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RESEARCH ARTICLE

Myocardial dysmorphism and functional failure: A cardiac sting in patients with advancing chronic obstructive pulmonary disease

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ABSTRACT

Background: Extrapulmonary cardiac manifestations in chronic obstructive pulmonary disease (COPD) exhibit a great deal of deterioration in ventricular geometry and function, contributing to high mortality and morbidity. Echocardiography data regarding ventricular remodeling and functional deterioration in COPD patients are rare and insufficient. **Aims and Objectives:** The aim of the study was to assess and compare the early deterioration of ventricular geometry and function by echocardiography in COPD (Group-2) and non COPD patients (Group-1). **Materials and Methods:** In this study, 150 individual patient data (IPD) patients diagnosed of COPD admitted in a pulmonary unit of Shri Ram Murti Smarak Institute of Medical Sciences, Bareilly, Uttar Pradesh, between 30 and 80 years of both the sexes, were analyzed randomly (Group-2). Retrospective comparative study was undertaken with the same number of IPD patients admitted for other ailments (Group-1). Pulmonary function test, electrocardiography, and two-dimensional-echocardiography were done to assess and compare the ventricular geometry and function. **Results:** The study revealed a significant increase in the echo parameters such as left ventricular (LV) internal dimension in diastole (<0.0001**) LV internal dimension in systole (<0.0345**), posterior wall thickness (<0.0067**), relative wall thickness (RWT) (<0.0001**), LV ejection fraction (EF) % (<0.0001**) E-velocity (<0.0001**), and E-and A-wave velocity ratio (<0.0001**). On the other hand, right ventricular (RV) parameters such as RV internal dimension in diastole (<0.0073**), RV mass (<0.0001**), RVEF, deceleration time (<0.0008**), pulmonary artery pressure (<0.0001**), and pulmonary flow (<0.0001**) were significantly high in COPD patients. Higher RWT (<0.0001**) in COPD was significantly associated with reduced forced expiratory volume at 1s (FEV1), forced vital capacity (FVC), and the ratio (FEV1/FVC). **Conclusion:** COPD patients exhibited biventricular hypertrophy and diastolic and systolic dysfunction resulting in four geometrical patterns, namely concentric hypertrophy, eccentric hypertrophy, concentric remodeling, and normal geometry. It also causes increase LV mass, LV mass index, and decrease in EF% and LV volume as a deteriorating compensatory effect.

KEY WORDS: Chronic Obstructive Pulmonary Disease; Two-Dimensional-echocardiography; Left Ventricular Hypertrophy; Diastolic Dysfunction

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INRODUCTION

Chronic obstructive pulmonary disease (COPD) has a worldwide distribution and a respiratory hazard which is capable of affecting the pulmonary vasculature and lung parenchyma. Later the brunt of attack falls on the myocardium in the form of extrapulmonary cardiac manifestations, apart from various other manifestations. There are multiple risk factors contributing to COPD among

which cigarette smoking is one of the primordial risk factors in terms of high mortality and morbidity due to early cardiac failure in COPD.[1]

Nowadays, the incidence of COPD is rising and therefore it is estimated that by 2020 perhaps it may surpass and becomes the third most leading cause of mortality and the fifth most leading cause of morbidity in the whole world.[2] Moreover, there are observations of increasing death rates in COPD among Indian and further estimated that around 30 million people may be suffering from the disease by 2020.[3] Epidemiological data suggest that most of the deaths in COPD occur due to pulmonary hypertension and cor pulmonale. Mild and moderate COPD is less likely to produce heart failure, but in long term, it may prove fatal. COPD may be responsible for chronic but significant reversible airflow restriction preceded by chronic inflammation of the cardiopulmonary vasculature. It includes chronic bronchitis, emphysema, and bronchiectasis. Pathophysiologically, it is produced as a result of the partial or complete destruction of the lung parenchyma. COPD may be further contributed by an abnormal inflammatory response in the alveoli and respiratory unit which may be triggered by various stimuli such as cigarette smoking (active or passive), smoke, dust, industrial or environmental pollution, and toxic gases.[4]

