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Serum C-Reactive Protein as a Marker of Airflow Limitation in Chronic Obstructive Pulmonary Disease: A Hospital-Based Cross-Sectional Study.

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is now recognised as a disorder with an abnormal inflammatory response extending beyond the lung, producing measurable low-grade systemic inflammation. C-reactive protein (CRP), an acute-phase reactant synthesised chiefly by hepatocytes, reflects the total systemic inflammatory burden and has been proposed as a simple, inexpensive surrogate marker of disease severity in COPD. Whether circulating CRP tracks the degree of airflow limitation remains inconsistently reported. ***Objectives:*** To determine the relationship between serum CRP and lung function, specifically post-bronchodilator forced expiratory volume in one second (FEV1) and the FEV1/forced vital capacity (FEV1/FVC) ratio, in patients with COPD, and to describe the clinico-demographic profile of this population. ***Materials and Methods:*** A descriptive cross-sectional hospital-based study was carried out over 18 months (January 2021 to June 2022) in the Department of General Medicine, Rajarajeswari Medical College and Hospital, Bengaluru. Fifty patients aged over 40 years with spirometrically confirmed COPD (post-bronchodilator FEV1 <80% predicted and FEV1/FVC <0.7) were recruited by purposive sampling. Patients with asthma, tuberculosis, bronchiectasis, recent acute coronary syndrome, heart failure, malignancy, autoimmune disease, renal or hepatic failure, active infection, recent surgery or trauma, and those on statins or hormone replacement therapy were excluded. Spirometry was performed to American Thoracic Society/European Respiratory Society standards before and 20 minutes after 400 µg salbutamol. Fasting venous samples drawn at rest were assayed for serum CRP by latex agglutination. Data were analysed in SPSS v26.0 using Pearson's correlation, with significance set at $p<0.05$. ***Results:*** Mean age was 65.56 ± 9.69 years and 45 (90%) participants were male; 22 (44%) were in the 61–70 year band. Breathlessness, cough and expectoration were present in all 50 (100%) patients, with a mean symptom duration of 4.76 ± 1.79 years. Thirty (60%) were current smokers. By GOLD spirometric grade, 35 (70%) had severe, 11 (22%) moderate and 4 (8%) very severe disease; none had mild disease. Mean serum CRP was 4.5 ± 2.4 mg/L, mean post-bronchodilator FEV1 was 0.43 ± 0.11 L and mean FEV1/FVC was 0.54 ± 0.93. CRP correlated significantly and inversely with post-bronchodilator FEV1 ($r = -0.444$, $p = 0.001$), whereas its correlation with FEV1/FVC was weak and non-significant ($r = -0.194$, $p = 0.177$). ***Conclusion:*** Serum CRP rises as post-bronchodilator FEV1 falls, indicating that systemic inflammatory burden parallels the severity of airflow limitation in COPD. CRP is not related to the FEV1/FVC ratio, which reflects the qualitative presence rather than the quantitative degree of obstruction. A simple latex-agglutination CRP assay may therefore serve as a low-cost adjunct to spirometry in assessing disease severity in resource-limited settings.

Keywords: Chronic obstructive pulmonary disease; C-reactive protein; FEV1; spirometry; systemic inflammation; GOLD classification.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable condition characterised by persistent airflow limitation that is usually progressive and is associated with an enhanced chronic inflammatory response of the airways and lung parenchyma to noxious particles and gases [1]. The structural consequences of this inflammation — small airway narrowing, mucus hypersecretion and loss of elastic recoil through emphysematous destruction — translate clinically into chronic cough, sputum production and exertional dyspnoea, with a spectrum of presentation ranging from minimal symptoms to frank respiratory failure [1,2].

The global burden of the disease is substantial and rising. The World Health Organization projects COPD to become the third leading cause of death worldwide [3]. The Global Burden of Disease Study 2019 documented 212.3 million prevalent cases and 3.3 million attributable deaths in that year alone [4]. India carries a disproportionate share of this burden: national GBD estimates identify COPD as the second leading cause of both death and disability-adjusted life years lost in the country [5], driven by tobacco use, biomass fuel exposure, occupational dust and ambient air pollution.

