JGPEMN
ISSN 2091-279X
eISSN 2362-1168

ORIGINAL ARTICLE

Lipid profile status in chronic obstructive pulmonary disease patients with acute exacerbation and its correlation with the severity and treatment of the disease presenting at a tertiary hospital in Nepal

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Date of submission: 18 Jan 2026

Date of acceptance: 30 Jan 2026

Date of Publication: 08 Feb 2026

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How to cite:
Nakarmi RN, Shrestha D, Shakya B, Budha B. Lipid profile status in chronic obstructive pulmonary disease patients with acute exacerbation and its correlation with the severity and treatment of the disease presenting in tertiary hospital in Nepal. J Gen Pract Emerg Med Nepal. 2026 Jun;13(1):6-10.

Online information
DOI:
https://doi.org/10.59284/jgpeman396

Abstract

Introduction: Most of the morbidity and mortality in chronic obstructive pulmonary disease patients result from extra-pulmonary complications, especially cardiovascular complications for which dyslipidemia is a major risk factor. So, the aim of our study was to correlate dyslipidemia with the severity of acute exacerbations of COPD(AECOPD).

Method: A hospital-based, prospective, cross-sectional study of 6 months' duration was conducted among 150 COPD patients presenting with acute exacerbation. Ethical approval was obtained from the Institutional Review Committee of the Institute of Medicine. Demographic and clinical variables, including age, sex, ethnicity, BMI, dyspnea grading, and severity of COPD presentation were recorded. Fasting lipid profile was analyzed and correlated with disease severity.

Result: The majority of patients were aged over 70 years (ranging from 42 to 83 years, with a mean age of 69.9 +/-9.5 years), were female (86, 57.3%), and belonged to the Brahmin ethnicity (60, 40%). Most of them presented in exacerbation with Dyspnea (MMRC grade III), 87 (58%), with a mean body mass index of 26.90 kg/m². On our evaluation, the median triglyceride, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol were found to be 140.22, 127.71, 38.7, 120; 175.28, 166.41, 30.24, 147.06; and 192.81, 183.83, 23.22, 158.67 mg/dl in moderate, severe, and very severe AECOPD cases, respectively.

Conclusion: Greater severity of AECOPD was associated with higher values of atherogenic lipid parameters (TC, TGs, and LDL) and lower HDL levels.

Keywords: Acute exacerbations, COPD, Dyslipidemia, Lipid profile

INTRODUCTION

Chronic Obstructive Pulmonary Disease is a disease of the respiratory system characterized by progressive, but not fully reversible, deterioration of lung function, encompassing both emphysema and chronic bronchitis.[1] It is associated with extra-pulmonary co-morbidities such as cardiovascular disease, metabolic syndrome, depression, cancer, as well as an increased risk of hospitalization and mortality.[2] It is one of the most important causes of morbidity and mortality worldwide, rising from the 4th most common non-communicable disease in 1990 to the 3rd in 2017.[3] In Nepal in 2019, chronic obstructive pulmonary disease (COPD) was responsible for 16.3% of total deaths, a significant increase from 6.1% of total deaths in 1990.[7]COPD becomes worse and complicated by frequent exacerbations known as Acute Exacerbation Chronic Obstructive Pulmonary Disease (AECOPD), which is characterized by intermittent worsening of chronic symptoms, including dyspnea, cough, sputum production, and sputum purulence.[8] Respiratory infection is the main factor associated with AECOPD, but other factors such as industrial pollutants, allergens, sedatives, congestive heart failure, and pulmonary embolism have also been identified to cause AECOPD.[9]

Cardiovascular disease (CVD) is an important cause of morbidity and mortality in COPD patients, and Dyslipidemia, an imbalance of lipids such as cholesterol (TC), Low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and high-density lipoprotein (HDL-C), is a major risk factor for cardiovascular disease (CVD).[10,11] Similarly, metabolic syndrome, an important risk factor for CVD, is also very common in COPD patients, ranging from 20 to 58 percent.[28] However, the association of serum lipid profile with COPD is not clearly understood till now. Some studies found that there is no link between serum lipid profile and COPD,[12,13,14] whereas others show an increase in total cholesterol, low-density lipoprotein, and triglycerides, and a lower level of high-density lipoprotein.[28,29,30] However, none of them studied the correlation between AECOPD and dyslipidemia. Thus, to resolve this contradiction and assess the correlation between AECOPD and dyslipidemia, we evaluated lipid profiles (TC, TG, LDL-C, VLDL-C, and HDL-C) in COPD patients with acute exacerbation and examined the relationship between dyslipidemia and exacerbation severity.