Extrapulmonary manifestations of COPD are primarily cardiovascular; therefore, the brunt of attack falls on the myocardium which manifests in the form of widespread ventricular remodeling and systolic and diastolic dysfunction. COPD is responsible for the rise in the pulmonary artery pressure (pulmonary hypertension) in severe and very severe disease in the long term may precipitate right ventricular (RV) failure (cor pulmonale). Coronary artery disease is among the other complications, which may lead to heart failure and sudden death in COPD patients.[5] Many times primary cardiac failure and COPD related cor pulmonale may mimic, and therefore, classification becomes really difficult. Thus, it is of utmost importance to categorize the type of heart failure to ascertain the management and prognosis.[6] Mortality in COPD may increase in other complications such as arrhythmia, myocardial infarction, valvular heart disease, and congestive heart failure. Both cardiac geometry and function are equally affected in COPD by failure of compensatory mechanisms procuring global cardiac remodeling.[7]

According to the severity, duration and pulmonary artery pressure COPD may be categorized as mild, moderate, and severe. Pulmonary artery pressure systolic pulmonary arterial pressure (sPAP) >30 mmHg is considered pulmonary hypertension mild when sPAP >30–50 mmHg, moderate when sPAP >50–70 mmHg, and severe when sPAP >70 mmHg, respectively.[8] Terminal changes in the RV occur when the compensatory mechanism fails to cope up with hemodynamic challenges procured by increasing pulmonary artery pressure whereby, failure of RV ensues in the form of cor pulmonale.[9]

Several investigation modalities are available for ascertaining the structural framework of the heart in COPD, but echocardiography has general acceptance. However, echocardiography data in COPD patients are incomplete and rare, which invited further exploration into this field of research. Thus, it becomes mandatory to assess the cardiac remodeling status in COPD. In the modern times, echocardiography remains a reliable modality to accurately assess the cardiac profile to reach a conclusive outcome. It is still a cost effective, reliable and non-invasive methodology of cardiac assessment.[10] Cardiac angiography and right heart catheterization are the alternative invasive investigations which may not have that practical acceptance. Timely echocardiography in COPD patients may change the outcome of the disease by finding out the geometrical deviation and dysfunction so that the clinical course of the disease may be modulated by early pharmacological and interventional management. Further, this domain of the study is still very inviting to us and throws challenges to explore the hidden vector.

Hence, a new interventional and management protocol may be postulated to redefine the clinical outcome and course of the disease.

MATERIALS AND METHODS

This study was undertaken with the single mindset that there is so much to suffer for a COPD patient who is apparently ignorant about the extent of ongoing disease in them and may not be guided properly by the health workers. Keeping this in mind, an early echocardiography initiative should be introduced in COPD patients to detect the extent of damage so that the association of the disease with cardiac geometry and function may be established.

The first step was to obtain a written and informed consent from the patients. Permission from the ethical committee of our college was sought before the initiation of the study. With this vision, the study was conducted in Sri Ram Murti Smarak Institute of Medical Sciences, Bareilly, Uttar Pradesh. 150, individual patient data (IPD) patients diagnosed with COPD of both the sexes between 30 and 80 years of age were randomly selected (Group-2). On the other hand, 150, IPD patients admitted for other ailments were selected (Group-1).

Patients with renal failure, hepatic failure, hypothyroidism, diabetes mellitus, hypertension, and other cardiac disorders were excluded from the study.

Clinical Examination

Elaborate history taking, clinical examination, blood pressure measurement, electrocardiography, two-dimensional (2-D) echocardiography, and color Doppler were undertaken in both the group of patients.

