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A COMPARITIVE STUDY OF PRE AND POST TREATMENT ECG AND ECHO FINDINGS IN ACUTE EXACERBATION OF COPD PATIENTS

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease characterized by progressive airflow limitation. COPD is associated with significant comorbidities like cardiac disease which is common. COPD patients experience frequent exacerbations which mitigate change in existing management. Pulmonary artery hypertension is common in COPD. This study was conducted to assess the changes in Electrocardiogram (ECG) and Echocardiogram pre and post exacerbation of COPD. COPD males with age more than 40 years without significant comorbidities who were admitted due to exacerbation were included in the study. ECG and Echocardiogram was performed at the baseline and on one month of follow up. Spirometry was performed at the baseline when patients were stabilized. 108 patients were included in this study. Majority of our patients were of grade III (severe) (46.3%) and grade IV COPD (very severe) (38.8%) classes based on post bronchodilator FEV1% on spirometry as per GOLD criteria. We could not find any significant changes in ECG, pre and post exacerbation of COPD, except normalization of sinus tachycardia. At baseline, mild PAH was observed in 88 patients(81.48%) with mean RVSP=38.59mmHg,moderate PAH was observed in 17 patients(15.74%) with mean RVSP=52.47 mmHg and severe PAH was observed in 3 patients (2.77%) with mean RVSP=70.33mmHg. While correlating spirometry with RVSP at the baseline, mild PAH was common in grade III (51.13%), whereas severe PAH were common in grade III and grade IV COPD (64.7 & 100% respectively). We found statistically significant decrease in right ventricular systolic pressure (RVSP) following treatment of exacerbation of COPD. Echocardiography screening of COPD patients is beneficial to assess the pulmonary artery pressure and identify patients with severe pulmonary artery hypertension, who may benefit with additional medications including phosphodiesterase inhibitors for those with severe PAH.

KEYWORDS: Pulmonary artery hypertension , echocardiography.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) refers to chronic bronchitis and emphysema, a pair of two commonly co existing diseases of the lungs in which the airways becomes narrowed. Global Initiative for Obstructive Lung Diseases (GOLD) defined COPD as a preventable and treatable disease. COPD is associated with significant extra pulmonary effects and is a common entity in clinical practice.

COPD is a leading cause of death and disability worldwide. According to World Bank data it is expected to move from its position in the year 2000 as the 4th and 12th most frequent cause of mortality and morbidity, respectively, to the 3rd and 5th leading cause of mortality and morbidity, respectively, in 2020.[1,2]

COPD exacerbation is defined as an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough and/or sputum that is beyond normal day-to-day variations, is acute in onset and may warrant a change in regular medication in a patient with underlying COPD.[3]

COPD is associated with significant extrapulmonary (systemic) effects among which cardiac manifestations are most common. Cardiovascular disease accounts for approximately 50% of all hospitalization and nearly one third of all deaths, usually when forced expiratory volume in one second (FEV 1) < 50% of predicted.[4] In more advanced disease cardiovascular disease account for 20 to 25% of all deaths in COPD.[5] COPD affects pulmonary blood vessels, right ventricle, as well as left ventricle leading to development of pulmonary hypertension, corpulmonale, right and left ventricular dysfunction.

In COPD, changes in the cardiac activity is reflected in the electrocardiography as change in P-wave axis and amplitude, rightward displacement of QRS and T-axis, reduction of amplitude of QRS complex in limb and

precordial leads, sinus tachycardia, Right bundle branch block (RBBB) and atrial fibrillation.[6,7,8,9,10]

Echocardiography provides a rapid, noninvasive portable and relatively accurate method to evaluate the right ventricle function, right ventricular filling pressure, tricuspid regurgitation, left ventricular function and valvular function.[11] Many studies have confirmed that echocardiographically derived estimate of pulmonary arterial pressure co-relate closely with pressures measured by right heart catheter ($r > 0.7$).[12,13]

The present study was aimed at ascertaining changes in pulmonary artery hypertension before and after treatment of acute exacerbation of COPD with bronchodilators, using ECG and Echocardiography.

