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Spirometry Findings in Patients Undergoing Coronary Angiogram

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ABSTRACT:

Background: Coronary artery disease (CAD) is a leading cause of disability and death worldwide. This study was done to analyse spirometry findings in patients undergoing coronary angiogram (CAG) and study if there is any correlation between low FEV1, FVC and CAD.

Methods: 87 patients who satisfied inclusion criteria were included within the study,spirometry was done either before CAG or 6 weeks later in unstable angina, myocardial infarction. Obstructive Airway Disease (OAD) was diagnosed based on clinical history and spirometry. Results were analysed and mean % predicted FEV1, FVC and severity of CAD were compared among non-smokers and those with COPD.

Results: In our study, out of 87 patients, 18.4% (16) were COPD and 1.1% (1) was asthmatic. Out of 70 patients without OAD, 85.7% (N=60) of patients were non-smokers, and 14.3% (N=10) were smokers. Among 60 non-smokers, mean % predicted FEV1 with normal CAG was 98.01± 7.891, with SVD was 78.74 ±5.263, with DVD was 73.35 ±2.203 and 69.39±7.898 in patients with TVD. Mean % predicted FVC with normal CAG was 96.38±6.929, with SVD was 78.54±6.025, with DVD was 75.64 ±2.996 and 71.25±6.162 in patients with TVD. With the severity of CAD, there is greater decline in mean % predicted FEV1 and mean % predicted FVC. By chi-square test p value is <0.005 which is clinically significant.

Conclusions: The patients with CAD had lower pulmonary function depending on the severity of CAD, independent of smoking status and respiratory comorbidities. Hence spirometry can be considered as one of the screening tool for risk assessment of CAD.

Key words: Coronary artery disease, coronary angiogram, FEV1 (Forced Expiratory Volume1), FVC (Forced Vital Capacity), spirometry

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Abbreviations: CAD: Coronary artery disease, FEV1: Forced Expiratory Volume1, FVC: Forced Vital Capacity, CAG: coronary angiogram, OAD: Obstructive Airway Disease, SVD: Single Vessel Disease, DVD: Double Vessel Disease, TVD: Triple Vessel Disease, COPD: Chronic Obstructive Pulmonary Disease.

Introduction

Coronary artery disease (CAD) is a leading cause of disability and death worldwide, and is one of the top five causes of death in Indian population[1]. Current evidence suggests that inflammation plays a significant role in CAD and it is a major driving force underlying the initiation of coronary plaques, their unstable progression, and eventual disruption, patients with a more pronounced vascular inflammatory response have a worse outcome[2].

Apart from the conventional risk factors, there is now compelling evidence to support inflammation as a significant risk factor [3]. Increased levels of fibrinogen, C-reactive protein, and other inflammatory markers have been related to poor lung function [4, 5, 6].

Reduced pulmonary function, as assessed by forced expiratory volume 1(FEV1) and forced vital capacity (FVC), is associated with increased incidences of cardiovascular disease and death. The cause for this association has beenestablished in both smokers and non-smokers. In several prospective studies, inverse relationships between lung function and the incidence of CAD have been reported [7, 8, 9, 10]. When other cardiovascular risk factors were taken into account, these associations remained significant, and the findings were comparable in studies of lifelong non-smokers[11].

COPD (Chronic Obstructive Pulmonary Disease) is a leading cause of mortality and morbidity worldwide[12]. COPD and cardiac comorbidities are frequently associated. CAD is more common among COPD. The incidence of COPD is higher in CAD than in the general population and often under diagnosed. Patients with comorbid COPD and cardiovascular disease have a greater risk of morbidity, which includes lower quality of life, dyspnoea, and exercise tolerance[13], as well as a higher risk hospitalisation [14]. All CAD patients, particularly those with serious cardiac dysfunction, history of smoking, and the elderly, should be screened for COPD on a regular basis. Only a comprehensive approach of treatment will have an effective outcome.

This study was done to analyse the correlation between pulmonary function and CAD.

Methods

The study was conducted in the Department of Pulmonary Medicine, Bagman Manager Jain Hospital, Bangalore. It was a cross-sectional study conducted from July 2015 to June 2016. Around 87 patients undergoing coronary angiogram were included in the study.