Anthropometric Parameters

Weight (Wt) was recorded with clothes off by digital weighing machine, height (Ht) was recorded in standing by stadiometer. Blood pressure was recorded by diamond mercurial sphygmomanometer, two readings 10 min apart after 30 min of rest during lying down position. Limits of normal blood pressure were taken as <120 mmHg systolic and <80 mmHg diastolic.[11] Pulse pressure (PP) was expressed as (PP) = Systolic blood pressure (SBP)−diastolic blood pressure (DBP), and mean arterial pressure as (MAP) = DBP+1/3 PP. Body surface area as BSA = ([Ht (cm) × Wt [kg]/3600)$^{1/2}$ and body mass index as BMI= Wt/Ht^2.

Pulmonary Function Test

Pulmonary parameters were recorded by the electronically calibrated spirometer (Siemens automated device) with graphical recording. Functional expiratory volume in 1 s (FEV1), forced vital capacity (FVC), functional residual capacity, total lung capacity, and the ratio (FEV1/FVC) was later calculated.

2-D Echocardiography and Color Doppler

2-D echocardiography was done in both the groups of patients by the portable echocardiography machine (Siemens, Aspen) at bedside by interventional cardiologist assisted by qualified echo technician. Transthoracic approach was adopted to make out the good images of all the views. Basic echocardiographic parameters were obtained and recorded on the pro forma of the patients; later calculations were done to find out the critical parameters such as left ventricular mass (LVM), LV mass index (LVMI), and relative wall thickness (RWT). Further, color Doppler was done to assess the hemodynamic profile. All the echocardiography parameters recorded by the general convention given by the American Society of Echocardiography. Following formulas were given to calculate the variables.

Systolic Echocardiographic Parameters

Basic systolic parameters are like stroke volume (SV) = (LV internal dimension in diastole [LVIDd])^3- (LV internal dimension in systole [LVIDs])^3, cardiac output (CO) = SV × heart rate (HR), and cardiac index (CI) = CO × BSA. Others are ejection fraction (EF %)= LVIDd^3 – LVIDs^3 × 100/LVIDd^3, and fractional fiber shortening (FFS)= LVIDd - LVIDs × 100/LVIDd. Stress parameters during systole are end-systolic stress = 0.334 × SBP × LVIDs/posterior wall thickness (PWTs) × (1 + PWTs/LVIDs) and end isovolumetric systolic stress =0.334 × DBP × LVIDs/PWTs × (1+PWTs/LVIDs), total peripheral resistance = (mean blood pressure × 80/CO).

Diastolic Echocardiographic Parameters

The blood flow velocities in terms of transmitral and transtricuspid flow across the valves in differing cardiac cycles and their wave patterns which depicts A velocity, E velocity, and (E-and A-wave velocity) E/A velocity ratio in both the ventricles.

Geometrical Parameters

Various geometrical parameters are LVM = 0.8 (1.04 [Interventricular septal (IVS) + LVIDd + PWT]^3 – [LVIDd]^3 + 0.6), LVMI = 1.04 (IVS thickness in diastole + LVIDd + PWTd)^3 − LVIDd^3/BSA and RWT = 2 × PWd/LVIDd.[12] Considering the LVMI and RWT, various geometrical patterns and cardiac remodeling were assessed. These are the mainstay in defining the ventricular hypertrophy by LVM/BSA when the data go beyond 0.95 g/m^2 in women and 0.115 g/m^2 in men, respectively, in Indian scenario.[13] Accordingly, LV hypertrophy (LVH) and RV hypertrophy (RVH) pattern is categorized as normal geometry (NG) when normal RWT and LVMI, concentric remodeling (CR) when increased RWT and normal LVMI, eccentric hypertrophy (EH) when normal RWT and increased LVMI, and concentric hypertrophy (CH) when increased RWT and increased LVMI were there.

Statistical Analysis

Statistical analysis was done after obtaining the gross data. Mean and standard deviation was calculated depending on the data categorization. All the parameters of both groups were compared. Student's *t*-test and Pearson correlations coefficient were applied to assess and establish the associations among COPD and controls groups.