MATERIALS AND METHODS

Subjects

A prospective study was done among COPD patients who were admitted in the hospital with exacerbation. Only male patients aged more than 40 years with tricuspid regurgitation on echocardiography at the time of admission were included. Patients with coronary artery disease, congenital heart disease, valvular heart diseases, systemic hypertension, old pulmonary tuberculosis, interstitial lung disease, diabetes mellitus, psychiatric illnesses and patients with persistent electrolyte derangements were excluded from the study.

The study was approved by institutional ethics committee. COPD patients meeting the inclusion criteria were selected. After obtaining the informed consent, clinical details and laboratory values were entered in data chart. All selected patients were subjected to routine blood investigations and electrocardiography within 24 hours of admission. All patients received standard treatment of nebulised and systemic bronchodilators, antibiotic and oxygen based on requirement.

Spirometry

Once stable clinically, the patients were subjected to spirometry to confirm the diagnosis and classified according to GOLD guidelines (post bronchodilator FEV 1 /forced vital capacity (FVC) ratio < 70% predicted) as GRADE I-mild COPD (FEV 1 ≥ 80% of predicted), GRADE II- moderate COPD (50% ≤ FEV 1< 80% predicted), GRADE III-severe COPD (30% ≤ FEV 1< 50% predicted) and GRADE IV-very severe COPD (FEV 1< 30% predicted).

Cardiac evaluation

Within 24-48 hours of admission resting two-dimension(2D) transthoracic Doppler echocardiography was done by cardiologist and valvular anatomy and function, left and right chamber size and cardiac function were assessed. Tricuspid regurgitant flow was identified by colour flow Doppler technique and the maximum jet velocity was measured by continuous wave Doppler. Right ventricular systolic pressure was estimated based on the modified Bernoulli equation and is considered to be equal to the systolic pulmonary artery pressure (sPAP) in the absence of right ventricular outflow obstruction: sPAP (mmHg) = right ventricular systolic pressure (RVSP) = trans-tricuspid pressure gradient (TTPG) + right atrial pressure (RAP), where trans-tricuspid gradient is $4V^2$ (V = peak velocity of tricuspid regurgitation, m/s).[5,12,13] RAP was estimated to be 5, 10, or 15 mmHg based on the variation in the size of inferior vena cava with inspiration, complete collapse, RAP = 5 mmHg; partial collapse, RAP = 10 mmHg; and no collapse, RAP = 15 mmHg.[14]

In the present study pulmonary artery hypertension (PAH) was defined as Resting PAP ≥ 30 mmHg.[15] PAH was further classified into classified into mild, moderate and severe category as resting PAP 30-50, 50-70, >70 mmHg, respectively.[16] Pulmonary artery pressure is measured by assessing the right ventricular systolic pressure (RVSP) which is determined by adding trans-tricuspid pressure gradient (TTPG) and right atrial pressure (RAP) (RVSP=TTPG + RAP), where trans-tricuspid gradient is $4V^2$ (V = peak velocity of tricuspid regurgitation, m/s).[4,12,13] In case of weak Tricuspid regurgitation signals, agitated saline bubble contrast was used for augmentation of signals and assessment of PAH. Right ventricle dimension was measured by M-Mode echo and right ventricular hypertrophy or corpulmonale was said to be present when right ventricular free wall thickness exceeded 0.4 cm. Right ventricle contractility was measured using Tricuspid annular plane systolic excursion (TAPSE) and right ventricular systolic dysfunction was said to be present when TAPSE was less than 1.8cm.[17] Left ventricular systolic function was said to be normal, when left ventricle ejection Fraction was more than 50%.

All Patients enrolled in the study were managed according toGOLD guidelines and pulmonary artery hypertension treated as per cardiologist advice. Hence patients of COPD with mild pulmonary artery hypertension received only inhaled bronchodilators along with short term xanthine derivative(oral doxophylline). COPD patients with moderate PAH and pedal edema received short term diuretics along with inhaled bronchodilators along with short term xanthine derivative (oral doxophylline). COPD patients with severe PAH and received vasodilators (selective phosphodiesterase inhibitors) along with short term diuretics, inhaled bronchodilators and short term xanthine derivative (oral doxophylline).

