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A Prospective Observational Study On the Assessment of Cardiovascular Risk in Patients with Exacerbation of COPD

Md. Afreen[1], Md. Samreen[2]

[1]Assistant professor, Balaji institute of pharmaceutical sciences

Abstract

The objectives of the study were to compare the following parameters in COPD patients' volunteers age and sex-matched control subjects: lipid parameters and atherogenic indices, viz., atherogenic index of plasma (AIP); cardiovascular risk ratio (CRR); and atherogenic coefficient (AC). To estimate ten-year cardiovascular risk in COPD patients using various cardiovascular risk score calculators, viz., Framingham risk score (FRS-CVD), Joint British Society-3 (JBS3) score, QRISK 2, Reynolds risk score (RRS).

The 10-year scores of the COPD subjects were calculated using CV risk score calculators. The FRS-CVD calculator could identify the maximum number of subjects with high CVD risk (risk score 10-20%), followed by the JBS-3 and QRISK2 calculators. The FRS-CVD calculator showed that of the subjects to be in the high-risk category. JBS3 and QRISK2 were second to FRS-CVD performed intermediately by categorizing 35% and 30% of the COPD subjects to be in the high-risk vents.

Keywords: Cardiovascular risk, COPD chronic obstructive pulmonary disease, Atherogenic index, lipid profile

1. Introduction

- A complex, heterogeneous, preventable, and treatable chronic respiratory disease, chronic obstructive pulmonary disease (COPD) is one of the leading causes of death and morbidity worldwide, accounting for 3 million deaths (GOLD 2016; WHO 2018a). The World Health Organization (WHO) states that COPD is not a single disease but rather a complex, poorly reversible airflow limitation that frequently manifests systemically. COPD is an umbrella term that causes limitations in lung airflow and is diseases, hypertension, and diabetes, COPD is not curable, but treatment can relieve symptoms, improve quality of life, and reduce the risk of death (Gupta et al. 2013). It is well established that COPD is associated with many comorbid diseases, which may be pulmonary or extra-pulmonary. These comorbid diseases in COPD are independently associated with a higher risk of indicative of an atherogenic lipid pattern. Hospitalization and mortality (Fabbri et al. 2008).
- Cardiovascular disease (CVD) is a very common cause of death in COPD patients. In a study involving more than 11,000 COPD patients, cardiovascular mortality was reported to be two times higher than in individuals without COPD (Curkendall et al. 2006).

- Increased cardiovascular risks are shared by many factors, such as smoking, advanced age, decreased physical activity, but chronic systemic inflammation plays a pivotal role in the pathogenesis of COPD and cardiovascular disease. Indeed, systemic inflammation is potentially the common risk factor/pathway of COPD and cardiovascular disease. COPD is characterized by the production of several pro-inflammatory cytokines such as C-reactive protein (CRP), fibrinogen, TNFα, MCP-1, IL-6, and IL-8 (Barnes & Celli 2009). Increased concentrations of circulating inflammatory mediators are also seen in cardiovascular disease. Large-scale prospective studies have identified elevated CRP concentrations as a predictor of future cardiovascular risk and mortality due to myocardial infarction, stroke, and peripheral arterial disease (Ridker 2003).

Therefore, a "spill-over" of increased local airway inflammation into the systemic circulation may occur in COPD, raising concentrations of certain systemic inflammatory markers and eventually contributing to cardiovascular disease. CRP and fibrinogen, two important indicators of cardiac damage, have been found to be elevated in patients with low FEV1 (Donaldson et al. 2010). According to reports, hs-CRP remains stable over an extended period of time in patients with stable COPD, who also have persistent low-grade systemic inflammation (Karadag et al. 2008; Emerging Risk Factors Collaboration 2010; Ridker 2016).

1.1 Global prevalence

According to the Burden of Obstructive Lung Disease (BOLD) study, the average prevalence of COPD is 10.1%, with wide variations. It is higher for males (14%) than for females 9–109–10%, smokers (15.4%), males (9.8%), and people with urban residence (10.2%). The prevalence of COPD (FEV1/FVC lower limits of normal) in never-smokers was 6.4%.

constituting 27% of all COPD subjects.

1.2 Indian prevalence

India suffers a significant, growing percentage of COPD mortality, one of the highest in the world. A systematic review of four studies identified general prevalence of chronic bronchitis in rural areas between 6.5% and reported COPD prevalence between 3 and 8% among Indian
males, and 2.5 to 4.5% for Indian females.

1.3 ETIOLOGY:

COPD is caused by prolonged exposure to harmful particles or gases. Cigarette smoking is the most common cause of COPD worldwide. Other causes may include second-hand smoke, environmental and occupational exposures, and alpha-1 antitrypsin deficiency (AATD).

1.4 DIAGNOSIS OF COPD:

Clinical feature:

COPD describes a group of lung conditions that make it hard to empty the air out of the lungs. This difficulty is characterized by increasing breathlessness or the feeling of being tired with related symptoms such as chronic cough or sputum production, exertion dyspnea, expectoration, wheeze. A clinical diagnosis

of COPD should be considered in any patient who has the above symptoms and a history of exposure to risk factors for the disease (Celli et
al., 2004). Clinical diagnosis needs to be confirmed by standardized spirometric tests in the presence of not-fully-reversible airflow limitation.