Exclusion criteria:

- Previous cardiac surgery
- Unstable angina
- Recent MI (1 month)
- Recent stroke, eye surgery, thoracic/abdominal surgery
- Hemoptysis
- Known thoracic, aortic or cerebral aneurysm
- Recent pneumothorax
- Uncontrolled hypertension

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- Pulmonary Embolism
- Patient's refusal

Patients fulfilling the inclusion and exclusion criteria were informed about the study and informed consent was taken. Each patient recruited was subjected to a questionnaire of thorough medical history and detailed history of associated comorbidities and smoking status was taken. A detailed clinical examination was done. Patients fulfilling inclusion criteria were offered spirometry. Patients with unstable angina and MI were followed up and spirometry was done after 6 weeks. Spirometry was done using JAEGER SPIROMETER.

Spirometric pulmonary function test:

Pulmonary function test was done using a computerised spirometer using JAEGER Lab Manager V5 30.0 software. The patient's information was entered. The patient was made to sit comfortably and the procedure was explained to them in their preferred language. At least three manoeuvres meeting the reproducibility and acceptability criteria (as per the ATS/ERS guidelines) were noted. The following parameters were measured

1. Forced expiratory volume in first second (FEV1)
2. Forced vital capacity (FVC)
3. FEV1/FVC ratio (FEV1/ FVC)

These measured pulmonary function values were presented as a percentage from the predictive values, which were computed according to the patient's age, sex and height.

CAG reports were collected.

Data was entered in Microsoft excel and analysed using SPSS (Statistical Package for Social Science, Ver.10.0.5) package. CAG and spirometry findings were analysed using chi square and ANOVA tests.

Results

The study included 87 patients who met the inclusion criteria. Spirometry was done either before CAG or 6 weeks later in unstable angina, MI. Obstructive airway disease (Asthma and COPD) was diagnosed based on clinical history and spirometry. Results were analysed separately in individuals with COPD and without OAD.

Analysis of data of patients without obstructive airway disease

Out of 70 patients without obstructive pulmonary disease, 39 patients were male which comprised 55.7% and 31 were females which comprised 44.3% of the group. 48.6% of patients were in the age group of 51-60, 31.4% of patients in 61-70 and 14.3% of patients were 41-50 and 5.7% of patients were 71-80Yrs and Mean age was 58.8±7.165. 85.7% (N= 60) of patients in this analysis group were non-smokers, and 14.3% (N=10) were smokers. Out of 70 patients without obstructive airway disease, about 34.3% (24) of patients had normal CAG, 25.7% had SVD, 21.4% had DVD and 18.6% had TVD.

In a group of 60 non-smokers, mean % predicted FEV1 with normal CAG was 98.01± 7.891, in patients with SVD was 78.74 ±5.263, in patients with DVD was 73.35 ±2.203 and 69.39±7.898 in patients with TVD. With the severity of CAD, there is greater fall in Mean % predicted FEV1.

In a group of 60 non-smokers, mean % predicted FVC with normal CAG was 96.38±6.929, in patients with SVD was 78.54±6.025, and in patients with DVD was 75.64 ±2.996 and

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71.25±6.162 in patients with TVD. With the severity of CAD, there is greater fall in Mean % predicted FVC.
Among non-smokers, individuals in the lowest FEV1 quartile had greater severity of CAD compared with those in the highest FEV1 quartile. Individuals in the lowest FVC quartile had greater severity of CAD compared with those in the highest FVC quartile.

Table 1: Comparison of Mean % Predicted FEV1 and FVC and severity of Coronary Artery Disease, among non-smokerswithout Obstructive Airway Disease.

	CAG	**N**	**Mean**	**SD**	**Min.**	**Max.**	**F value**	**P value***
% Predicted FEV1	Normal	19	98.01	7.891	86.75	111.10	64.812	***<0.001***
	SVD	16	78.74	5.263	71.20	92.20		
	DVD	13	73.35	2.203	68.50	76.61		
	TVD	12	69.39	7.898	58.70	86.29		
	Total	60	81.80	13.148	58.70	111.10		
% Predicted FVC	Normal	19	96.38	6.929	84.19	106.07	58.223	***<0.001***
	SVD	16	78.54	6.025	71.01	94.40		
	DVD	13	75.64	2.996	71.08	82.30		
	TVD	12	71.25	6.162	58.60	81.00		
	Total	60	82.10	11.621	58.60	106.07		

Analysis of data in patients with COPD:
Mean % Predicted FEV1, in COPD (N=16) patients was 66.45±5.494 and in patients without Obstructive airway disease was 82.03±12.667. Patients with COPD and CAD had greater fall in FEV1 than in patients with CAD without obstructive airway disease.
Mean % Predicted FVC, in COPD (N=16) patients was 77.48±7.755 and in patients without Obstructive airway disease was 82.15±11.498. There was no significant difference in % Predicted FVC among patients with COPD and without any obstructive airway disease.