Multivariate regression analysis with all the echocardiography parameters to establish an association between the two groups. SPSS software was used to assess and analyze the data. $P < 0.05$ was considered significant, a value <0.0001 considered highly significant and $P > 0.05$ was considered nonsignificant.

RESULTS

The demographic data suggest in this study that age, Ht, Wt, BMI, and BSA did not inflict much impact on the two groups; however, there are insignificant variations. Moreover, blood pressure profile was slightly altered although SBP, DBP, and MAP. PP, HR, and CI showed a significant elevation in the control group [Table 1]. CO and SV did not show any significant variation between the two groups.

Spirometry tests revealed the highly significant decline of dynamic parameters in COPD (Group-2) especially FEV1 (<0.0001**), FVC (<0.0001** and FEV1/FVC (0.0008**) ratio as compared to non COPD (Group-1). Pulmonary vascular parameters did show significance in terms of sPAP (<0.0001**), right atrial pressure-(<0.0026*), and tricuspid regurgitation velocity-(<0.0042*). Although, mean

pulmonary arterial pressure-<0.0237 did not show significant variation in two groups [Table 2].

It was notworthy in the study that there is a significant decline in the chamber size in both the ventricles more of the left ventricle in COPD predicted by LVIDd, LVIDs, and interventricular septal thickness (IVST). Moreover, it was not the case in the control group which revealed the kind of stress which is acquired during the high pressure hampering compensatory mechanism to operate inadvertently. With this reason, CH is considered highly fatal, which increases mortality in COPD patients COPD, therefore, has a definite relationship with the RWT [Table 3].

It was evident that there is RV diastolic failure, which ensures early in the course of the disease given by the declining E/A ratios in patients with COPD.

We observed LVH patterns as CH – 5%, EH – 6%, CR – 5%, and NG – 84%.

Table 4 indicates mild compensatory changes occurring in the RV dimensions in COPD. Due to high pulmonary artery pressure in severe and very severe COPD patients. The compensatory changes fail and the hemodynamic profile gets deranged leading to hypertrophy. Transmitral blood flow and transtricuspid flow is equally affected. This establishes a significant relationship among FEV1, FVC, and FEV1/FVC ratio with RV hypertrophy.

We observed RVH patterns as CH – 10%, EH – 23%, CR – 17%, and NG – 50%.

DISCUSSION

It is a well-known fact that COPD is primarily a respiratory insufficiency disorder affecting cardiac geometry and function as an extrapulmonary manifestation of the disease in the long run. Chronic inflammation of the lung parenchyma and other vascular changes are the hallmark of the disease apart from ventricular hypertrophy and heart failure. RV is largely affected due to the immense pressure gradient in the pulmonary vasculature due to pulmonary hypertension. Although it is worth to remember that left ventricle is equally affected

Table 1: Demographic and cardiovascular profile in COPD and control groups

Demographic and cardiovascular parameters	Control Group-1 (*n*=150)	COPD Group-2 (*n*=150)	*P*
Age (years)	55.87±10.76	56.67±11.65	0.5372
Ht (m)	1.65±0.04	1.45±10.91	0.8225
Wt (kg)	71.87±13.8	69.56±12.97	0.1363
BMI (kg/m^2)	25.88±11.87	24.56±10.22	0.3029
BSA (m^2)	1.77±4.23	1.66±4.11	0.8195
SBP (mmHg)	130±10	131±10	0.3872
DBP (mmHg)	80±7	81±9	0.2836
PP (mmHg)	55.23±5.6	53.16±5.9	0.0020*
MAP (mmHg)	95±7	96±3	0.1089
HR (beats/min)	86±6	90±6	0.0001**
CO (L/min)	5.2±0.4	5.1±0.8	0.1719
CI	3.33±0.12	4.00±0.11	0.0001**
SV (ml)	66±4	66±9	1.0000