Spirometric diagnosis:

Spirometry is required to make a clinical diagnosis of COPD; the presence of a post-fixed ratio of forced expiratory volume in one second (FEV1) to forced vital capacity (FVC), 0.75 to define airflow obstruction (Renzetti et al., 2015). Subsequent ATS documents generically defined airflow obstruction as a decline of FEV1/FVC, without recommending any numerical cut-off point (ATS, 1991). By contrast, ERS guidelines (Quanjer et al., 1993) proposed the diagnosis of airflow obstruction be based on a ratio of FEV1 to slow vital capacity (VC), 88 and 89% of predicted in males and females, respectively. Since 2001, GOLD took a step back, defining COPD by a fixed FEV1/FVC of chau, 2003) evaluating the impact of different definitions of airflow obstruction on the epidemiology of COPD. At variance with the GOLD guidelines, the recent ATS/ERS guidelines on lung function testing stressed the use of lower limits of normality (LLN), i.e. the lower fifth percentile of the frequency distribution of a healthy population, to define pulmonary function
abnormalities.

Differential diagnosis:

A major differential diagnosis is asthma. Other potential diagnoses are usually easier to distinguish from COPD and include congestive heart failure, bronchiectasis, tuberculosis, obliterative bronchiolitis, diffuse panbronchiolitis.

1.5 Four-stage classification:

The GOLD has introduced a four-stage classification of COPD [Table 1] severity based on their degree of airflow limitation measured during pulmonary function tests (PFTs) (Huijsmans, de Haan, ten Hacken, Straver, & van't Hul, 2008). Spirometric classification has proved useful in predicting health status (Ferrer et al., 1997).

utilization of healthcare resources (Dewan et al., 2000), development of exacerbation (Burge et al., 2000), and mortality (Anthonisen, Wright, & Hodgkin, 1986) in COPD.

Severity	Postbronchodilator FEV1/FVC	FEV1 % predicted
Mild COPD	≤0.7	≥80
Moderate COPD	≤0.7	50-80
Severe COPD	≤0.7	30-50
Very severe COPD	≤0.7	<30

PFT alone does not explain the heterogeneous features of COPD. Therefore, the GOLD 2011 document proposed a new multidimensional grading system that assessed the respiratory and systemic expressions

of COPD and would better classify, predict outcome, and exacerbation risk in these patients (Vestbo, Hurd, & Rodriguez-Roisin, 2012).

BODE index:

The BODE index, a simple multidimensional 10-point scale in which higher scores indicate a higher risk of death, The BODE index is a multidimensional scale comprising the body-mass index (B), the degree of airflow obstruction (O), functional dyspnea (D), and exercise capacity (E) as assessed by the 6 minute walk test (6 MWT). The scale ranges from 0 to 10 points, with higher scores indicating a greater risk of death. It can be divided into four quartiles: those among patients with COPD.

1.6 CONVENTIONAL MANAGEMENT OF COPD

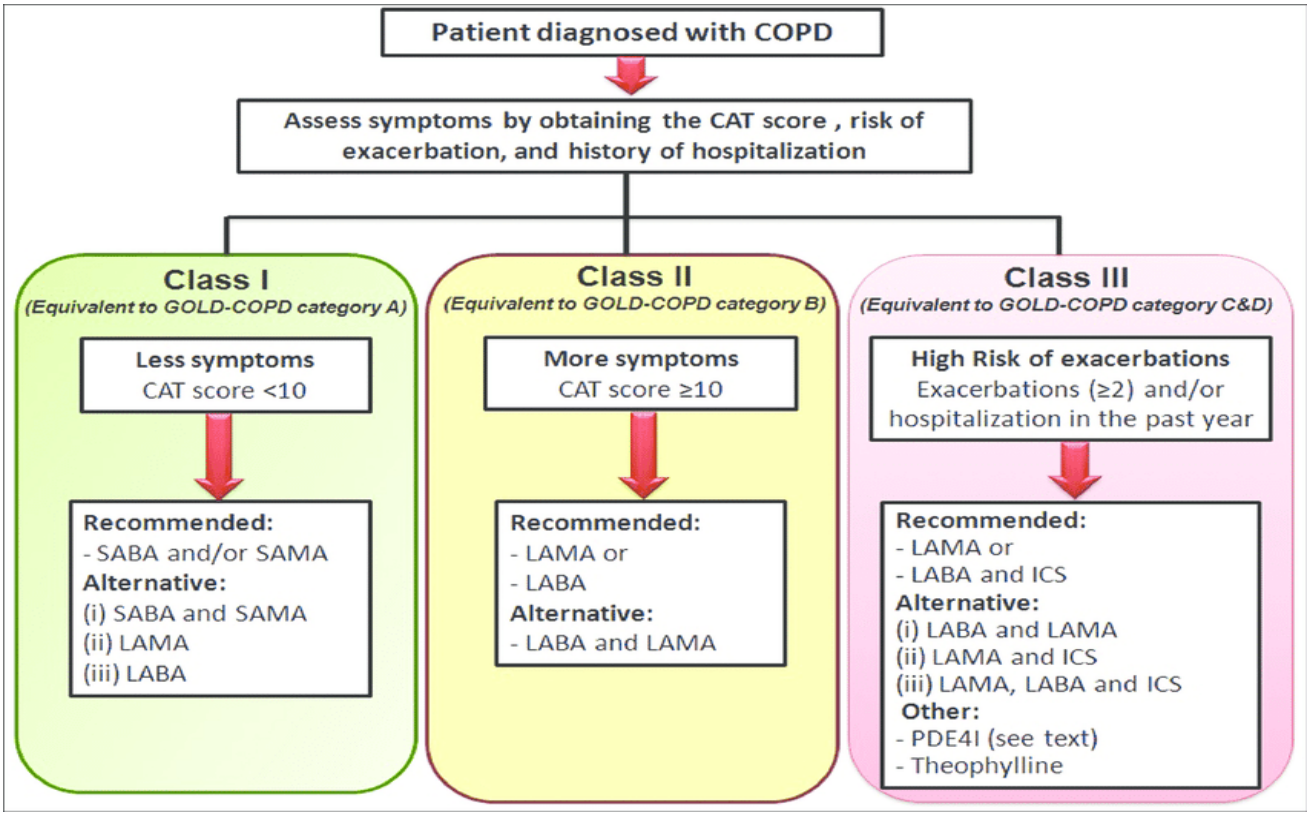
The conventional therapeutic strategy in the management of COPD is directed mostly at the management of the patient's presenting symptoms, such as breathlessness, fatigue, depression, anxiety, pain, and insomnia. COPD is incurable; oxygen therapy has been shown to increase survival in selected patients. Smoking cessation may slow the decline Medications have been shown to help stabilize the breathing passages and decrease swelling. These medications must be taken every day, probably for the rest of your event disease progression, to relieve symptoms, improve exercise tolerance, improve health status, prevent and treat complications and exacerbations, manage stable COPD, assess and monitor disease, trim down risk factors, reduce ability. A pulmonary rehabilitation program can be helpful in learning to use the lung power more efficiently.