Table 2: Comparison of Mean % predicted FEV1 in patients without Obstructive Airway Disease and with COPD.

		N	**Mean**	**SD**	**Min.**	**Max.**	**F value**	**P value***
% Predicted FEV1	COPD	16	66.45	5.494	56.81	76.24	22.304	***<0.001***
	Without Obstructive airway disease	70	82.03	12.667	58.70	111.10		

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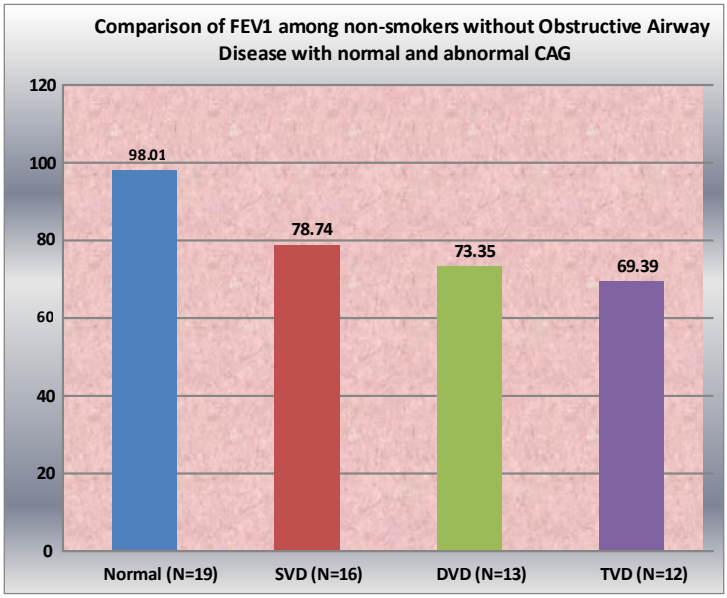

Figure 1: Comparison of FEV1 among non-smokers without Obstructive Airway
(FEV1: Forced Expiratory Volume1, SVD: Single Vessel Disease, DVD: Double Vessel Disease, TVD: Triple Vessel Disease, CAG: Coronary Angiogram)

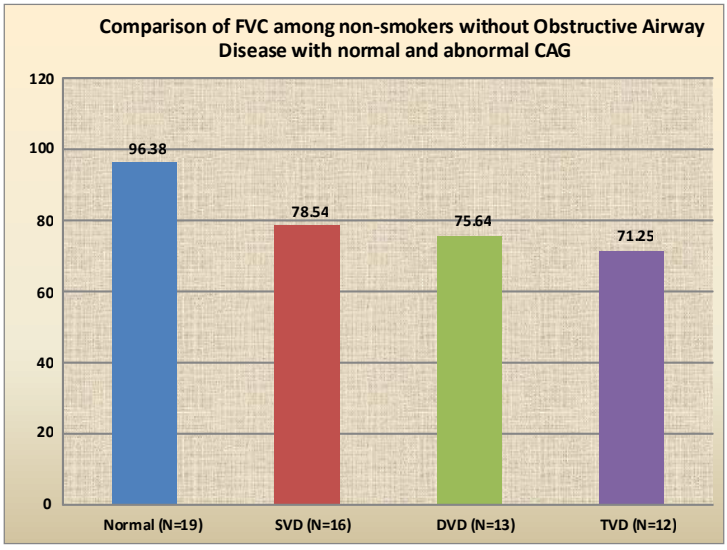

Figure 2: Comparison of FVC among non-smokers without Obstructive Airway
(FEV1: Forced Expiratory Volume1, SVD: Single Vessel Disease, DVD: Double Vessel Disease, TVD: Triple Vessel Disease, CAG: Coronary Angiogram)