For several decades COPD was conceptualised as a disease confined to the lung. Evidence accumulated over the last twenty years has decisively reframed it as a systemic disorder. Patients demonstrate persistent low-grade systemic inflammation that manifests as unintentional weight loss, skeletal muscle dysfunction, osteoporosis, depression and an increased risk of cardiovascular events [6,7]. Donaldson and colleagues demonstrated that airway and systemic inflammatory activity accompanies accelerated decline in lung function [8], and Yende et al. linked circulating inflammatory markers to ventilatory limitation and muscle dysfunction in elderly subjects with obstructive lung disease [9]. This systemic component is not captured by spirometry, which measures only the mechanical consequence of airway disease.

Among the inflammatory mediators studied, C-reactive protein (CRP) has attracted the most attention because it is stable, reproducible and cheaply measured in routine laboratories. CRP is a pentameric acute-phase protein synthesised predominantly by hepatocytes under interleukin-6 stimulation in response to tissue injury or inflammation, and its serum concentration reflects the aggregate systemic inflammatory burden of the individual [10]. Pinto-Plata et al. showed that CRP concentrations are significantly higher in patients with COPD than in smoking and non-smoking controls, independent of current smoking status [11]. de Torres et al. and Broekhuizen et al. subsequently related raised CRP to impaired functional capacity and adverse clinically important outcomes in stable disease [12,13]. In a large population cohort, Dahl et al. established elevated CRP as an independent predictor of hospitalisation and death from COPD [14].

The relationship between CRP and the degree of airflow limitation itself, however, remains contested. Several investigators report an inverse association between CRP and FEV1 [15,16], while others find no such relationship once confounders are accounted for [17]. Much of this inconsistency arises from heterogeneity in disease stage, smoking status, comorbidity and assay methodology across studies. Data from Indian hospital populations, in whom biomass exposure and lower body mass index are common, are particularly scarce. The present study was therefore undertaken to assess the relationship between serum CRP and lung function, measured as post-bronchodilator FEV1 and the FEV1/FVC ratio, in patients with COPD attending a tertiary care centre in southern India.

MATERIALS AND METHODS

This was a descriptive cross-sectional hospital-based study conducted in the Department of General Medicine, Rajarajeswari Medical College and Hospital, Bengaluru, over a period of 18 months from January 2021 to June 2022. Participants were drawn from patients diagnosed with COPD attending the outpatient and inpatient services of the department.

Ethical considerations: Clearance was obtained from the Institutional Ethics Committee before commencement of the study. Written informed consent in the participant's own language was obtained from every subject prior to data collection, and confidentiality of records was maintained throughout.

Sample size: Departmental records indicated an average of three eligible cases per month, giving an expected population of N = 54 over the 18-month period. Applying the Yamane formula for a known population, $n = N/(1 + Ne^2)$, with a margin of error of 0.05 at a 95% confidence level, $n = 54/(1 + 54 \times 0.05^2) = 47.78$. The sample size was accordingly rounded and fixed at 50. Participants were enrolled by purposive sampling until this figure was reached.

Inclusion criteria: Patients older than 40 years with chronic cough and sputum production, a modified Medical Research Council (mMRC) dyspnoea grade ≥3, exposure to recognised risk factors (current or former tobacco use, or occupational exposure to dusts and chemicals), and a baseline post-bronchodilator FEV1 <80% predicted with FEV1/FVC <0.7 following inhalation of 400 µg salbutamol. Patients presenting in acute exacerbation, defined as an acute change in symptoms beyond normal day-to-day variation, were eligible.

Exclusion criteria: A history or presence of asthma, pulmonary tuberculosis, bronchiectasis, acute coronary syndrome, myocardial infarction within the preceding six months, congestive cardiac failure, collagen vascular or autoimmune disease, pulmonary embolism, malignancy, renal insufficiency or hepatic cirrhosis. Patients with active infection, recent trauma or surgery, and those with prior use of statins or hormone replacement therapy were also excluded, as these conditions and agents independently alter circulating CRP.

Data collection: A pre-tested, semi-structured questionnaire administered by interview captured sociodemographic details, symptom profile, symptom duration, exacerbation frequency, smoking and occupational history, and comorbidities. Height was measured to the nearest centimetre using a stadiometer with the subject standing barefoot, heels together, back and head against the rod and eyes directed forward. Weight was recorded to the nearest 0.1 kg on an electronic scale in light clothing. Body mass index was derived as weight in kilograms divided by height in metres squared.