METHOD

This is a quantitative hospital-based non-probability prospective cross-sectional study carried out in the Emergency Department and the Medical Observational Ward of Tribhuvan University Teaching Hospital. The study was approved by the Institutional Review Committee of the Institute of Medicine, Tribhuvan University [IRC: 502 (6-11) E2 077/078]. The study was done from 18th June 2021 to 18th December 2021.

Sample size was calculated using the following formula:

$$\text{Sample size (N)} = \frac{Z_{1-\alpha}^{2}\, p(1-p)}{d^{2}}$$

Here,

$Z_{1-\alpha}$ = standard normal variate (at 5% type I error, $p<0.05$, it is 1.96)

P = expected proportion of COPD based on previous studies 0.117 [5]

d = precision of estimate or absolute error, taken as 5%

Applying the above values to the formula, the sample size comes out to be 158.74 ~159 → 160

Adding 10% non-response rate, the final sample size will be 176

Initially, 176 patients were enrolled in the study. The study included patients with known COPD who presented to the Emergency Department with acute exacerbation and were admitted to the observation and medicine wards. Those patients who were on lipid-modifying drugs, known cases of cardiac co-morbidities, critically ill patients, diagnosed cases of CKD, diagnosed thyroid disorders, patients with bronchial asthma, bronchiectasis or pulmonary TB, and patients on steroid therapy were excluded from this study. We enrolled 150 cases that met the inclusion criteria in the study, while 26 were excluded [16 for not meeting the inclusion criteria and 10 for not giving consent]. (Among 16 cases, 10 cases were found to be on statin therapy previously but non-compliant during follow-up with lipid profile, so these 10 cases are excluded from the study duration, leading to a sample size of 150). Informed consent was obtained after a brief introduction and explanation of the study to the patients and attending families.

Various study variables (age, sex, ethnicity, BMI, dyspnea grading, and severity of COPD presentation) were recorded. The patient's weight was measured in kilograms and height in meters to calculate body mass index (BMI). The World Health Organization (WHO) defines the following cut-points for BMI: <18.5 kg/m^2 underweight, 18.5–24.9 kg/m^2 healthy weight, 25.0–29.9 kg/m^2 overweight, and ≥30 kg/m^2 obese.[20] The diagnosis of AECOPD was made according to the *Anthonisen criteria*.[21] These criteria define AECOPD into three categories based on increased sputum volume, increased sputum purulence, and increased dyspnea. In addition to the *Anthonisen criteria*, detailed histories were obtained from all patients. Furthermore, clinical examination and radiological tests were performed to confirm the presence of AECOPD. The Modified Medical Research Council (mMRC) dyspnea scale was used to stratify dyspnea severity.[22, 23] After a 12-hour fast, 5 ml fasting blood samples were collected from all the participants in the morning. TC, HDL-C, and TG were directly analyzed using the standard enzymatic techniques. The *Friedewald equation* was used to calculate LDL-C.[24] To minimize measurement bias, standardized diagnostic criteria and laboratory methods were used for all participants.

Data entry was performed using Microsoft 2013 and,

upon completion of data collection, SPSS v26. Statistical techniques were used to evaluate the data. Contineous variables were summarized as mean±standard deviation or median(interquartile range), as appropriate and compared using *the Kolmogorov-Smirnov test for distributional differences and the Kruskal-Wallis test for hypothesis testing*. Tables and bar graphs were created as needed. All analytical tests had a 0.05 level of significance; if the p-value was less than 0.05, it was considered statistically significant.

RESULT

The minimum age in the study sample was 42 years, and the maximum age was 83 years. The mean age of the study participants was 69.87±9.53 years. There is an increasing trend in the number of cases and the severity of AECOPD with increasing age among the study participants.