1.7 TREATMENT:

- The primary goals of treatment are to control symptoms, improve the quality of life, and reduce exacerbations and mortality. The non-pharmacological approach includes smoking cessation and pulmonary rehabilitation.
- Annual influenza vaccination is recommended in all patients with COPD. Patients aged 65 and over should receive the 13-valent pneumococcal conjugate vaccine (PCV13) and the 23-valent pneumococcal polysaccharide vaccine (PPSV23) at least one year apart. The PPSV23 is recommended for those aged 64 and younger with significant comorbidities (e.g., diabetes mellitus, chronic heart disease, chronic lung disease). The classes of commonly used medications in COPD include bronchodilators (beta2-agonists, antimuscarinics, methylxanthines), inhaled corticosteroids (ICS), systemic glucocorticoids, phosphodiesterase-4 (PDE4) inhibitors, and antibiotics.
- Beta2-agonists work by relaxing the smooth muscle in the airways. SABAs and long-acting beta2-agonists (LABA) are commonly used in treatment. SABAs are used as needed to provide immediate relief. LABAs are typically used for maintenance therapy.
- Antimuscarinics work by blocking the M3 muscarinic receptors in the smooth muscle and therefore preventing bronchoconstriction. Short-acting antimuscarinic agents (SAMA) like SABAs provide a rapid onset of action and are used on an as-needed basis. Long-acting antimuscarinic agents (LAMA), like LABAs, are used as maintenance therapy.
- Methylxanthines are also used in maintenance therapy, usually after LABA or LAMA treatment as additional relief. Methylxanthines work by relaxing the smooth muscle in the airways, causing mild bronchodilation. The mechanism of action is unknown; however, it may be due to the

inhibition of phosphodiesterase (PDE) III and IV. Theophylline is a commonly used methylxanthine, and when used in combination with salmeterol, has shown to provide significantly greater improvement in FEV1 compared to salmeterol alone.

- Inhaled corticosteroids are often used in combination with LABAs and LAMAs to decrease inflammation. A combination of ICS and LABA has been shown to be more beneficial than either of the drugs when used alone. Physicians and patients should be aware of an increased risk of developing pneumonia when treated with an ICS. Oral glucocorticoids are not indicated for long-term use and can have multiple side effects. These should instead be reserved for the management of acute exacerbations.
- Phosphodiesterase-4 inhibitors work by inhibiting the breakdown of intracellular cyclic AMP and thus reducing inflammation. Roflumilast is a PDE4 inhibitor used for patients with severe disease and has been shown to decrease the number of exacerbations in this population.
- Recent studies have shown that the use of azithromycin may reduce the number of exacerbations in patients with COPD. Azithromycin is typically prescribed as 250 mg.
- Acute exacerbations of COPD can be managed in an outpatient or inpatient setting, depending on the severity. The assessment of severity is discussed in the previous section. Mild cases can be treated in the outpatient setting with bronchodilators, corticosteroids, and antibiotics. For moderate and severe cases, inpatient management is indicated. Hospitalized patients often require oxygen and bronchodilator therapy in the form of a SABA with or without a SAMA. Long-acting bronchodilators are typically used when the patient becomes stable and ready for discharge. Oral corticosteroids may be used; however, intravenous corticosteroids have not been shown to be more efficacious compared to oral and instead may cause increased side effects. Intravenous corticosteroids may be considered if patients have a contraindication to oral intake (e.g., aspiration risk or continuous BiPAP therapy). Antibiotics should be considered if there is suspicion for a bacterial infection. Oxygen therapy can range from a nasal cannula to mechanical ventilation, depending on the severity of the exacerbation.
- For long-term therapy, the choice of treatment varies and should be tailored to each patient. Management is largely based on the severity of the disease and symptoms as outlined by GOLD. for initial and commonly used medications in the treatment of COPD.
- Severe cases may require surgical intervention, including bullectomy, lung volume reduction surgery, or lung transplantation. Surgical intervention is indicated in severe conditions where symptoms are not controlled with medical therapy alone and may improve quality of life. Pulmonary rehabilitation is indicated in all stages of COPD. It is a comprehensive plan that is tailored to patients and may involve therapies such as exercise training, education, and behavioral changes. Its purpose is to improve a patient's physical function and psychological condition.