Pulmonary function testing: Spirometry was performed with an automated flow-sensing spirometer (Helios v3.1.80) with the subject seated, in accordance with the 2005 ATS/ERS standardisation recommendations [18]. Between three and eight forced expiratory manoeuvres were obtained to satisfy acceptability and repeatability criteria, and the highest FVC and FEV1 from acceptable curves were used. Testing was repeated 20 minutes after nebulised salbutamol. Predicted values were derived from age, sex, height and ethnicity. Severity was graded by the GOLD spirometric classification: GOLD 1 (mild, FEV1 ≥80% predicted), GOLD 2 (moderate, 50–79%), GOLD 3 (severe, 30–49%) and GOLD 4 (very severe, <30%).

Biochemical assay: Venous blood was drawn with the patient at rest after four hours of fasting and before any other investigation was performed. Serum CRP was estimated by the latex agglutination method. Chest radiography, electrocardiography, two-dimensional echocardiography, complete blood count and renal function tests were performed where clinically indicated.

Statistical analysis: Data were compiled in Microsoft Excel and analysed using SPSS version 26.0. Qualitative variables are expressed as frequencies and percentages; quantitative variables as mean ± standard deviation. Comparison of means across groups used analysis of variance (ANOVA), and associations between continuous variables were examined by Pearson's correlation coefficient. The level of significance was fixed at 5% ($\alpha = 0.05$).

RESULTS

Fifty patients meeting the eligibility criteria were studied.

Table 1. Age and sex distribution of study participants (n = 50)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	41–50	5	10.0
	51–60	12	24.0
	61–70	22	44.0
	71–80	10	20.0
	81–90	1	2.0
	Mean ± SD	**65.56 ± 9.69**	—
Sex	Male	45	90.0
	Female	5	10.0
	Total	**50**	**100.0**

The cohort was elderly, with a mean age of 65.56 ± 9.69 years. Nearly two-thirds (64%) were aged 61 years or above, the single largest group being the 61–70 year band (22 patients, 44%). Only 5 patients (10%) were below 51 years, consistent with the cumulative exposure-dependent natural history of COPD. There was a marked male preponderance, with 45 males (90%) to 5 females (10%), reflecting the higher prevalence of tobacco smoking and occupational dust exposure among men in this population.

Table 2. Symptom profile, symptom duration and exacerbation frequency (n = 50)

Variable	Category	Frequency (n)	Percentage (%)
Symptoms present	Breathlessness	50	100.0
	Cough	50	100.0
	Expectoration	50	100.0
Duration of symptoms (years)	1–3	12	24.0
	4–6	30	60.0
	7–9	8	16.0
	Mean ± SD	**4.76 ± 1.79**	—
Exacerbations per year	1–2	19	38.0
	3–4	26	52.0
	5–6	7	14.0
	6–7	4	8.0
	Mean ± SD	**2.86 ± 1.41**	—

The classical symptom triad of breathlessness, cough and expectoration was universal, present in all 50 patients (100%), which is expected given that an mMRC grade ≥3 was an entry requirement. Symptom duration was concentrated in the 4–6 year range (30 patients, 60%), with a mean of 4.76 ± 1.79 years, indicating established rather than incident disease. Exacerbations averaged 2.86 ± 1.41 per year, and just over half the cohort (26 patients, 52%) reported three to four exacerbations annually, placing the majority in the frequent-exacerbator phenotype associated with accelerated lung function decline.

Table 3. Distribution of risk factors and comorbidities (n = 50)

Risk factor / comorbidity	Present, n (%)	Absent, n (%)
Current smoker	30 (60.0)	20 (40.0)*
Occupational exposure to dust/chemicals	9 (18.0)	41 (82.0)
Alcohol consumption	24 (48.0)	26 (52.0)
Diabetes mellitus	23 (46.0)	27 (54.0)
Systemic hypertension	26 (52.0)	24 (48.0)

*The 20 patients categorised as non-current smokers were all ex-smokers; every participant had a tobacco or occupational exposure history as required by the inclusion criteria.

