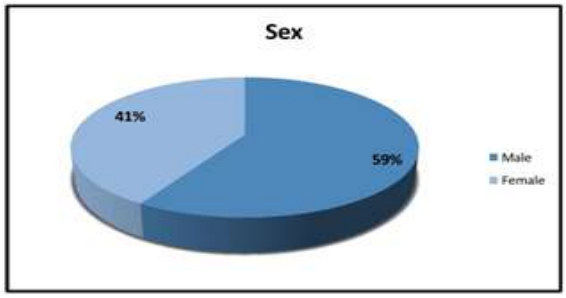

Figure 2: Sex distribution of study participants.

Table 3: Distribution of study population according to BMI

BMI category	Number of patients	Percentage (%)
Normal	28	40.0
Overweight	25	35.7

Underweight	17	24.3
Total	70	100.0

Table 4: Distribution of study population according to smoking status

Smoking status	Number of patients	Percentage (%)
Non-smoker	9	12.9
Current smoker	54	77.1
Ex-smoker	7	10.0
Total	70	100.0

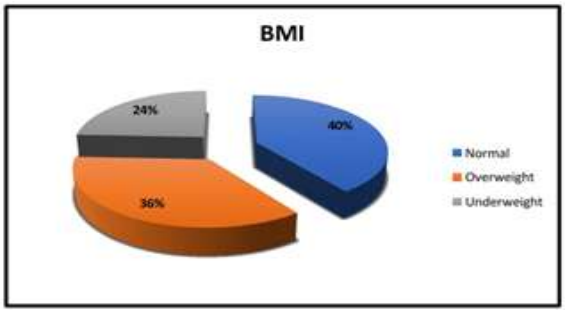

Figure 3: Distribution of study participants according to BMI

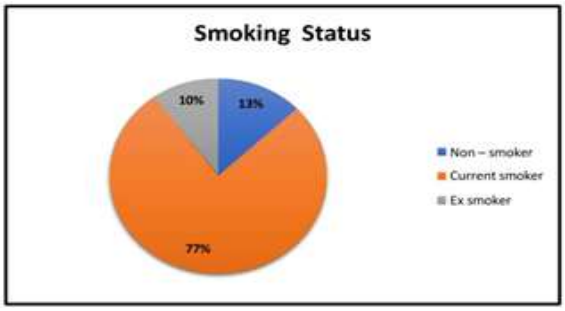

Figure 4: Distribution of study participants according to smoking status.

Table 5: Distribution of study population according to GOLD staging

GOLD stage	Number of patients	Percentage (%)
1	5	7.1
2	33	47.1
3	23	32.9
4	9	12.9
Total	70	100.0

Table 6: Distribution of study population according to symptoms

Symptom	Number of patients	Percentage (%)
Breathlessness	55	78.6
Wheezing	36	51.4
Cough with sputum	21	30.0
Fever	8	11.4

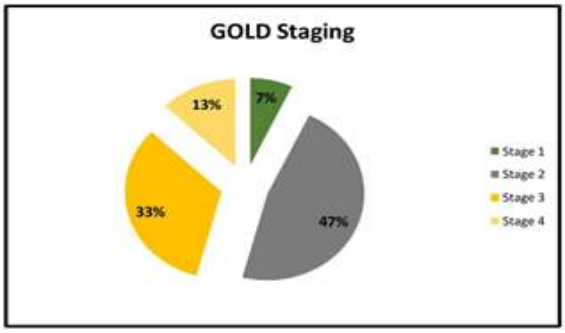

Figure 5: Distribution of study participants according to GOLD staging

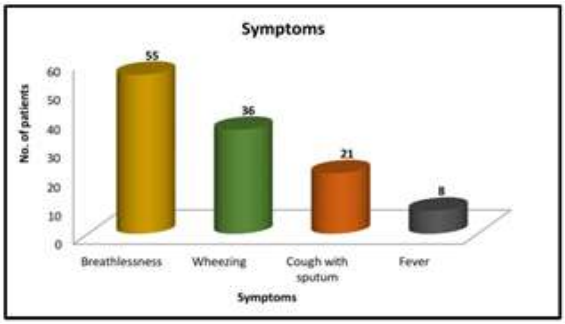

Figure 6: Distribution of study participants according to symptoms.