Air Quality Index (AQI) is a tool for effective communication of air quality status to people. AQI transforms

AQI Category	**AQI scale**	**Possible Health Impacts**
Good	0-50	Minimal impact
Satisfactory	51-100	Sensitive people: Minor breathing discomfort
Moderate	101-200	People with lungs, asthma and heart diseases: Breathing discomfort
Poor	201-300	Most people: Breathing discomfort on prolonged exposure

Systemic inflammation, COPD and Atherosclerosis (Cardiovascular Event)

Decreased pulmonary function is linked with high levels of systemic inflammatory markers, which may have important pathophysiological and therapeutic implications for stable COPD subjects. The mechanism of development of systemic inflammation in COPD subjects is not clear. Intense inflammatory processes in the airways, parenchyma, and pulmonary vasculature are reported to occur in COPD.

Peripheral lung inflammation is triggered by the inflammatory response to noxious particles or gases, mainly cigarette smoke, which cause airway and/or alveolar abnormalities. Thus, it, leads to COPD, which is manifested as irreversible persistent respiratory symptoms and airflow limitation.

Peripheral lung inflammation may cause a "spillover. "spill-over' pill-over" cytokines, such as interleukin (IL)-6, IL Ib, and tumor necrosis factor-alpha, into the systemic circulation, which may increase acute-phase proteins such as C-reactive protein. Systemic inflammation may initiate various co-comorbid

conditions, such as cardiovascular co-morbidities. Co-morbidities markedly affect health outcomes in COPD. Non-respiratory diseases were reported as one of the leading causes of mortality in COPD patients and COPD may increase death risk from other co-morbid conditions

Cardiovascular Risk Calculators:

Cardiovascular diseases (CVDs) include coronary death, myocardial infarction, coronary ischemic attack, peripheral arterial ischemic attack, peripheral arterial disease, and failure. It's incidence is rapidly increasing worldwide, and many of its risk factors are considered modifiable by specific preventive measures. In the INTER-HEART study, 9 modifiable factors accounted for over 90% of the population-attributable risk for the first myocardial infarction (MI) that include smoking, dyslipidemia (ApoB/ApoAI ratio), diabetes, hypertension, abdominal obesity (waist/hip ratio), dietary patterns (lack of daily consumption of fruits and vegetables), regular physical activity, regular alcohol consumption, and psychosocial factors (e.g., depression, perceived stress, and life events). Smoking and abnormal lipids were identified as the 2 most important risk factors, which together accounted for about two-thirds of the population attributable risks of an acute myocardial infarction. Considering these findings, aggressive risk factor modification can prevent CVD in a large proportion who are at risk of developing the disease

Lifetime risk: It is a measure of the absolute risk beyond IO years. It is recommended that lifetime risk should be estimated in young population between 20 and 59 years of age who are free from CVD and are not at high short-term

Framingham risk calculator (FRS), JBS-3, QRISK-2, Reynolds Risk Score and ASCVD are discussed in below

Framingham risk calculator:

It is based on the data derived from the Framingham Heart Study. The initial FRS was developed in 1998 and predicted only coronary heart disease (CHD) risk. Subsequently, a new general risk prediction tool was developed in 2008 to predict the overall CVD risk.

It has been validated in a number of populations,

Limitations: It does not take into account many of the nonconventional risk factors, e.g., obesity, physical activity, and family history of premature CVD, which are being increasingly recognized as important contributors to the development of atherosclerotic vascular disease. It also relies heavily on age as a determinant of CVD risk and underestimates CVD risk in young individuals, despite the presence of multiple other risk factors.

QRISK:

The QRISK calculator was developed to predict CVD risk in patients from different ethnic groups living in England and Wales. The updated version,

It's algorithm includes risk predictors used in the modified FRS model plus ethnicity, socio economic status, history and other medical variables, e.g. chronic renal disease, atrial fibrillation and rheumatoid arthritis

It is well known that atherosclerosis is underlying the most CVD, which is rarely the result of a single risk factor but more usually the end result of the combined effect of several risk factors (Coony et al. 2010). This is one of the key reasons for the use of CVD risk calculators. Also, a combination of several seemingly modest factors may result in a much higher total risk than a single, more impressively raised factor. Therefore, systems have been developed to help estimate total risk. The need to consider the likely impact of all CVD risk factors before making the clinical decision and to recommend a system of evaluating combined risk factor effects has been advocated.

Joint British Societies (JBS) risk score calculator:

It is based on the QRIS lifetime CV risk calculator and considered many of the same variables from the QRISK scores.

It is for use in British populations, but unlike most other risk scores, it includes data on Indians (under ethnicity) and allows separate risk evaluation for Indian ethnicity.

Reynolds Risk Score:

It is designed to predict the risk of having a future heart attack, stroke, or other major heart disease in the next IO years for a healthy person without diabetes.

In addition to age, blood pressure, cholesterol levels, and smoking status, this risk score uses information for I) a measure of inflammation a measure of genetic risk (whether or not either of parents had a heart attack before they reached age 60 years) **(Ridker et al. 2007, Bassuk, 2008).**

WHO/ISH RISK PREDICTION CHARTS

The risk factors behave differently for population groups based on the differences in their geographical location, resulting in significant variability in the absolute risk predicted by the major risk factors. Addressing the same, a series of risk prediction charts were published, where each chart is dedicated to a different ethnic-geographic region.

These prediction charts have been developed using a statistical modeling approach, and the absence of population-derived epidemiological data in many ethnic groups is the major limitation making it, such that obesity and family history of premature CVD are not included in these charts, which may lead to underestimation of CV risk.