2 D echocardiography and electrocardiogram were done in all patients after 1 month to reassess the cardiac status and pulmonary artery pressure. Pretreatment and post treatment quality of life and symptoms scoring was done using a COPD assessment test (CAT scoring) questionnaire for each patient.[18]

Data analysis was done using SPSS version 20 and statistical method used for determining significance was paired T test and 95% confidence interval was calculated using chi square test.

RESULTS AND DISCUSSION

In our study we included male patients with age more than 40years. Most of the patients were within the age group 61 – 70 years (49.07%). 108 patients were included in this study. Mean age of the study population was 66.78 (SD 7.589) (Table 1).

Spirometry

Majority of patients were of grade III COPD(severe) which included 46.3% (50 patients) and grade IV COPD (very severe) which included 38.8%(42 patients) severity of air flow limitation based on post bronchodilator FEV1% on spirometry as per GOLD criteria. (Fig.1).

Electrocardiography findings

Among 108 COPD patients admitted with exacerbation, maximum i.e. 68 (62.96%) patients were having sinus tachycardia (rate more than 100), while 20 (18.51%) were having heart rate less than 100. The mean heart rate recorded among COPD patients was 106.14 per minute, followed by 19(17.59%) were having peaked p wave (p wave amplitude > 2.5mm) with right axis deviation and one patient (0.9%) with atrial fibrillation with irregularly irregular rhythm with loss of p wave. Duration of QRS and ST segment were normal in these patients (Fig. 2).

During follow up post treatment there were no ECG changes compared to ECG taken during exacerbation status except sinus tachycardia has been normalized in 50 patients (73.5%) among 68 patients of sinus tachycardia of heart rate more than 100 (Fig. 3).

Pulmonary artery hypertension

In our study, patients were further classified into mild, moderate and severe pulmonary artery hypertension category based on resting PAP 30-50, 50-70, >70 mmHg, respectively[10], which was measured by 2D resting echocardiography by estimating the right ventricular systolic pressure (RVSP) which in turn is determined by the sum of trans-tricuspid pressure gradient (TTPG) and right atrial pressure (RAP) (RVSP=TTPG + RAP), where trans-tricuspid gradient is $4V^2$ (V = peak velocity of tricuspid regurgitation, m/s).

Comparison of pulmonary artery hypertension with airflow limitation

Mild PAH was observed in 88 patients(81.48%) with mean RVSP=38.59mmHg. Among them 1 patient(1.13%) was grade I (FEV1%=81), 14(15.99%) patients were grade II COPD (mean FEV1%=58.33), 45 patients(51.13%) were grade III(mean FEV1%=39.288) and 28 patients(31.81%) were grade IV(mean FEV1%=25.89)COPD.Moderate PAH was observed in 17 patients(15.74%) with mean RVSP=52.47mmHg. Among them 1 patient(5.8%) was grade II COPD (mean FEV1%=57), 5patients(29.41%) were grade III(mean FEV1% =43) and 11 patients(64.70%) were grade IV COPD(mean FEV1%=26.45). Severe PAH was observed in 3 patients (2.77%) with mean RVSP=70.33mmHg among them all 3 patients (100%) were of grade IV COPD(mean FEV1%=24.66) (Table 2 & 3).

Treatment options

COPD patients with mild pulmonary artery hypertension were managed with inhaled bronchodilators and short term oral xanthine drug (Doxophylline). COPD patients with moderate pulmonary artery hypertension and pedal edema were treated with inhaled bronchodilators, short term oral xanthine drugs (Doxophylline) and short term diuretics.COPD patients with severe pulmonary artery hypertension and pedal edema were managed with inhaled bronchodilators, short term diuretics and selective phosphodiesterase inhibitors (selective PDE inhibitors). Pretreatment and post treatment quality of life and symptoms scoring was done using eight questions based COPD questionnaire called COPD assessment test (CAT scoring) for each patient. Pretreatment mean CAT scoring was 30.33 and post treatment mean CAT score had been reduced to 20.20 with a symptomatic reduction of 10.13 score with p value < 0.01 (Fig 4).