Tobacco exposure was universal, with 30 patients (60%) actively smoking at the time of enrolment and the remaining 20 (40%) being ex-smokers. Occupational exposure to dusts and chemicals was documented in 9 patients (18%). Cardiometabolic comorbidity was common, hypertension being present in 26 patients (52%) and diabetes mellitus in 23 (46%), a burden consistent with the shared systemic inflammatory and smoking-related pathways linking COPD to vascular disease.

Table 4. Distribution by GOLD spirometric severity grade (n = 50)

GOLD grade	Criterion (FEV1 % predicted)	Frequency (n)	Percentage (%)
GOLD 1 — Mild	≥80%	0	0.0
GOLD 2 — Moderate	50–79%	11	22.0
GOLD 3 — Severe	30–49%	35	70.0
GOLD 4 — Very severe	<30%	4	8.0
Total		**50**	**100.0**

The cohort was skewed towards advanced disease. Severe (GOLD 3) obstruction predominated, affecting 35 patients (70%), with a further 4 patients (8%) in the very severe category; together, 78% of participants had GOLD 3–4 disease. No patient had mild disease, an expected consequence of the mMRC ≥3 entry criterion and of the referral pattern to a tertiary hospital, where patients typically present only after substantial functional limitation has developed.

Table 5. Mean serum CRP and pulmonary function values (n = 50)

Variable	Mean ± SD
Serum C-reactive protein (mg/L)	4.50 ± 2.40
Post-bronchodilator FEV1 (L)	0.43 ± 0.11
FEV1/FVC ratio	0.54 ± 0.93

Mean serum CRP was 4.50 ± 2.40 mg/L, a modest elevation above the conventional normal range that is characteristic of the persistent low-grade systemic inflammation of COPD rather than of acute sepsis. Severe airflow limitation is confirmed by a mean post-bronchodilator FEV1 of only 0.43 ± 0.11 L and a mean FEV1/FVC ratio of 0.54, well below the 0.70 diagnostic threshold.

Table 6. Correlation of serum CRP with lung function parameters (n = 50)

Lung function parameter	Pearson correlation coefficient (r)	p value	Interpretation
Post-bronchodilator FEV1	–0.444	0.001*	Significant, moderate negative
FEV1/FVC ratio	–0.194	0.177	Not significant, weak negative

*Significant at $p<0.05$.

This is the principal finding of the study. Serum CRP showed a statistically significant, moderate inverse correlation with post-bronchodilator FEV1 ($r = –0.444$, $p = 0.001$); an r value of –0.444 indicates that approximately 20% of the variance

in FEV1 ($r^2 \approx 0.197$) is shared with variation in systemic inflammatory burden. In practical terms, the lower the FEV1, the higher the circulating CRP. By contrast, the correlation between CRP and the FEV1/FVC ratio was weak and did not reach statistical significance ($r = –0.194$, $p = 0.177$). This dissociation is physiologically coherent: FEV1 is a quantitative measure of the magnitude of airflow limitation, whereas FEV1/FVC is largely a qualitative diagnostic ratio that plateaus once obstruction is established, and in advanced disease is further blunted by the concomitant fall in FVC due to gas trapping.

DISCUSSION

The mean age of 65.56 ± 9.69 years in the present cohort closely mirrors that reported by Rossato Silva et al. (64.8 ± 8.5 years) [19] and Milačić (67.76 ± 9.39 years) [15], confirming that COPD requiring hospital care is a disease of the seventh decade. The male preponderance of 90% is consistent with Aksu et al., in whom 86.5% of patients were male, and with Milačić, who reported 73.2% [15,20]. This gender skew reflects the historically higher prevalence of smoking among Indian men, although the contribution of biomass exposure among women is likely under-recognised in hospital-based series.

All 50 patients presented with the full symptom triad, and mean symptom duration of 4.76 ± 1.79 years was comparable with the median of five years reported by Sarioglu et al. [21]. Mean exacerbation frequency of 2.86 ± 1.41 per year was higher than in that series, plausibly reflecting delayed presentation, poorer access to maintenance inhaled therapy and higher ambient pollution exposure in this setting. Current smoking prevalence of 60% exceeded the 29.2% reported by Aksu et al. and the 43% reported by Milačić [15,20], while occupational exposure at 18% was considerably lower than the 62.9% of Aksu et al. Comorbid diabetes (46%) and hypertension (52%) were both substantially commoner than the 10% and 21.3% respectively in the Turkish cohort, underscoring the cardiometabolic clustering characteristic of Indian populations.