Table 7: Distribution of study population according to signs

Sign	Number of patients	Percentage (%)
Pedal oedema	26	37.1
Tachypnoea	52	74.3
Loud P2	17	24.3
Parasternal heave	16	22.9
Raised JVP	37	52.9
Ascites	8	11.4

Table 8: Distribution of study population according to chest X-ray findings

Chest X-ray finding	Number of patients	Percentage (%)
Hyperinflation of lungs	35	50.0
Low flat diaphragm	32	45.71
Prominent bronchovascular markings	35	50.0
Cardiomegaly	34	48.57

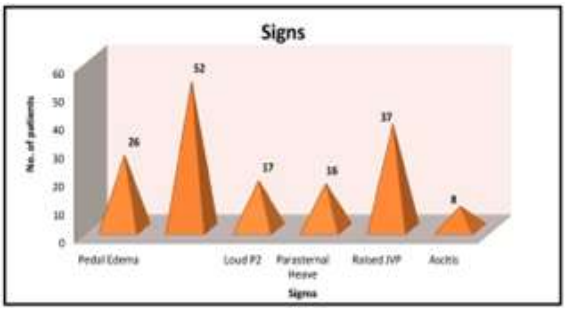

Figure 7: Distribution of study participants according to signs

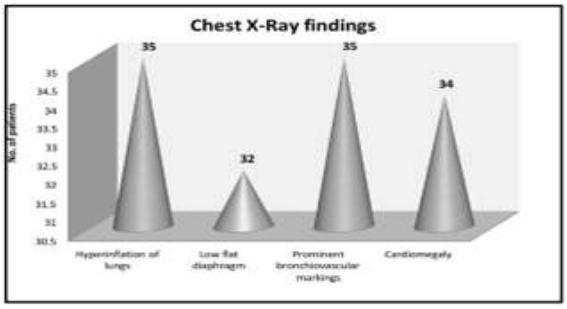

Figure 8: Distribution of study participants according to chest X-ray findings.

Table 9: Distribution of study population according to ECG findings

ECG finding	Number of patients	Percentage (%)
Right axis deviation	26	37.1
P pulmonale	26	37.1
Low-voltage QRS	4	5.7
Poor R-wave progression	15	21.42
Right ventricular hypertrophy	34	48.57

Table 10: Distribution of study population according to 2D-echo findings

2D-echo finding	Number of patients	Percentage (%)
Pulmonary artery hypertension	36	51.40
Right ventricular hypertrophy	34	48.57
Right ventricular dysfunction	18	25.71
LV systolic dysfunction	7	10.0
LV diastolic dysfunction	21	30.0

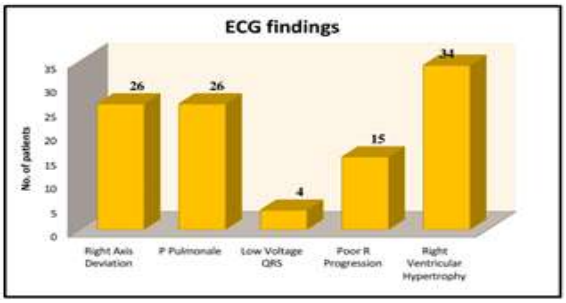

Figure 9: Distribution of study participants according to ECG findings

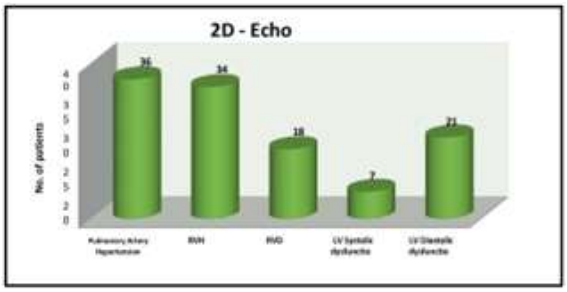

Figure 10: Distribution of study participants according to 2D-echo findings.

Table 11: Association of GOLD staging with hs-CRP levels

GOLD stage	hs-CRP <1 mg/L, n (%)	hs-CRP 1-3 mg/L, n (%)	hs-CRP >3 mg/L, n (%)	Total, n (%)
1	4 (80)	1 (20)	0 (0)	5 (7.1)
2	3 (9.1)	9 (27.3)	21 (63.6)	33 (47.1)
3	0 (0)	5 (21.7)	18 (78.3)	23 (32.9)
4	0 (0)	1 (11.1)	8 (88.9)	9 (12.9)
Total	7 (10)	16 (22.9)	47 (67.1)	70 (100)

Table 12: Association of GOLD staging with mean hs-CRP levels

GOLD stage	Patients, n (%)	hs-CRP, mg/L (mean ± SD)
1	5 (7.1)	0.69 ± 0.4
2	33 (47.1)	2.95 ± 1.1
3	23 (32.9)	4.06 ± 1.1
4	9 (12.9)	4.41 ± 0.9
Total	70 (100)	3.34 ± 1.41

Table 13: Correlation of hs-CRP with FEV1 and GOLD staging

Parameter	Correlation (r)	P value
FEV1	-0.450	<0.001
GOLD staging	0.603	<0.001

DISCUSSION

This study examined the clinical, radiological and echocardiographic profile of COPD and the relationship between serum hs-CRP and disease severity in 70 patients. The majority belonged to the 51-60 year age group (37.1%), with a mean age of 61.06 ± 9.57 years, indicating that COPD

predominantly affects middle-aged and elderly individuals. This is consistent with Mourya et al., who reported that most COPD patients were in the 51-60 year age group.[13]