Comparison of pre and post treatment pulmonary artery hypertension using right ventricular systolic pressure

All COPD patients with mild PAH (n=88, 81.48%) had mean RVSP 38.59mmHg (SD 4.968) and were treated according to standard treatment protocol which comprised of inhaled bronchodilators and short term oral xanthine (doxophylline). PAH was re-assessedduring follow up which showed mean RVSP of 35.02mmHg (SD 5.121). Pre and post treatment RVSP analysis showed reduction of RVSP of 3.57mmHg with 95% confidence interval of 2.033-5.013(p = <0.01) along with improvement in quality of life and symptoms.COPD Patients with moderate PAH (n=17, 15.74%) had mean RVSP of 52.47mmHg (SD 3.484) were treated usingshort term diuretics, inhaled bronchodilators and short term oral xanthine (Doxophylline). During follow up we found mean RVSP of 46.53mmHg (SD 5.121). Pre and post treatment RVSP analysis showed reduction of RVSP by 5.928 mmHg (95% confidence interval 1.940-9.942) with p value of <0.01 along with improvement in quality of life and symptoms.COPD patients with severe PAH (n=3, 2.77%) with mean RVSP=70.33mmHg with standard deviation of 0.577mmHg were treated with vasodilators(selective phosphodiesterase inhibitor), short term diuretics, inhaled bronchodilators and oral xanthine (Doxophylline.During follow up mean RVSP was 59mmHg (SD 1.000). Pre and post treatment RVSP analysis showed reduction of RVSP by 11.330mmHg (95% confidence interval 7.539-15.128) with p value of

0.006along with improvement in quality of life and symptoms (Table 4).

Among 108 COPD patients who were recruited 2D resting echocardiography showed mean pretreatment RVSP of 41.66mmHg (SD 8.441). Reevaluation of pulmonary artery hypertension status showed mean post treatment RVSP of 37.50mmHg (SD 7.587). There was reduction of RVSP by 4.16mmHg (95% confidence interval 2.756-5.559) with p value of <0.01. This analysis showed reduction in pulmonary hypertension status with treatment of COPD along with improvement in quality of life and symptoms (Table 5).

Among 108 COPD patients considered for the present study 6 patients(5.54%) had corpulmonale. Of the 6 patients, 3 patients had severe PAH(100%) and 3 patients had moderate PAH(17.64%).

Demography variation

In our study we included male patients aged more than 40 years and majority belonged to the age group 61 – 70 years (49.07%) with mean age of 66.78 years (SD 7.589). A population based prevalence study of COPD in five Latin American cities (PLATINO STUDY) in post bronchodilator airflow limitation in persons aged above 40 years showed increased prevalence of COPD in patients aged above 60 years of age which is comparable with our study.[19]

Pulmonary function test

In our study majority of patients who were recruited fell under grade III (severe) which included 46.3%(50 patients) and grade IV (very severe) COPD, which included 38.8%(42 patients).There is good quality evidence from large studies that worsening airflow limitation is associated with increasing mortality and hospitalization rates, as well as increased risk of exacerbations.[20,21,22]

In our study the distribution of pulmonary artery hypertension in mild, moderate, severe and very severe COPD were 0.09%, 13.8%, 46.3% % and 38.8%, respectively which showed as severity of COPD increases the incidence of PAH also becomes high especially when FEV1% <50. Similar study which analyzedthe frequency of PAH in mild, moderate, severe, and very severe COPD were 16.67, 54.55, 60.00 and 83.33% respectively.[23] In another study, the frequency of PAH was also found to be 25%, 43% and 68% in mild, moderate, and severe COPD, respectively.[24]

Electrocardiogram

Among 108 COPD patients admitted with exacerbation, maximum i.e. 68 (62.96%) patients were having sinus tachycardia, while 20 (18.51%) were having normal heart rate. The mean heart rate recorded among COPD patients was 106.14 per minute. Followed by 19(17.59%) were having peaked p wave (p wave amplitude > 2.5mm) with right axis deviation and one patient (0.9%) with atrial fibrillation with irregularly irregular rhythm with loss of p wave. Duration of QRS and ST segment were normal in these patients.

The mean heart rate in the present study was recorded as 106.14 per minute as compared to 86 per minute obtained by Calatayudet al.[6] Normal sinus rhythm was recorded in 57.1% cases. Sinus tachycardia was present in 28.6% cases. Scott RC et al[7] reported arrhythmias other than sinus tachycardia to be uncommon in chronic corpulmonale.