Table 1. Sociodemographic and clinical profile of patients with severity of AECOPD(n=150)

Patients profile	Description	*f* (%)
Age (Years)	40-49	6(4.00%)
	50-59	13(8.70%)
	60-69	38(25.3%)
	>70	93(62.0%)
Sex	Female	86(57.3%)
	Male	64(42.7%)
Ethnicity	Brahmin	60(40.0%)
	Tamang	13(8.70%)
	Chhetri	32(21.3%)
	Magar	12(8.00%)
	Newar	14(9.30%)
	Others	19(12.7%)
BMI (Kg/m^2)	20-24	48(32.0%)
	25-28	71(47.33%)
	29-32	22(14.67%)
	33-36	9(6.0%)
Dyspnea	Grade 3	87(58.0%)
	Grade 4	63(42.0%)
AECOPD Severity	Type I (very severe)	42(28.0%)
	Type II (severe)	91(60.7%)
	Type III (moderate)	17(11.3%)

The frequency of AECOPD is higher in people aged more than 70 years, and the people in this age group have the most severe cases of AECOPD.

In our study, female patients, 86 (57.3%), presented with more exacerbations than male patients, 64 (42.7%). In each age category, the number of females predominates the number of males. Type II severity of AECOPD is more common in both males and females, followed by Type I.

In our study, most of the patients were from Brahmin ethnicity, 60 (40%), followed by Chhetri, 32 (21.3%), and then by Newar, 14 (9.3%). Type II severity of AECOPD was the most common type in all the ethnic groups.

Among 150 patients, 87 (58%) patients presented with grade 3 dyspnea, while 63 (42%) patients presented with Grade 4 dyspnea. In both dyspnea grades, type II severity was more common, followed by type I.

Because lipid parameters were not normally distributed across COPD severity, as confirmed by the Kolmogorov-Smirnov test, the Kruskal-Wallis test was used to assess differences in these parameters among moderate to very severe cases of AECOPD. The distributions of TG, TC, HDL, and LDL were significantly different across Type III, Type II, and Type I AECOPD severity.

DISCUSSION

We found that most cases were in people aged 50 or older, which is the common age group for COPD globally. Multiple factors play a role in the delayed occurrence of COPD, such as impairment of the immune function of both innate and adaptive immunity, as well as the cumulative exposure to smoking and pollutants over the long term.[4] Also, with aging, there is a higher pro-inflammatory and oxidative state in leukocytes than in controls, which accelerates the destruction of alveolar septa and thus causes COPD.[18,19]

Across all age groups, we found that more women have AECOPD, suggesting greater susceptibility. Several other studies have also claimed gender differences in the diagnosis and clinical manifestations of COPD. Possible reasons behind this finding can be that women are less responsive to long-term exercise therapy,[20] score lower in quality-of-life questionnaires,[21] manifest more reactive airways,[22]and report more dyspnea than men.[23]

Table 2. Association of severity of COPD and lipid parameters (all values are in mmol/L and values in brackets are in mg/dL)

Severity of COPD	Type III			Type II			Type I (most severe)			p
Lipid parameters	Median	Q1	Q3	Median	Q1	Q3	Median	Q1	Q3	
TG	1.6 (140.22)	1.3	1.8	2(175.28)	1.8	2.2	2.2(192.81)	1.875	2.4	<0.001
TC	3.3(127.71)	2.85	4.35	4.3(166.41)	3.3	4.9	4.75(183.83)	3.9	5.3	<0.001
HDL	1(38.7)	0.8	1.2	0.8(30.24)	0.6	1	0.6(23.22)	0.4525	0.8	<0.001
LDL	3.1(120)	2.8	3.95	3.8(147.06)	3.1	4.4	4.1(158.67)	3.425	4.625	0.025

Significance level is 0.050.
[Statistical tests used to obtain the p-values: Kolmogorov–Smirnov and Kruskal–Wallis H test]
Kolmogorov–Smirnov test was first used to assess the normality of lipid parameters (TG, TC, HDL, and LDL). Since the lipid parameters were not normally distributed, a Kruskal–Wallis H test was applied. The Kruskal–Wallis test was used to compare the median values of lipid parameters across three independent groups (Type III, Type II, and Type I AECOPD severity). The reported p-values in the table represent the results of the Kruskal–Wallis test.