The distribution of disease severity was heavily weighted towards GOLD 3, with 70% of patients in this grade compared with 29.2% in Aksu et al. and 50.3% in Milačić [15,20]. No patient had mild disease, whereas Aksu et al. reported 11.2%. Mean CRP of 4.50 ± 2.40 mg/L was considerably lower than the 25.09 ± 29.72 mg/L of Milačić, a difference attributable to assay methodology and to the inclusion of acutely exacerbating patients in that series. Mean post-bronchodilator FEV1 of 0.43 ± 0.11 L was comparable with the 0.542 ± 0.19 L of Aksu et al. and lower than the 1.26 ± 0.54 L of Milačić, while the FEV1/FVC ratio of 0.54 approximated the 0.608 ± 0.92 of Aksu et al. [20].

The central observation — a significant inverse correlation between CRP and post-bronchodilator FEV1 ($r = –0.444$, $p = 0.001$) — is concordant with a substantial body of evidence. Broekhuizen et al. [13], de Torres et al. [12], Corsonello et al. [16], Dahl et al. [14], Lazovic [22], Aksu et al. [20] and Agarwal et al. [23] all reported significant associations between CRP and FEV1. Pinto-Plata et al. similarly demonstrated significantly higher CRP concentrations in COPD patients than in controls [11]. Ghobadi et al., by contrast, found no significant correlation between CRP and FEV1 [17], a discordance likely attributable to their exclusively stable cohort and to use of the high-sensitivity assay.

The absence of correlation with FEV1/FVC ($r = –0.194$, $p = 0.177$) has a coherent physiological basis, as the ratio behaves as a dichotomous diagnostic threshold rather than a graded severity measure and is confounded by progressive hyperinflation reducing FVC in parallel with FEV1.

Limitations: The single-centre design, modest sample of 50, purposive sampling and use of a semi-quantitative latex agglutination assay rather than high-sensitivity CRP limit generalisability. The cross-sectional design precludes inference about causality or prognosis, and residual confounding from smoking intensity and unmeasured comorbidity cannot be excluded.

CONCLUSION

In this hospital-based cross-sectional study of 50 patients with COPD, serum C-reactive protein correlated significantly and inversely with post-bronchodilator FEV1 ($r = –0.444$, $p = 0.001$), demonstrating that the systemic inflammatory burden increases in step with the severity of airflow limitation. No significant relationship was observed between CRP and the FEV1/FVC ratio, which functions as a diagnostic rather than a severity index. The study population was predominantly elderly and male, with severe (GOLD 3) obstruction in 70% and a high prevalence of cardiometabolic comorbidity. These findings support the concept of COPD as a systemic inflammatory disorder and suggest that serum CRP, measurable by an inexpensive and widely available assay, may serve as a useful adjunct to spirometry in the assessment of disease severity — particularly in resource-constrained settings where reliable spirometry is not always accessible. Prospective longitudinal studies with high-sensitivity CRP assays and larger, multicentre samples are needed to establish whether serial CRP measurement adds independent prognostic value.