During follow up post treatment there were no ECG changes compared to ECG taken during exacerbation status except sinus tachycardia has been normalised in 50 patients(73.5%) among 68 patients. No similar follow up studies were available to compare this outcome.

Echocardiography

Among 108 patients recruited mild PAH was observed in 88 patients(81.48%) with mean RVSP=38.59mmHg with standard deviation of 4.968mmHg. Moderate PAH was observed in 17 patients (15.7%) with mean RVSP=52.47mmHg with standard deviation of 3.484mmHg. Severe PAH was observed in 3 patients(2.77%) with mean RVSP=70.33mmHg with standard deviation of 70.33mmhg. It is similar to the studies which showed incidence of pulmonary artery hypertension directly proportional to severity of airflow limitation.[27,28,29]

Comparison of pulmonary artery hypertension with airflow limitation

Mild PAH was observed in 88 patients(81.48%) with mean RVSP=38.59mmHg. Among them 1 patient (1.13%) was grade I (FEV1=81%), 14(15.99%) patients were grade II COPD(mean FEV1=58.33%), 45 patients(51.13%) were grade III(mean FEV1=39.288%) and 28 patients(31.81%) were grade IV(mean FEV1=25.89%) COPD.Moderate PAH was observed in 17 patients (15.7%) with mean RVSP=52.47mmHg. Among them 1 patient(5.8%) had grade II COPD (mean fev1%=57%), 5patients(29.41%) were grade III(mean fev1% =43%) and 11 patients(64.70%) were grade IV COPD(mean fev1%=26.45%). Severe PAH was observed in 3 patients (2.77%) with mean RVSP=70.33mmHg among them all 3 patients (100%) were of grade IV COPD(mean fev1%=24.66%). This showed a positive correlation of airflow limitation with pulmonary artery hypertension. It is similar to the studies which showed incidence of pulmonary artery hypertension directly proportional to severity of airflow limitation.[23,24,30]

In our study the distribution of pulmonary artery hypertension in mild, moderate, severe and very severe COPD were 0.09%, 13.8%, 46.3% % and 38.8%, respectively which showed the severe the COPD, the incidence of PAH is high if in spirometry FEV1% <50%. Similar study which analysed frequencies of PAH

in mild, moderate, severe, and very severe COPD were 16.67%, 54.55%, 60.00% and 83.33%, respectively[23] In another study, the frequency of PH was also found to be 25%, 43% and 68% in mild, moderate, and severe COPD, respectively.[24]

In our study showed all patients with grade I, grade II, grade III spirometry had only mild to moderate pulmonary artery hypertension. Among patients with grade II spirometry grading 28/18(25.92%) had mild PAH, 11/108 (10.18%) had moderate PAH and 3/18 (2.77%) had severe PAH. All patients with severe PAH during acute exacerbation of COPD (3/108-2.77%) had grade IV spirometry grading.

Comparison of pre and post treatment pulmonary artery hypertension using right ventricular systolic pressure

The pathophysiology of the development of PAH in COPD is poorly understood and is likely multifactorial. The central stimulus to these processes remains chronic exposure of airways to noxious stimuli like tobacco and biomass smoke. Hypoxia has been classically considered to be the major pathogenic mechanism of pulmonary hypertension in COPD.[30,31] Chronic hypoxia induces predominant medial hypertrophy and is associated with complete reversal of pulmonary hypertension a few weeks after return to sea level.[32] Pathologic studies of lung specimens from patients with COPD have shown all vessel wall layers to be involved extensive pulmonary vascular remodeling with prominent intimal thickening, medial hypertrophy, and muscularization of small arterioles.

No studies are available on any correlation of PAH status with pre and post treatment of underlying airflow limitation and hypoxemia. In our study we analyzedpulmonary artery systolic pressure before and after treatment of acute exacerbation of COPD with use of right ventricular systolic pressure which was calculated using 2D resting echocardiography.