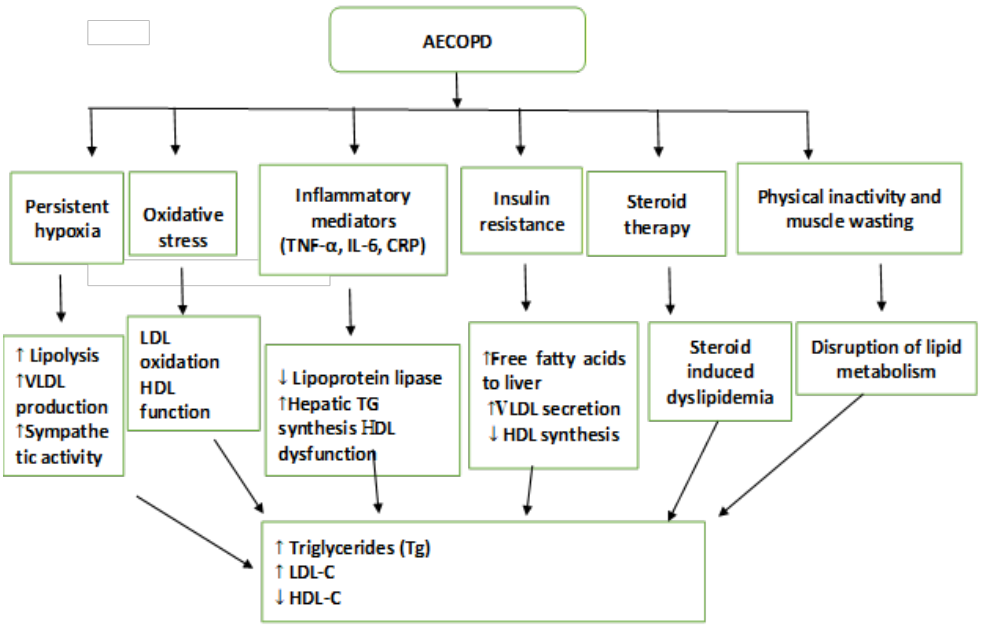

Figure 1. Pathophysiology of dyslipidemia in AECOPD

Some studies concluded that lower BMI accelerates the progression towards the bad stage, and obesity seems to be a protective factor for COPD.[18,19] Rafie et al found that patients with an underprivileged background have lower BMI due to poor nutritional status, which accelerates the severity of COPD. [13]But in our study, most patients with type II and type I AECOPD were overweight, whereas the comparative study uses a denominator that includes stable patients. Similar findings were seen in another study done by Lambert et al.[26] Sedentary lifestyle and increased use of steroids might be the reasons for the increasing BMI with increasing severity of COPD in our study.

Our study primarily focused on lipid profiles in patients with AECOPD and found a strong correlation between dyslipidemia and disease severity. AECOPD patients had significantly higher serum levels of TC, TG, and LDL, and lower HDL levels, and the severity of these derangements was greater with more severe AECOPD. One meta-analysis by Xuan L et al. in 2018 concluded that there were no significant differences in lipid profiles between COPD patients and controls; however, in a subgroup analysis excluding participants taking lipid-modifying drugs, they found higher TG levels in stable COPD patients than in controls.[28] However, none of the studies took Acute Exacerbations of COPD into consideration, which was the main factor we evaluated. In our study, not only was a deranged lipid profile present in AECOPD patients, but the severity of the derangements was positively correlated with the severity of the AECOPD. Dyslipidemia in COPD patients might be due to smoking, aging, inflammatory markers, and sedentary lifestyle, but greater derangements with greater severity of exacerbations are most likely due to recurrent use of corticosteroids, significant limitation in physical activity, and pro-inflammatory states of the patients.

With dyslipidemia, patients with AECOPD may encounter cardiovascular complications, which are among the major causes of morbidity and mortality in these patients. This was a single-center study with a limited number of patients; therefore, the results cannot be generalized. Only acutely exacerbated cases were included, so the prevalence of dyslipidemia in stable COPD may have been missed.

CONCLUSION

Although there are ambiguous findings regarding the correlation between COPD patients and dyslipidemia in earlier studies, there is a significant correlation between COPD patients with acute exacerbations and dyslipidemia. The more severe the exacerbation, the higher the values of lipid parameters (TC, TGs, LDL) and the lower the HDL level.