REFERENCES

1. Vestbo J, Hurd SS, Agustí AG, Jones PW, Vogelmeier C, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2013;187(4):347–65.
2. Singh D, Agusti A, Anzueto A, Barnes PJ, Bourbeau J, Celli BR, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2019. Eur Respir J. 2019;53(5):1900164.
3. World Health Organization. World health statistics 2022: monitoring health for the SDGs, sustainable development goals. Geneva: World Health Organization; 2022.
4. Safiri S, Carson-Chahhoud K, Noori M, Nejadghaderi SA, Sullman MJM, Ahmadian Heris J, et al. Burden of chronic obstructive pulmonary disease and its attributable risk factors in 204 countries and territories, 1990–2019: results from the Global Burden of Disease Study 2019. BMJ. 2022;378:e069679.
5. Salvi S, Kumar GA, Dhaliwal RS, Paulson K, Agrawal A, Koul PA, et al. 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;6(12):e1363–74.
6. Gan WQ, Man SFP, Senthilselvan A, Sin DD. Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and a meta-analysis. Thorax. 2004;59(7):574–80.
7. Garrod R, Marshall J, Barley E, Fredericks S, Hagan G. The relationship between inflammatory markers and disability in chronic obstructive pulmonary disease (COPD). Prim Care Respir J. 2007;16(4):236–40.
8. Donaldson GC, Seemungal TAR, Patel IS, Bhowmik A, Wilkinson TMA, Hurst JR, et al. Airway and systemic inflammation and decline in lung function in patients with COPD. Chest. 2005;128(4):1995–2004.
9. Yende S, Waterer GW, Tolley EA, Newman AB, Bauer DC, Taaffe DR, et al. Inflammatory markers are associated with ventilatory limitation and muscle dysfunction in obstructive lung disease in well functioning elderly subjects. Thorax. 2006;61(1):10–6.
10. Karadag F, Kirdar S, Karul AB, Ceylan E. The value of C-reactive protein as a marker of systemic inflammation in stable chronic obstructive pulmonary disease. Eur J Intern Med. 2008;19(2):104–8.
11. Pinto-Plata VM, Müllerova H, Toso JF, Feudjo-Tepie M, Soriano JB, Vessey RS, et al. C-reactive protein in patients with COPD, control smokers and non-smokers. Thorax. 2006;61(1):23–8.
12. de Torres JP, Cordoba-Lanus E, López-Aguilar C, Muros de Fuentes M, Montejo de Garcini A, Aguirre-Jaime A, et al. C-reactive protein levels and clinically important predictive outcomes in stable COPD patients. Eur Respir J. 2006;27(5):902–7.
13. Broekhuizen R, Wouters EFM, Creutzberg EC, Schols AMWJ. Raised CRP levels mark metabolic and functional impairment in advanced COPD. Thorax. 2006;61(1):17–22.
14. Dahl M, Vestbo J, Lange P, Bojesen SE, Tybjærg-Hansen A, Nordestgaard BG. C-reactive protein as a predictor of prognosis in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2007;175(3):250–5.
15. Milačić N, Milačić B, Dunjić O, Milojković M. Correlation of C-reactive protein and COPD severity. Acta Clin Croat. 2016;55(1):41–7.
16. Corsonello A, Pedone C, Battaglia S, Paglino G, Bellia V, Antonelli Incalzi R. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) as inflammation markers in elderly patients with stable chronic obstructive pulmonary disease (COPD). Arch Gerontol Geriatr. 2011;53(2):190–5.
17. Ghobadi H, Fouladi N, Beukaghazadeh K, Ansarin K. Association of high sensitive CRP level and COPD assessment test scores with clinically important predictive outcomes in stable COPD patients. Tanaffos. 2015;14(1):34–41.
18. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al. Standardisation of spirometry. Eur Respir J. 2005;26(2):319–38.
19. Rossato Silva D, Coelho Gazzana M, Knorst MM. C-reactive protein levels in stable COPD patients: a case-control study. Int J Chron Obstruct Pulmon Dis. 2015;10:1719–25.
20. Aksu F, Capan N, Aksu K, Ofluoğlu R, Canbakan S, Yavuz B, et al. C-reactive protein levels are raised in stable chronic obstructive pulmonary disease patients independent of smoking behavior and biomass exposure. J Thorac Dis. 2013;5(4):414–21.
21. Sarioglu N, Alpaydin AO, Coskun AS, Celik P, Ozyurt BC, Yorgancioglu A. Relationship between BODE index, quality of life and inflammatory cytokines in COPD patients. Multidiscip Respir Med. 2010;5(2):84–91.
22. Lazovic B. Correlation of CRP and serum level of fibrinogen with severity of disease in chronic obstructive pulmonary disease patients. Med Arh. 2012;66(3):159–60.
23. Agarwal R, Zaheer MS, Ahmad Z, Akhtar J. The relationship between C-reactive protein and prognostic factors in chronic obstructive pulmonary disease. Multidiscip Respir Med. 2013;8(1):63.