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, PP: Pulse pressure, MAP: Mean arterial pressure, HR: Heart rate, CO: Cardiac output, CI: Cardiac index, SV: Stroke volume, COPD: Chronic obstructive pulmonary disease, BSA: Body surface area, BMI: Body mass index

Table 2: Spirometry and pulmonary vascular parameters in COPD and control group

Spirometry and pulmonary vascular parameters	Control Group-1 (*n*=150)	COPD Group-2 (*n*=150)	*P*
FEV1 (L)	0.55±0.07	0.43±0.04	0.0001**
FVC (L)	1.32±0.08	1.22±0.06	0.0001**
FEV1/FVC (%)	42.50±10.68	38.57±9.45	0.0008**
SPAP	26.60±0.6	30.97±0.5	0.0001**
RAP	8.01±0.04	7.99±0.07	0.0026*
TRV	2.55±0.034	2.56±0.036	0.0042*
MPAP	18.92±0.05	18.91±0.02	0.0237

FEV1: Forced expiratory volume in 1s, FVC: Forced vital capacity, SPAP: Systolic pulmonary arterial pressure, RAP: Right atrial pressure, TRV: Tricuspid regurgitation velocity, MPAP: Mean pulmonary arterial pressure, COPD: Chronic obstructive pulmonary disease

Table 3: LV echocardiographic parameters in COPD and control groups and its relationship with cardiac geometry

LV Echo parameters	Control group (*n*=150)	COPD group (*n*=150)	*P*	CH (10%)	Eccentric hypertrophy EH (23%)	CR (17%)	NG (50%)	*P*
LVIDd (mmHg)	42.89±4.23	45.76±5.56	0.0001**	47.89±4.23	41.76±5.56	41.89±4.23	40.76±5.56	0.0001**
LVIDs (mmHg)	13.98±3.11	14.77±3.33	0.0345*	14.98±3.11	12.77±3.33	13.98±3.11	13.77±3.33	0.1977
PWT (mmHg)	13.36±5.55	14.88±3.95	0.0067**	14.36±5.55	13.88±3.95	11.36±5.55	11.88±3.95	0.0418*
IVST (mmHg)	12.86±2.87	9.34±3.21	0.4143	14.82±2.87	10.14±3.21	12.26±2.87	12.34±3.21	0.0067**
LVM (gm)	120.22±14.67	122.56±88	0.9791	131.22±14.67	119.56±88	117.22±14.67	122.56±88	0.7060
LVMI (gm/m)	44.65±9.98	45.43±8.87	0.4749	46.65±9.98	43.43±8.87	44.65±9.98	43.43±8.87	0.2120
RWT (mmHg)	0.45±0.047	0.55±0.065	0.0001**	0.55±0.047	0.45±0.065	0.46±0.047	0.44±0.065	0.0001**
LVEF (%)	67.98±11.76	60.34±12.0	0.0001**	55.98±11.76	63.34±12.0	66.98±11.76	67.34±12.0	0.0012**
E velocity (m/s)	65.89±17.88	55.98±18.34	0.0001**	0.44±0.03	0.65±0.054	0.45±0.033	0.65±0.065	0.0001**
A velocity (m/s)	77.34±18.78	74.67±13.67	0.1602	55.89±17.88	65.98±18.34	63.89±17.88	64.98±18.34	0.0820
E/A ratio	0.65±0.30	0.88±0.45	0.0001**	0.66±0.30	0.87±0.45	0.540.30	0.84±0.45	0.1421
ESS	1.080±0.23	1.074±0.54	0.9005	1.076±0.23	1.074±0.54	1.056±0.45	0.912±0.78	0.4179
EISS	0.44±0.01	0.80±0.03	0.0001**	0.533±0.01	0.87±0.03	0.66±0.04	0.657±0.65	0.0001**
TPR	1165±88	1160±87	0.6211	1175±88	1167±87	1065±99	1099±87	0.0027*