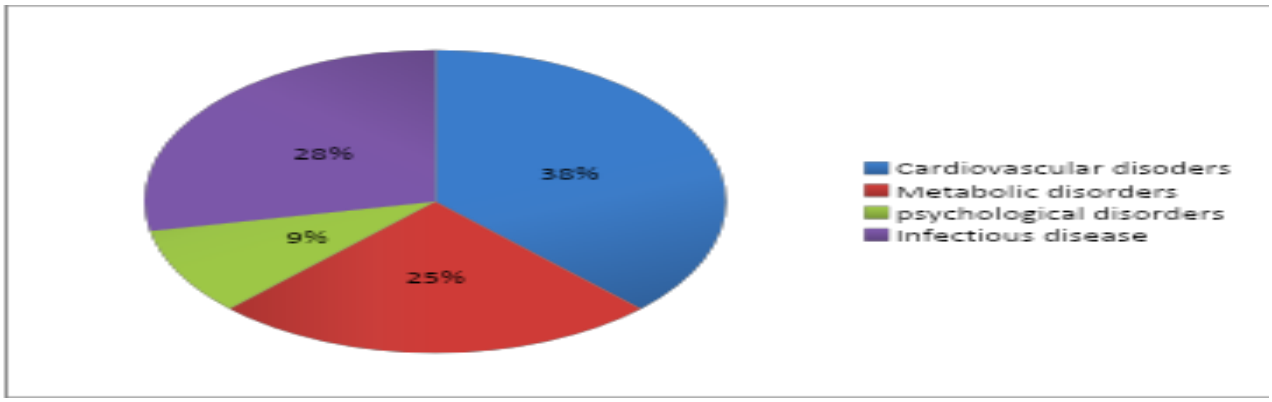
2. METHODOLOGY:

A prospective and observational study was conducted at an outpatient in the clinical setting of the MGM hospital Warangal to determine the cardiovascular risk rates for patients with COPD from the time of diagnosis and to the time of treatment in the association with the incidence of chronic pulmonary disorder.

3. RESULTS:

- A total of 60 physician-diagnosed COPD subjects and age-, gender-matched 55 healthy subjects were screened between the study periods to enroll 40 COPD cases and 40 healthy controls. A large number of follow-up cases of COPD subjects who were admitted in the hospital were excluded due to the presence of co-morbid conditions. Subjects meeting the initial screening criteria were further evaluated.
- Key cardiovascular comorbidities in COPD patients were hypertension, coronary artery disease, and stroke. Key metabolic disorders in COPD patients were diabetes, thyroid disease, and nutrition deficiencies such as anemia and vitamin D deficiency. Cases of liver disease, gallbladder disease, and kidney/prostate disease were the main inflammatory/chronic conditions in COPD patients. Key brain-nervous system disorders were Parkinson disease, neuropathy, schizophrenia, which were grouped together.
- The demographic characteristics, pulmonary function parameters of subjects in both groups (COPD, n = 40; healthy controls, n - 40), and COPD stages of the cases are outlined in Table 1

Age was statistically similar in both groups, with mean ages of 50.58 (9.89) years and 55.32 (12.40) years, respectively.



DEMOGRAPHY AND CLINICAL CHARACTERISTICS OF COPD AND CONTROL GROUPS

Table:1

PARAMETERS		CONTROL	COPD	P-VALUE
Age (years), mean + SD		50.58(9.89)	55.32(12.40)	**0.483**
Gender				
Male		30 (75%)	30 (75%)	
Female		10 (25%)	10 (25%)	
BMI(kg/m)mean+ SD		25.72 5.59	27.40 5.45	**0.189**
Smoking status, n (%)				
Current smoker		7(17.5)	19(47.5)	**< 0.001**
Ex-smoker		5(12.5)	8(20)	
Non-smoker		28(70)	13(32.5)	
Systolic BP (mm/hg), mean SD		126.21+2.89	130+2.362	**0.061**
Diastolic BP (mm/hg), mean SD		82.75+5.36	84.75 + 2.46	**0.258**
Fasting plasma glucose (mg/d1)		95.21 11.02	96.13 14.56	**0.885**
Pulmonary Function Test				
FVC (Liter)	IQR	0.90	0.73	**< 0.001**
	Quartile 1	2.66	1.44	
	Median	3.00	1.88	
	Quartile 3	3.51	2.17	
FEVi % predicted	IQR	17.00	18.48	**< 0.001**
	Quartile 1	85.75	33.53	
	Median	94.50	41.72	
	Quartile 3	104.5	52.01	
FEV,/FVC%	IQR	9.15	16.62	

predicted	mantle 1	79.13	48.89	**< 0.001**
	Median	84.65	56.52	
	Quartile 3	**88.78**	**65.5 I**	

Student t test; Chi-square test; " Mann-Whitney U test; $p < 0.05$; $p < 0.001$
Abbreviations: FEV: forced expiratory volume in one second.

Table 2 : Gold Stage and risk category of COPD Patients

	Stage/Risk Category	Number of subjects (%)
stages of COPD, *(%)*	Phase A	**11 (27.5)**
	Phase B	**9 (22.5)**
	Phase C	**7 (17.5)**
	Phase D	**13 (32.5)**
Risk category, n (%)	Lower risk	**16(40)**
	Higher risk	**24 (60)**

Lower risk: Stage A and Stage B; High risk: Stage C and Stage D

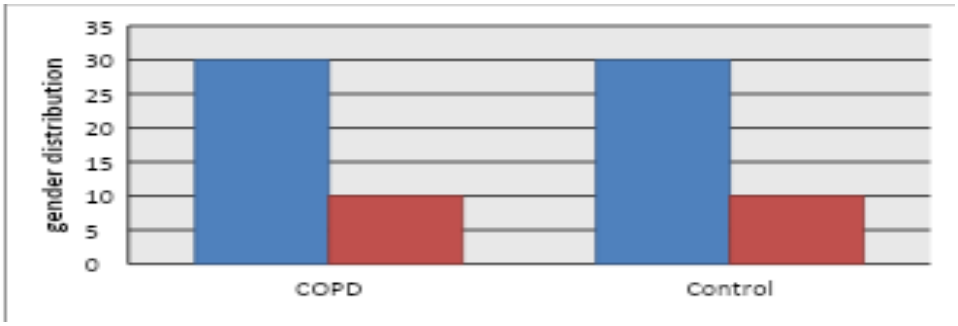


Figure 2: Gender Distribution
The gender distribution is similar in both COPD and healthy control groups.