DECLARATIONS

Acknowledgement

We express our sincere thanks to the Department of General Practice and Emergency Medicine and the Respiratory Medicine Unit of Tribhuvan University Teaching Hospital, Kathmandu, for providing us with the opportunity to conduct this study on their premises. Special acknowledgment to all who were directly or indirectly involved in this research for their valuable help.

Conflict of Interests

None

Funding

None

Ethical Clearance

Ethical clearance was obtained from Institutional Review Committee of Institute of Medicine, Tribhuvan University [IRC: 502 (6-11) E2 077/078]. Written consent was obtained from the Department of General Practice and Emergency Medicine, Tribhuvan University Teaching Hospital, before data collection.

Consent for Publication

Written informed consent was obtained from all participants prior to their inclusion in the study.

Availability of Data

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

REFERENCES

1. Viegi G, Pistelli F, Sherrill DL, Maio S, Baldacci S, Carrozzi L. Definition, epidemiology and natural history of COPD. Eur Respir J. 2007 Nov; 30(5):993–1013. | DOI | PubMed | Full Text |
2. Global initiative for chronic obstructive lung disease (GOLD). Pocket guide to COPD diagnosis, management, and prevention: a guide for health care professionals. 2023. | DOI | Weblink |
3. 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 burden of disease study 2010. Lancet. 2012 Dec 1; 380(9859):2095–128. | DOI | PubMed | Full Text |
4. Bhandari GP, Angdembe MR, Dhimal M, Neupane S, Bhusal C. State of non-communicable diseases in Nepal. BMC Public Health. 2014 Jan 10; 14(1):23. | DOI | PubMed | Full Text |
5. Dhimal M, Karki KB, Sharma SK, Aryal KK, Shrestha N, Poudyal A, Mahato NK, Karakheti A, Sijapati MJ, Khanal PR, Mehata S, Vaidya A, Yadav BK, Adhikary KP, Jha AK. Prevalence of selected chronic non-communicable diseases in Nepal. J Nepal Health Res Counc. 2019 Nov 14; 17(3):394-401. | DOI | PubMed | Full Text |
6. Bhandari R, Sharma R. Epidemiology of chronic obstructive pulmonary disease: A descriptive study in the mid-western region of Nepal. Int J Chron Obstruct Pulmon Dis. 2012; 7:253–7. | DOI | PubMed | Full Text |
7. Nepal Burden of Disease 2019: A country report based on the GBD 2019 Study. [Cited 2022 Jun 26]. | Weblink | Full Text |
8. Sharma S, Anthonisen N. Role of antimicrobial agents in the management of exacerbations of COPD. Treat Respir Med. 2005; 4(3):153–67. | DOI | PubMed | Full Text |
9. Hogea SP, Tudorache E, Fildan AP, Fira-Mladinescu O, Marc M, Oancea C. Risk factors of chronic obstructive pulmonary disease exacerbations. Clin Respir J. 2020 Mar; 14(3):183–97. | DOI | PubMed | Full Text |
10. Fakhrzadeh H, Tabatabaei-Malazy O. Dyslipidemia and cardiovascular disease. In: dyslipidemia - from prevention to treatment [Internet]. 2012 [cited 2022 Jul 3]. | DOI | Weblink | Full Text |
11. Kuo PT. Dyslipidemia and coronary artery disease. Clin Cardiol. 1994; 17(10):519–27. | DOI | PubMed | Full Text |
12. Basili S, Ferroni P, Vieri M, Cardelli P, Ceci F, Paradiso M, et al. Lipoprotein(a) serum levels in patients affected by chronic obstructive pulmonary disease. Atherosclerosis. 1999 Sep 9; 147(2):249–52. | DOI | PubMed | Full Text |
13. Rafie S, Moitra S, Brashier BB. Association between the serum metabolic profile and lung function in chronic obstructive pulmonary disease. Turk Thorac J. 2018; 19(1):13. | DOI | PubMed | Full Text |