Among 108 COPD patients who were recruited 2D resting echocardiography showed mean pretreatment RVSP of 41.66mmHg with standard deviation of 8.441mmHg where started on treatment and kept under follow up. Reevaluation of pulmonary artery hypertension status showed mean post treatment RVSP of 37.50mmHg with standard deviation of 7.587mmHg with reduction of RVSP of 4.16mmHg with 95% confidence interval 2.756-5.559 with p value of 0.000 (<0.01). Our study showed significant reduction in right ventricular systolic pressures in all three groups of mild, moderate and severe PAH patients with significant p value of <0.01 on one month follow up with optimum medical management and symptomatic improvement. This analysis showed reduction in pulmonary artery pressure with treatment of COPD. No similar studies had done in the past to compare the outcome. Hence study with larger sample size is recommended.

TABLE 1: demography of the study population.

	NUMBER	MEAN AGE	STANDARD DEVIATION
MALES	108	66.78	7.589

TABLE 2: Distribution of pulmonary artery hypertension in the study population.

GRADING	NUMBER	MEAN RVSPmmHg	STANDARD DEVIATION
MILD	88 (81.48%)	38.59	4.968
MODERATE	17 (17.04%)	52.47	3.484
SEVERE	3 (2.77%)	70.33	0.577

TABLE 3: Comparison of pulmonary artery hypertension with airflow limitation.

PAH GRADING	SPIROMETRY GRADING	NUMBER (%)	MEAN FEV1 (%)
MILD PAH (N= 88 PATIENTS)	GRADE I	1 (1.13%)	81%
	GRADE II	14 (15.99%)	58.33%
	GRADE III	45 (51.13%)	39.29%
	GRADE IV	28 (31.81%)	25.89%
MODERATE PAH (N=17 PATIENTS)	GRADE II	1 (5.8%)	57%
	GRADE III	5 (29.81%)	43%
	GRADE IV	11 (64.70%)	26.45%
SEVERE PAH (N=3 PATIENTS)	GRADE IV	3 (100%)	24.66%

TABLE 4: Comparison of pre and post treatment right ventricular systolic pressure in study population

PAH GRADING	SPIROMETRY GRADING	MEAN PRE TREATMENT RVSP	MEAN POST TREATMENT RVSP	DIFFERENCE
MILD PAH (N= 88 PATIENTS)	GRADE I (N=1)	32 MMHG	30 MMHG	-2MMHG
	GRADE II (N=15)	39.07MMHG	34 MMHG	-5.07MMHG
	GRADE III (N=45)	38.71MMHG	35.11MMHG	-3.6OMMHG
	GRADE IV (N=28)	38.46MMHG	35.50MMHG	-2.96MMHG
MODERATE PAH (N=17 PATIENTS)	GRADE II (N=1)	51MMHG	41MMHG	-10MMHG
	GRADE III (N=5)	53.40MMHG	44.80MMHG	-8.60MMHG
	GRADE IV (N=11)	51.81MMHG	47MMHG	-4.81MMHG
SEVERE PAH (N=3 PATIENTS)	GRADE IV (N=3)	70.33MMHG	59MMHG	-11.33MMHG

TABLE 5: Statistical analysis of pre and post treatment right ventricular systolic pressure in study population (N=108)

PAH STATUS	RVSP	MEAN RVSP	STANDARD DEVIATION	95% CONFIDENCE LIMITS	P VALUE
MILD PAH (N=88)	PRE TREATMENT	38.59MMHG	4.968MMHG	2.033-5.103	.000 (<0.01)
	POST TREATMENT	35.02MMHG	5.121MMHG		
MODERATE PAH (N=17)	PRE TREATMENT	52.47MMHG	3.484MMHG	1.940-9.942	.000 (<0.01)
	POST TREATMENT	46.53MMHG	5.928MMHG		
SEVERE PAH (N=3)	PRE TREATMENT	70.33MMHG	0.577MMHG	7.539-15.128	.006 (<0.01)
	POST TREATMENT	59.00MMHG	1.000MMHG		
TOTAL (N=108)	PRE TREATMENT	41.66MMHG	8.441MMHG	2.756-5.559	.000 (<0.01)
	POST TREATMENT	37.50MMG	7.587MMHG		