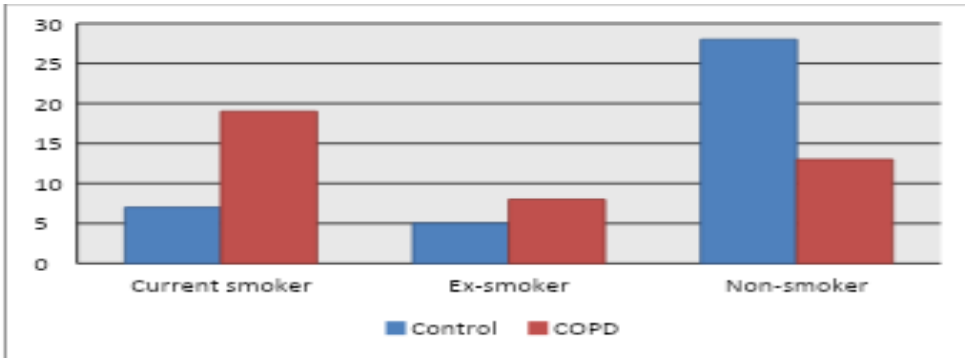


Figure 3: Smoking Status

There were more smokers and ex-smokers in COPD group vs.
Figure 4: Distribution of Forced Vital **Capacity** in **Study** Participants

Compared to healthy controls, COPD patients had a substantially lower FCC value.

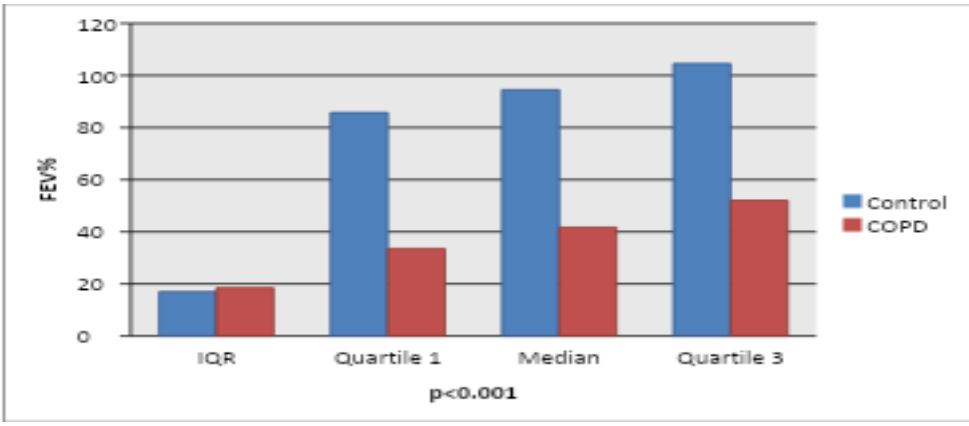


Figure 5 : Distribution of Percentage Predicted Forced Expiratory Volume in Study Participants FEV% predicted was significantly less in COPD patients than in healthy controls. Abbreviation : IQR- (interquartile range; FEV (%) predicted -Percentage Predicted Forced Expiratory Volume in 1 second;

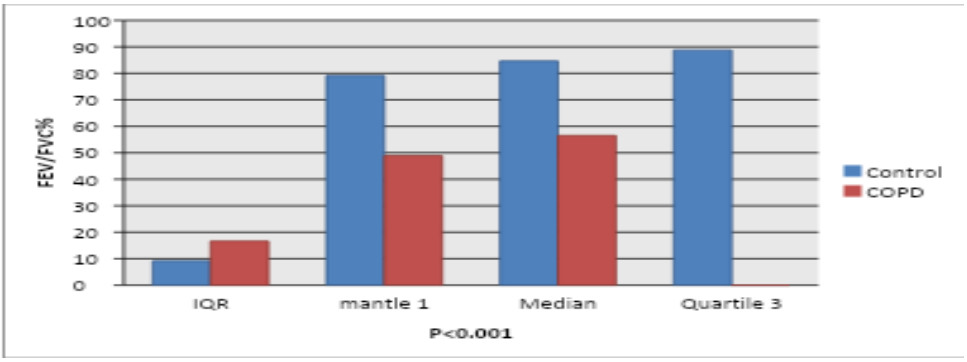


Figure 6 : Distribution of FEV/FVC% predicted in Study Participants

Abbreviation: IQR: inter quartile range; FEV: forced expiratory Expiratory Volume in 1 second; FVC: forced vital Vital Capacity

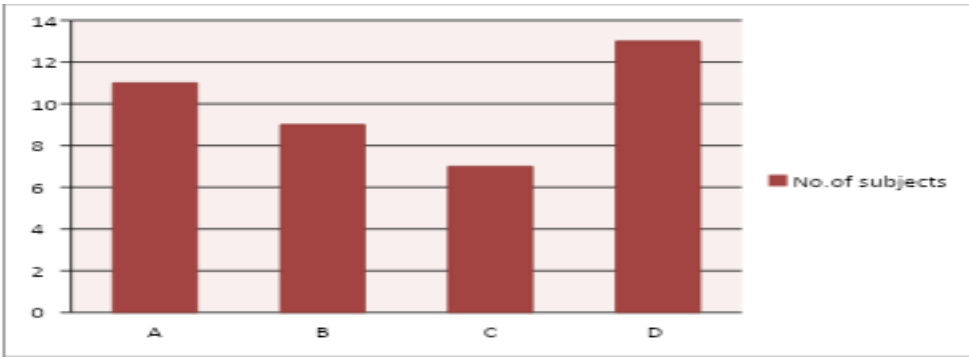


Figure 7: Severity of COPD

Majority of COPD patients were categorized under Stage D of GOLD Classification followed by Stage B and Stage C