LVIDd: Left ventricular internal dimension in diastole, LVIDs: Left ventricular internal dimension in systole, PWT: Posterior wall thickness, IVST: Interventricular septal thickness, LVM: Left ventricular mass, LVMI: Left ventricular mass index, RWT: Relative wall thickness, LVEF: Left ventricular ejection fraction, EISS: End isovolumetric systolic stress, COPD: Chronic obstructive pulmonary disease, NG: Normal geometry, ESS: End-systolic stress, TPR: Total peripheral resistance

Table 4: RV Echocardiographic parameters in COPD and control groups and its relationship with the type of cardiac hypertrophy

RV ECHO parameters	Control (Group-1) (150)	COPD (Group-2) (150)	*P*	CH (5%)	EH (6%)	CR (5%)	NG (84%)	*P*
RVIDd (mm)	42.87±12.65	46.87±13.0	0.0073 **	43.87±12.65	44.87±13.0	40.87±12.65	45.87±13.0	0.5863
RVIDs (mm)	25.45±11.0	26.98±11.66	0.2433	27.45±11.0	25.98±11.66	23.45±11.0	24.98±11.66	0.4519
RVM (gm)	134.20±14.56	143.10±13.98	0.0001**	138.20±14.56	134.10±13.98	136.20±14.56	133.10±13.98	0.2035
RVMI (g/m)	33.98±11.0	38.70±12	0.0004	37.70±11.0	37.70±12	32.98±11.0	32.70±12	0.1392
RVEF (%)	38.22±12.0	30.23±10.23	0.0001**	32.22±12.0	41.23±10.23	41.22±12.0	41.23±10.23	0.0033**
Tricuspid Epv (cm/s)	37.9±8.9	42.9±8.7	0.3259	39.2±7.8	40.2±9.98	38.54±10	37.87±32	0.6488
Tricuspid Apv (cm/s)	41.5±9.5	40.1±7.1	0.1493	41.33±12	37.77±07	39.21±10	40.98±11	0.9047
DTEE′ pv (cm/s)	11.1±4.0	12.6±3.7	0.0008**	12.33±3.8	13.87±3.8	12.98±11	13.33±12	0.4623
DTEA′ pv (cm/s)	16.3±4.3	17.5±4.2	0.0151	17.33±3.9	16.45±3.3	18.76±12	15.67±4.1	0.3335
Tricuspid E/Apv (cm/s)	1.0±0.3	1.0±0.4	1.0000	1.01±0.2	1.02±0.3	1.03±0.3	1.01±0.1	1.0000
Pulmonary pressure (mmHg)	29.9±8.2	40.8±3.4	0.0001**	29.54±7.8	30.33±8.1	29.67±7.0	30.01±0.1	0.4644
Pulmonary flow (m/s)	117±29	141±22	0.0001**	117±22	118±21	116±19	120±18	0.6934

RVIDd: Right ventricular internal dimension in diastole, RVIDs: Right ventricular internal dimension in systole, RVM: Right ventricular mass, RVMI: Right ventricular mass index, RVEF: Right ventricular ejection fraction, COPD: Chronic obstructive pulmonary disease, NG: Normal geometry

in later stages in severe and very severe COPD. This study highlights about the structural and functional compensatory alterations operating in COPD, leading to the development of cardiac hypertrophy. LV echo parameters deranged are LVIDd, LVIDs, LVM, LVMI, LVEF%, etc. Similarly, RV parameters deranged are RVM, RVMI, RVEF %, etc., and the velocities also mimic the same effect. Spirometric parameters such as FEV1, FEV2, and FEV1/FVC ratio are significantly deranged in COPD as compared to non COPD group. These parameters were found to be reduced in accordance with the

grade of COPD; thereby, it gave us an idea about a strong link between pulmonary parameters and ventricular echo parameters.