14. Begum K, Begum M, Sarker Z, Dewan M, Siddique M. Lipid profile status of chronic obstructive pulmonary disease in hospitalized patients. Bangladesh J Med Biochem. 2013; 3(2):42–5. | DOI | Weblink |
15. Hajiro T, Nishimura K, Tsukino M, Ikeda A, Koyama H, Izumi T. Analysis of clinical methods used to evaluate dyspnea in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1998; 158 (4):1185–9. | DOI | PubMed | Full Text |
16. Mahler DA, Wells CK. Evaluation of clinical methods for rating dyspnea. Chest. 1988 Mar; 93(3):580–6. | DOI | PubMed | Full Text |
17. Martin SS, Blaha MJ, Elshazly MB, Toth PP, Kwiterovich PO, Blumenthal RS, et al. Comparison of a novel method vs the Friedewald equation for estimating low-density lipoprotein cholesterol levels from the standard lipid profile. JAMA. 2013 Nov 20; 310 (19):2061–8. | DOI | PubMed | Full Text |
18. Maté I, Martínez de Toda I, Arranz L, Álvarez-Sala JL, de la Fuente M. Accelerated immunosenescence, oxidation and inflammation lead to a higher biological age in COPD patients. Exp Gerontol. 2021 Oct 15; 154:111551. | DOI | PubMed |
19. Lange P, Celli B, Agustí A, et al. Lung-function trajectories leading to chronic obstructive pulmonary disease. N Engl J Med. 2015;373 (2):111-122. | DOI | PubMed | Full Text |
20. Foy CG, Rejeski WJ, Berry MJ, Zaccaro D, Woodard CM. Gender moderates the effects of exercise therapy on health-related quality of life among COPD patients. Chest. 2001; 119(1):70–6. | DOI | PubMed |
21. Cabrita B, Gonçalves G, Cabrita A, Dias S. Quality of life in chronic obstructive pulmonary diseases. In: Esquinas AM, editor. Pulmonary function measurement in noninvasive ventilatory support. Cham: Springer; 2021. | DOI | Weblink |
22. Kanner RE, Connett JE, Altose MD, Buist AS, Lee WW, Tashkin DP, et al. Gender difference in airway hyperresponsiveness in smokers with mild COPD: The lung health study. Am J Respir Crit Care Med. 1994; 150(4):956–61. | DOI | PubMed |
23. Zejda J, Skoczyński S, Brożek G, Kotulska B, Glinka K, Waz S, et al. Gender differences in dyspnea and its association with lung function in COPD and asthma. Eur Respir J. 2017 Sep; 50(Suppl 61):PA4536. | DOI | Weblink |
24. Mohsen SM, Chakroun S, Chaker A, Ayed K, Jameleddine S. Body mass index in COPD: what relationship? Eur Respir J. 2020 Sep 7; 56(Suppl 64):2439. | DOI | Weblink |
25. Zhou Y, Wang D, Liu S, Lu J, Zheng J, Zhong N, et al. The Association between BMI and COPD: The Results of two population-based studies in Guangzhou, China. Chron Respir Dis. 2013; 10(5):567–72. | DOI | PubMed | Full Text |
26. Lambert AA, Putcha N, Drummond MB, Boriek AM, Hanania NA, Kim V, et al. Obesity is associated with increased morbidity in moderate to severe COPD. Chest. 2017; 151(1):68–77. | DOI | PubMed | Full Text |
27. Arora S, Jain R, Mohan A, Tiwari P, Guleria R. Lipid profile status in chronic obstructive pulmonary disease and its association with disease severity. Int J Respir Pulm Med. 2018; 5:089. | DOI | Weblink | Full Text |
28. Xuan L, Han F, Gong L, et al. Association between chronic obstructive pulmonary disease and serum lipid levels: a meta-analysis. Lipids Health Dis. 2018; 17:263. | DOI | PubMed | Full Text |
29. Markelic I, Hlapcic I, Rogic D, Vukovic L, Barisic K, Gojanovic M, et al. Lipid profile and atherogenic indices in patients with stable chronic obstructive pulmonary disease. Nutr Metab Cardiovasc Dis. 2020 Aug 6; 30(10):1714–20. | DOI | PubMed |
30. Krakhmalova O, Hetman O. Lipid profile in COPD patients with coronary artery disease comorbidity. Eur Respir J. 2013; 42: P1865. | DOI | Weblink | Full Text |