Lower risk: Stage C and Stage B; High risk: Stage A and Stage D

LIPID PROFILE, ATHEROGENIC INDICES:

The lipid density lipoprotein cholesterol level was significantly higher in subjects with COPD than in control subjects (p = 0.OF 9).

Atherogenic indices, cardiogenic risk ratio (CRR) and atherogenic coefficient (AC), were significantly higher in subjects with COPD than in control subjects for both the parameters (p = 0.003). Atherogenic index of plasma (AIP) were numerically higher in COPD subjects versus controls but do not have any statically significance.

Parameters	Control	COPD	p-value
Total Cholesterol (mg/dL), mean SD	168.10 +35.41	171.81 + 19.81	**0.517**
HDL Cholesterol (mg/dL), mean USD	47.67+ 9.99	40.01+5.83	**< 0.001**
LDL Cholesterol (mg/dL), mean SD	98.35+22.43	101.42 + 18.76*	**0.019**
AIP, mean – SD	0.47 + 0.26	0.49 + 0.19	**0.696**
CRR, mean SD	3.86+1.07	4.64 + 1.22**	**0.003**
AC, mean SD	**2.86 + 1.07**	**3.64 + 1.22****	**0.003**

Table 3: Lipid Profile and Atherogenic Indices of COPD and Control Subjects: Student's t test; * p < 0.05; ** p 0.01; p <0.001; SD stands for standard deviation, and HDL-AC stands for atherogenic coefficient.

Table 4: Distribution of Triglycerides in study participants

The specifications		Control	COPD
Triglycerides (mg/dL)	IQR	125.25	37.75
	Quartile 1	88.50	98.00
	Median	128.50	108.50
	Quartile 3	213.75	135.75

Figure 8: Total cholesterol levels in study participants: Total cholesterol levels were higher in COPD subjects than in healthy control subjects.

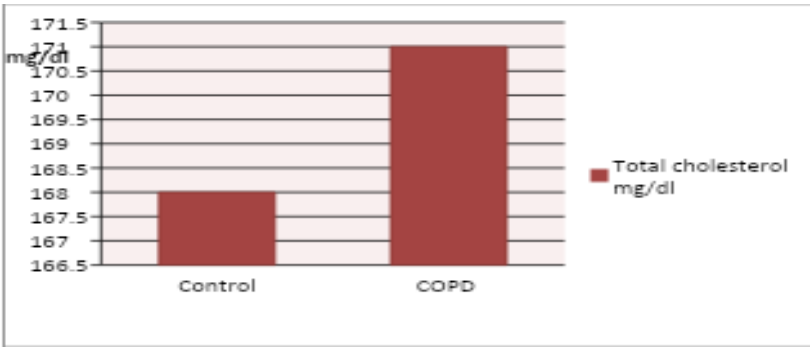
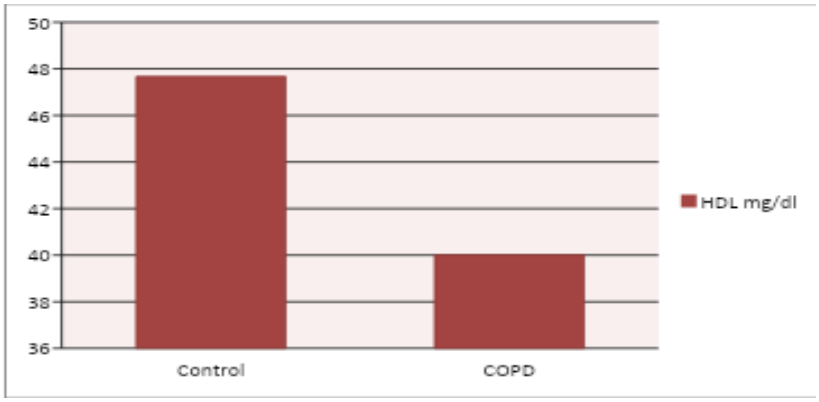


Figure 9: Subjects with COPD have lower high-density lipoproteins.



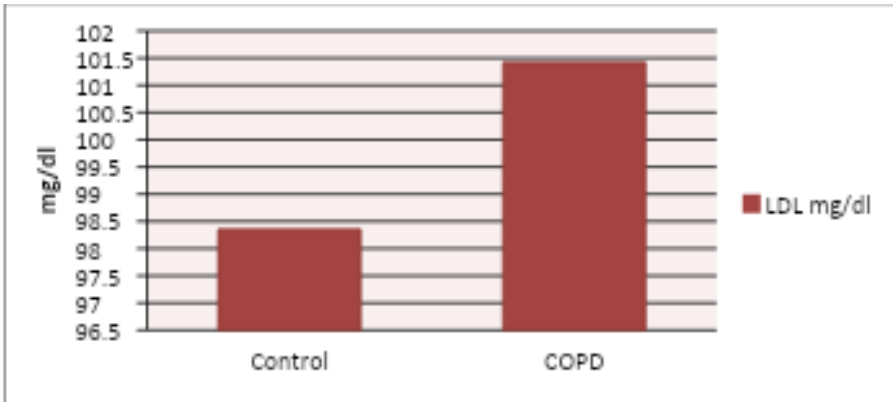


Figure 10: low-density lipoprotein cholesterol levels in study participants: Serum low-density lipoprotein cholesterol levels were significantly higher in subjects with COPD than the healthy control subjects