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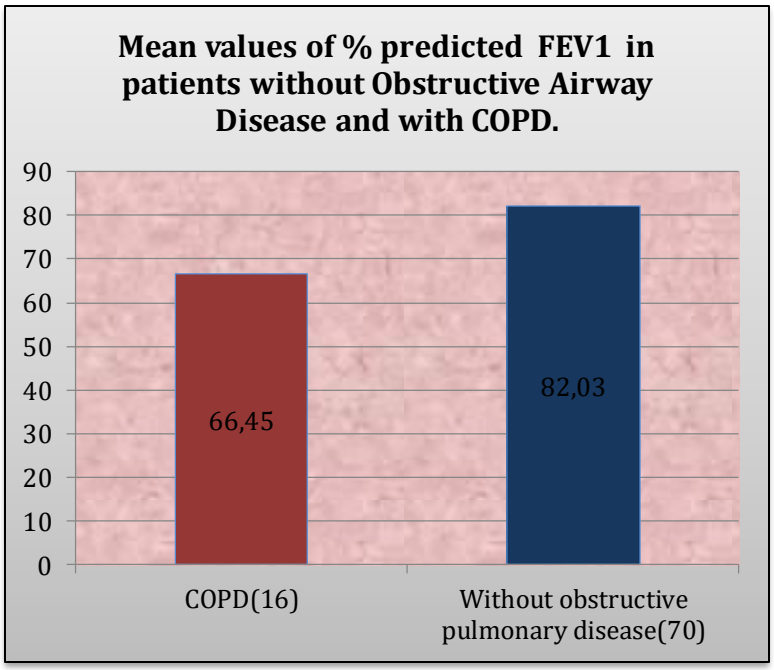

Figure 3: Mean values of % predicted FEV1 in patients without Obstructive Airway Disease and with COPD.
(FEV1: Forced Expiratory Volume1, COPD: Chronic Obstructive Pulmonary Disease)

Discussion

Prevalence of coronary artery disease is on the rise in both developing and developed countries. Inflammation plays a significant role in CAD. Increased levels of inflammatory markers have been associated with decreased pulmonary function. Studies have looked at the role of lung function in the risk of CAD morbidity and mortality in addition to conventional risk factors like smoking, hypertension, and diabetes mellitus.

The correlation between lung function, as measured by forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC), and the 10-year incidence of coronary artery disease was investigated by Schroeder EB et al. [8] in the Atherosclerosis Risk in Communities Research, which included 14,480 participants (1987-1998). In both the full cohort and never smokers, they discovered a strong association between lung function and the incidence of CAD in women, but a weaker association in men.

The similar results were found in our study, it was found that there was an impaired spirometry finding in individuals with CAD and there was significant correlation between FEV1, FVC and severity of CAD independent of smoking status. % predicted FEV1 and FVC were categorised into 4 groups. % predicted FEV1 and FVC and severity of CAD were compared in each group. Among non-smokersquartile group, individuals in the lowest quartile had greater severity of CAD compared with those in the highest quartile.

There are many studies that have looked into the correlation between poor lung function and cardiovascular mortality independent of smoking status. Don et al. [11] conducted a study to determine the relationship between reduced FEV1 and cardiovascular mortality, independent of smoking. The risk of cardiovascular mortality was compared across FEV1 quintiles. The FEV1quintile with the lowest FEV1 had the highest risk of cardiovascular mortality. Study

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concluded that low FEV1 is a marker for cardiovascular mortality independent of smoking history, age, gender.

A study of lifelong non-smokers by Hole DJ et al. [15] found inverse relationships between FEV1 and mortality from all causes, ischaemic heart disease, stroke, respiratory disease, and other causes of death.

In a community health research cohort, Gerard Ryan et al. [16] explored the relationship between FEV1 decline and mortality. This study added to the growing body of evidence that there is a correlation between FEV1 decline and mortality, regardless of cardiovascular disease risk factors, cigarette smoking, or average FEV1.

Decline in lung function is a predictor of mortality [17, 18].

The research by STAVEM et al. [19] contributes to our understanding regarding the association between level of lung function and all-cause and cardiovascular mortality by documenting that this relationship cannot be explained solely by tobacco exposure and poor physical fitness.

Our study concluded that there is association between poor lung function (FEV1 and FVC) and CAD independent of smoking status. Reduced lung function independent of smoking is a significant risk CAD. The severity of decrease in FEV1 and FVC correlated with severity of CAD.

Conclusion

Reduced pulmonary function is associated with increased risk of cardiovascular disease and severity and this association has been established in both smokers and non-smokers. FEV1, which is easily measured in ambulatory clinic settings, offers additional prognostic information, which may help to better risk-stratify patients and populations for potential cardiovascular events. Spirometry is a non-invasive, simple, cost effective test and has to be recommended in routine health check-up and to identify people at risk of coronary artery disease (CAD). The sample size was limited in this analysis, which resulted in a lack of correlation between certain variables.Furthermore, larger studies are recommended to confirm the correlation between poor pulmonary function and risk for CAD, as well as the risk attributed to each other. The importance of spirometry in non-pulmonary disease should be emphasised and implemented.