The cardinal finding in our study was the existence of RV remodeling and hypertrophy preceded by RV dysfunction. Biventricular hypertrophy had differing geometrical patterns, namely CH, CR, EH, and NG. Mild COPD was found to be more associated with EH and CR, whereas severe and very severe COPD were found to be associated with CH. This seems true because the hemodynamic changes occurring are mainly due to the high pulmonary pressure gradient, which ultimately reflects in the RV. Modern echocardiography and color Doppler technique are capable of revealing early compensatory changes in COPD.[14]

This study demonstrated the relationship of RWT, LVMI, and BSA in defining biventricular hypertrophy. It was found that RWT values >0.42 are more linked with the COPD group and are likely to procure CH, which is the fatal form of hypertrophy. The severity of COPD and deranged PFT will adversely affect the type of RV hypertrophy especially concentric.[15]

RWT was found to be more reliable indicators which showed a strong association with the RV hypertrophy. In our study, the RWT was significantly elevated in COPD, which is complying with the earlier studies by Anderson *et al*.[16] These findings are in line with the similar findings observed in other studies.[16] Furthermore, previous studies revealed the higher association of COPD with LVM in comparison to non COPD group.[17] Age factor has a strong yet unclear role in modulating LVM, the same was observed in our study, though, we did not have any significant age variation between the two groups. BMI and BSA perhaps may modify the LVM and therefore LVMI.[18]

This study, however, did find few modifiable differences in LVH in COPD, which relates its association with a significant increase in RWT as compared with controls. Determination of RWT and RVM may be crucial for the outcome of the disease and may reflect in mortality and morbidity.[19] The study showed the link between reduced RV cavity in COPD due to pulmonary hypertension leading to increase in RV afterload. On the other hand, there is a lower preload to LV. Many authors theorize about microcardia in COPD contributed by lung hyperinflation and reduced intrathoracic compliance.[20] This mechanism may solitary or in conjunction with the other compensatory mechanisms which fails to meet the demands in terms of EF% in COPD. Later, it may precipitate diastolic dysfunction due to incomplete relaxation of the two chambers. However, systolic dysfunction may also occur due to insufficiency of SV to maintain adequate CO. We, therefore, did not find significant alterations of SV, CI, and CO in the COPD group in the study.

Cardiac compliance may not be solely affected by inflow pattern and ventricular relaxation in terms of SV, CI, and CO, but it is influenced by preload and heart rate. This is justified because it is a proven fact that there is a direct relationship of preload to the myocardial fiber length and hence the degree of ventricular relaxation during diastole.[21] Moreover, cardiac hemodynamic velocities (E, A, and E/A ratio) were found to significantly deranged in our study which gave us an indication about the quantum of load to the ventricle, RV becomes more vulnerable for diastolic dysfunction and failure.[22]

Further, it was observed that once RV pressures had passed beyond the compensatory limits, the pathology reflected in LV also, in terms of IVST and deviation to the opposite side. Hence, our findings are in agreement with many other studies in which there was no RV enlargement despite impending pulmonary artery pressures.

Limitations of the Study

Our limitation for the study was the small sample size, though it may involve a large cohort for further studies. Advance invasive investigations should be introduced such as cardiac angiography and right heart catheterization for the accurate assessment of the underlying pathological process in COPD. Follow-up of the study subjects could not be done to ascertain the prognostic implications of medical management.

CONCLUSION

This study concludes that the most important echocardiography parameter in the assessment of cardiac hypertrophy in COPD patient is perhaps RVM, RVMI, and RWT. Many factors are, therefore, influencing these parameters as a triggering factor for the initiation of a cascade of events leading to the development of ventricular hypertrophy in COPD. It is, therefore, advisable to start an awareness program to detect COPD in its initial mild form so as to restrict the pathological changes to occur. Moreover, a campaign should be introduced to undergo echocardiography in the early stages so that mortality and morbidity associated with COPD can be curtailed. This will bring a paradigm shift in the clinical course of the disease outcome in the future. Therefore, this area is wide open and inviting for future studies.

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