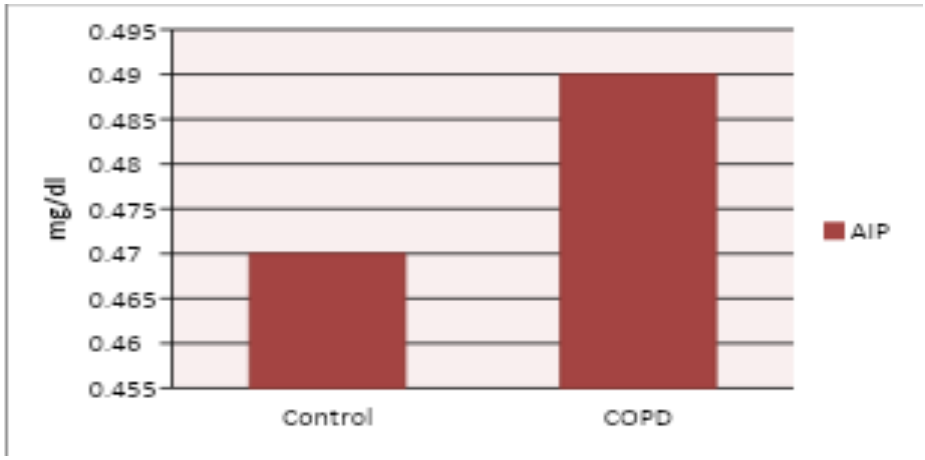


Figure 11 : atherogenic index of plasma levels in study participants
Atherogenic index of plasma was higher in subjects with COPD than in the healthy control subjects

Cardiovascular Risk Score Calculation:

5. Ten-year risk scores of the COPD subjects were calculated and presented in **Table** The FRS-CVD

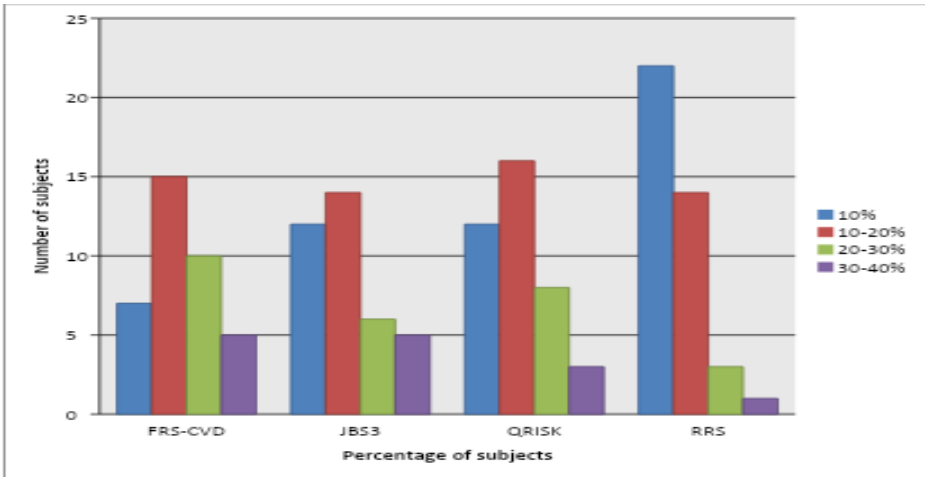
The calculator could identify the maximum number of subjects with high CVD risk (risk score * 20%), followed by JBS3 and QRISK 2 calculators. The RRS calculator has stratified more than 50% of the subjects to have a 10-year CVD risk to be less than 10%.

Table 5: Ten year Cardiovascular Risk Scores in COPD patients

Ten-year risk	FRS-CVD	JBS3	QRISK2	RRS
<I0%	7 (17.5%)	12 (30%)	12 (30%)	**22 (55%)**
10%—<20%	15 (37.5%)	14 (35%)	16 (40%)	**14 (35%)**
20%—<30%	10 (25%)	6 (15%)	8 (20%)	**3 (7.5%)**
30%—<40%	5 (12.5%)	5 (12.5%)	3 (7.5%)	**1 (2.5%)**
***40%**	**3 (7.5%)**	**3 (7.5%)**	**1 (2.5%)**	**0 (0.0%)**

Data presented as n (%)

The following are abbreviations: RRS: Reynolds risk score; FRSfiVD: Framingham risk score-cardiovascular disease; QRISK2: Q-risk 2 by the UK National Health Service; and JBS3: Joint British Society risk calculator 3: Ten-year Cardiovascular Risk Assessment Figure 12



Cardiovascular Risk Scores for Patients with COPD Over a 10-Year Period

Forty-five percent of patients with COPD had high CVD risk, according to the FRS-CVD calculator.

Abbreviations: JBS3: Joint British Society risk calculator 3; QRISK2: Q-risk 2 by UK National Health Service; RRS: Reynolds risk score; FRS-CVD: Framingham risk score-**6.**

Conclusion:

The study concluded that atherogenic indices levels in COPD subjects were significantly higher than in control subjects. There was a positive correlation between atherogenic indices, including atherogenic index of plasma, cardiogenic risk ratio, and atherogenic coefficient can be used as early predictors of CV risk in subjects with COPD. Framingham risk score-cardiovascular disease (FRS-CVD) can be used for assessing the cardiovascular risk in subjects.

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