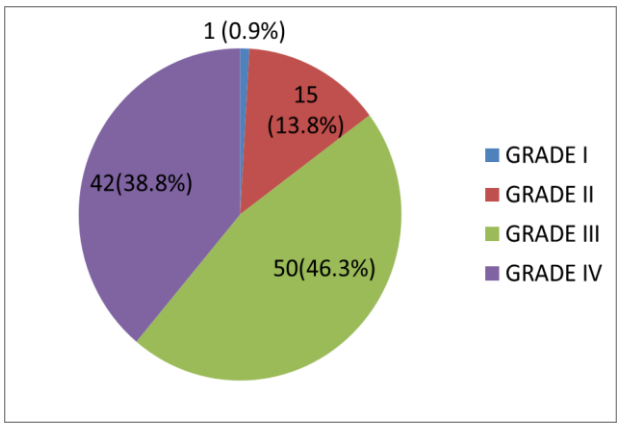

Fig. 1: Distribution of airflow limitation in study population (n=108).

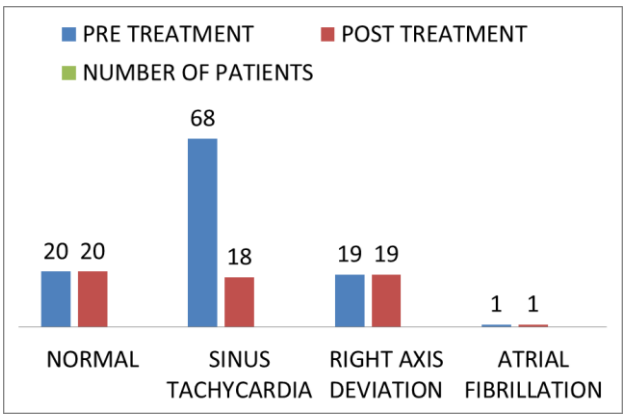

Fig. 3: Pre and post treatment ECG variation observed in the study population (n=108).

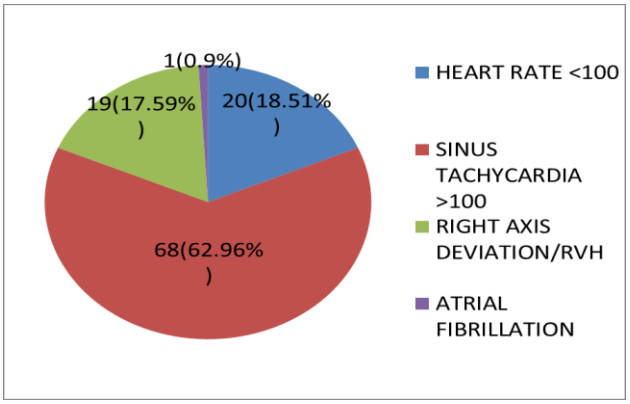

Fig. 2: ECG findings in the study population (n=108).

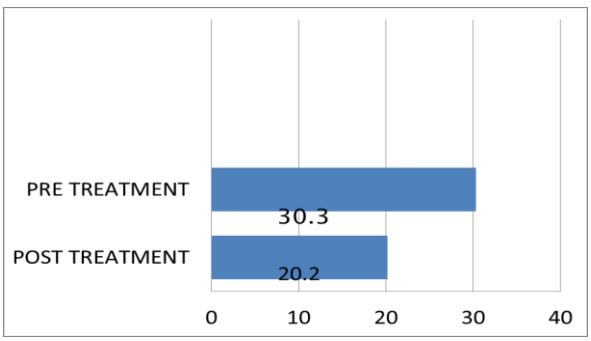

Fig. 4: Mean pre and post treatment COPD assessment test (CAT) scoring in study population (n=108).

CONCLUSION

Most of the patients admitted with acute exacerbation of COPD had only mild to moderate pulmonary artery hypertension.The pulmonary artery pressure comes down significantly on one month follow up with optimum medical management. Most common ECG abnormality during an exacerbation was sinus tachycardia and close to 75% had normalized during the follow up. Echocardiography screening of COPD patients is beneficial to assess the pulmonary artery ressure and identify patients with severe pulmonary artery hypertension, who may benefit with addition of phosphodiesterase inhibitors. Study with larger sample size is recommended.

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