A male predominance was observed (58.6%; male-to-female ratio 1.41:1). Gupta et al. similarly reported a higher prevalence of COPD in males (11.4%) than females (7.4%).[14] Most patients had a normal BMI (40%), followed by overweight (35.7%) and underweight (24.3%) individuals; Khatri et al. likewise found that most COPD patients were of normal weight.[15] Current smokers formed the majority (77.1%), reinforcing smoking as the principal aetiological factor; Hanga et al. reported COPD prevalence to be markedly higher among current smokers than ex- or non-smokers.[16]

Most patients presented in GOLD stage 2 (47.1%), followed by stages 3, 4 and 1, indicating that patients commonly present with moderate-to-severe airflow limitation. Parasuramalu et al. (stage 2, 42.6%) and Haughney et al. (stage 2, 52.2%) reported comparable distributions.[17,18]

Breathlessness was the commonest symptom (78.6%), followed by wheezing (51.4%), in keeping with the classical clinical profile of COPD; Miravitlles et al. emphasised the contribution of dyspnoea to disease burden, and Peng et al. reported wheezing in approximately half of COPD patients.[19,20] Tachypnoea was the commonest sign (74.3%); Mathangi et al., in a study of cor pulmonale, also reported tachypnoea in nearly all patients, along with raised JVP and pedal oedema in a substantial proportion.[21] Peripheral signs of right heart failure such as raised JVP (52.9%) and pedal oedema (37.1%) were common, and a loud P2 (24.3%) and parasternal heave (22.9%) suggested pulmonary hypertension in a subset, in line with Aurangabadkar et al., who reported pulmonary hypertension in around 40% of COPD patients.[22]

On chest radiography, hyperinflation and prominent bronchovascular markings were each seen in half of the patients, with cardiomegaly and a low flat diaphragm in a substantial proportion; these classical features are comparable to those reported by Khatri et al.[15] ECG and 2D-echo demonstrated a high prevalence of right heart involvement—right ventricular hypertrophy, right axis deviation and P pulmonale on ECG, and pulmonary artery hypertension and right ventricular hypertrophy on echocardiography—consistent with the recognised progression of COPD towards cor pulmonale. Kumari et al. and Nalabothu et al. reported similar ECG associations,[23,24] and Nasir et al. described a high prevalence of elevated pulmonary artery pressures and right ventricular hypertrophy in COPD.[25]

The central finding of this study is a strong, statistically significant association between GOLD stage and hs-CRP. The proportion of patients with hs-CRP above 3 mg/L rose progressively from 0% in stage 1 to 88.9% in stage 4, and mean hs-CRP increased from 0.69 ± 0.4 mg/L in stage 1 to 4.41 ± 0.9 mg/L in stage 4. hs-CRP correlated negatively with FEV1 ($r = -0.450$, $P < 0.001$) and positively with GOLD staging ($r = 0.603$, $P < 0.001$).

These findings agree with previous work. Srivastava et al. showed that CRP correlates with severity and functional impairment in stable COPD,[26] and Agarwal et al. reported a strong negative correlation of hs-CRP with FEV1 ($r = -0.813$) and a positive correlation with GOLD stage ($r = 0.797$); the somewhat weaker correlations in the present study may reflect differences in sample size and severity distribution.[27] Alavi et al. also found a significant positive correlation between hs-CRP and GOLD stage,[28] while Lin et al. reported consistent but weaker correlations across GOLD categories.[29] Shankar et al. further linked higher hs-CRP with increased exacerbation frequency and worse outcomes, supporting its prognostic relevance.[30]

Limitations: This was a single-centre study with a modest sample size and a cross-sectional assessment of hs-CRP, which limits inference about causality and longitudinal change. hs-CRP was measured largely in stable patients, and exacerbation-related dynamics were not fully captured. Larger, multicentre studies with longitudinal follow-up are warranted to confirm the prognostic value of hs-CRP in COPD.

CONCLUSION

COPD is a systemic inflammatory disease with important pulmonary and extra-pulmonary manifestations, occurring predominantly in older males with significant smoking exposure. Serum hs-CRP correlated significantly with GOLD stage and inversely with FEV1, indicating that it may serve as a simple, cost-effective biomarker for assessing disease severity and systemic inflammation. The high prevalence of pulmonary hypertension and right ventricular involvement underscores the need for comprehensive cardiovascular evaluation. COPD management should therefore incorporate not only spirometric assessment but also evaluation of systemic inflammation and cardiac complications for better prognostication and outcomes.

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