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REFERENCES

1. Gupta R, Guptha S, Sharma KK, Gupta A, Deedwania P. Regional variations in cardiovascular risk factors in India: India heart watch. World J Cardiol. 2012 Apr 26; 4(4):112-20.
2. Zakynthinos E, Pappa N. Inflammatory biomarkers in coronary artery disease. J Cardiol. 2009 Jun; 53(3):317-33.
3. Biasillo G, Leo M, Della Bona R, Biasucci LM. Inflammatory biomarkers and coronary heart disease: from bench to bedside and back. Intern Emerg Med. 2010 Jun; 5(3):225-33.
4. Dahl M, Tybjaerg-Hansen A, Vestbo J, Lange P, Nordestgaard BG. Elevated plasma fibrinogen associated with reduced pulmonary function and increased risk of chronic obstructive pulmonary disease. Am J RespirCrit Care Med. 2001 Sep 15; 164(6):1008-11.
5. Mendall MA, Strachan DP, Butland BK, Ballam L, Morris J, Sweetnam PM, et al. C-reactive protein: relation to total mortality, cardiovascular mortality and cardiovascular risk factors in men. Eur Heart J. 2000 Oct; 21(19):1584-90.

ISSN:0975-3583,0976-2833 VOL12,ISSUE04,2021

6. James AL, Knuiman MW, Divitini ML, Musk AW, Ryan G, Bartholomew HC. Associations between white blood cell count, lung function, respiratory illness and mortality: the Busselton Health Study. EurRespir J. 1999 May; 13(5):1115-9.
7. Lange P, Nyboe J, Appleyard M, Jensen G, Schnohr P. Spirometric findings and mortality in never-smokers. J ClinEpidemiol. 1990; 43(9):867-73.
8. Schroeder EB, Welch VL, Couper D, Nieto FJ, Liao D, Rosamond WD, Heiss G. Lung function and incident coronary heart disease: the Atherosclerosis Risk in Communities Study. Am J Epidemiol. 2003 Dec 15; 158(12):1171-81.
9. Engström G, Lind P, Hedblad B, Wollmer P, Stavenow L, Janzon L, et al. Lung function and cardiovascular risk: relationship with inflammation-sensitive plasma proteins. Circulation. 2002 Nov 12; 106(20):2555-60.
10. Kannel WB, Hubert H, Lew EA. Vital capacity as a predictor of cardiovascular disease: the Framingham study. Am Heart J. 1983 Feb; 105(2):311-5.
11. Sin DD, Wu L, Man SF. The relationship between reduced lung function and cardiovascular mortality: a population-based study and a systematic review of the literature. Chest. 2005 Jun; 127(6):1952-9.
12. Global initiative of obstructive pulmonary disease, 2021 update.
13. Miller J, Edwards LD, Agustí A, Bakke P, Calverley PM, Celli B, et al. Evaluation of COPD Longitudinally to Identify Predictive Surrogate Endpoints (ECLIPSE) Investigators. Comorbidity, systemic inflammation and outcomes in the ECLIPSE cohort. Respir Med. 2013 Sep; 107(9):1376-84.
14. Mannino DM, Thorn D, Swensen A, Holguin F. Prevalence and outcomes of diabetes, hypertension and cardiovascular disease in COPD. EurRespir J. 2008 Oct; 32(4):962-9.
15. Hole DJ, Watt GC, Davey-Smith G, Hart CL, Gillis CR, Hawthorne VM. Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. BMJ. 1996 Sep 21; 313(7059):711-5; discussion 715-6.
16. Ryan G, Knuiman MW, Divitini ML, James A, Musk AW, Bartholomew HC. Decline in lung function and mortality: the Busselton Health Study. J Epidemiol Community Health. 1999 Apr; 53(4):230-4.
17. Tockman MS, Pearson JD, Fleg JL, Metter EJ, Kao SY, Rampal KG, Cruise LJ, et al. Rapid decline in FEV1. A new risk factor for coronary heart disease mortality. Am J RespirCrit Care Med. 1995 Feb; 151(2 Pt 1):390-8.
18. Sircar K, Hnizdo E, Petsonk E, Attfield M. Decline in lung function and mortality: implications for medical monitoring. Occup Environ Med. 2007 Jul; 64(7):461-6.
19. Stavem K, Aaser E, Sandvik L, Bjørnholt JV, Erikssen G, Thaulow E, et al. Lung function, smoking and mortality in a 26-year follow-up of healthy middle-aged males. EurRespir J. 2005 Apr; 25(4):618-25.