

ORIGINAL ARTICLE

Early Detection of Right Ventricular Dysfunction in Chronic Obstructive Pulmonary Disease by Echocardiography

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Abstract

Objectives: This study is designed to investigate the effects of pulmonary arterial hypertension on RV systolic and diastolic functions in cases of COPD and to correlate RV systolic and diastolic functions with pulmonary arterial pressure.

Material and Methods: 100 patients admitted in various medical wards of tertiary care Hospital and a primary care hospital with stable chronic obstructive pulmonary disease persons with age and sex-matched. 35 age and sex matched person without any associated and known disease were taken as control subjects. Selection of cases has been made on basis of detailed history, thorough clinical examination, electrocardiography, chest x-ray, pulmonary function tests.

Observation: RV Systolic function (RVEF and RVWT) are significantly abnormal in patients of stable compensated COPD and they are significantly correlated with PAP($p < 0.002$). RV diastolic function i.e., E/A ratio and PFR are altered in 60%(n=60) of patients of COPD studied against control subjects and significantly correlated with PAP($p < 0.002$).

Conclusion: Echocardiography is a non invasive method to detect changes of right ventricular dysfunctions in early stages with very good significant sensitivity and specificity.

Introduction

Chronic obstructive pulmonary diseases (COPD) is among the major causes of disability and mortality worldwide and recently its incidence is on an uphill. As the disease progresses, pulmonary arterial hypertension develops insidiously which leads to development of RV hypertrophy, dilatation and failure. Once Cor-pulmonale becomes evident, the prognosis is poor. Early detection and therapy can lead to a reduction of symptoms, as well as the rate of progression of the disease. Hypertrophy and/or dilatation of the right ventricle are the major factors which contribute to the consequences of the disease.

Various non-invasive techniques such as x-ray chest, electrocardiography, vector cardiography, Thallium 201 myocardial imaging and gated equilibrium blood pool imaging are available, but individually each of them has low sensitivity in detection of right ventricular function. Echocardiography offers promise in

identifying clinically occult pulmonary arterial hypertension, right ventricular enlargement, hypertrophy and right ventricular systolic and diastolic functions in patients with COPD. RV diastolic properties can be evaluated with ease with the help of pulse Doppler echocardiography.

This study was designed to investigate the effects of pulmonary arterial hypertension on RV systolic and diastolic functions in cases of COPD and to correlate RV systolic and diastolic functions with pulmonary arterial pressure.

Material and Methods

100 patients admitted in various medical wards of tertiary care Hospital and a primary care hospital with stable

chronic obstructive pulmonary disease persons with age and sex matched. 35 age and sex matched person without any associated and known disease were taken as control subjects.

Selection of cases has been made on following criteria:

1. **Clinical History:** According to American Thoracic Society on Diagnostic Standard for COPD, patients had to have history of cough with sputum for at least three consecutive months for each of two consecutive years or presence of breathlessness with radiological evidences of emphysema or both.
2. **X ray Chest -Plain/Digital X-ray** of the chest showing over-inflation with increased bronchovascular markings. Chronic bronchitis cannot be diagnosed on the basis of radiological features which are helpful in the diagnosis of emphysema alone. Characteristic features of emphysema include depression and flattening of diaphragm, irregular radiolucency of lung fields, increase in retrosternal space, long tubular heart. Attenuation of peripheral pulmonary vasculature, widening of intercostal spaces and prominence of main branches of pulmonary artery, emphysematous bullae etc.
3. **Electrocardiography:** evidences consistent with COPD and its sequelae like progressive right axis deviation of P wave and QRS, R:S ratio becomes less than 1 in lead V6 and increasing amplitude of P

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Received: 15.06.2018; Accepted: 06.08.2018

Table 1: Right ventricular functions measured by 2D echocardiography of controls and cases

Variable	Patients Mean ± SD	Controls Mean ± SD	Student's t test	D.F.	P value
PAP (mm)	29.520± 7.180	14.820 ±2.680	6.546	34	0.01
RVEF (%)	39.760± 6.670	57.820 ±3.950	-8.324	34	0.01
RVWtd (mm)	0.970 ±0.226	0.550 ±0.180	5.390	34	0.01
"E" Wave (mm)	0.540 ±0.068	0.550 ±0.090	-0.448	34	NS
"A" Wave (mm)	0.550 ±0.073	0.580 ±0.071	-0.493	34	NS
E/A	0.980 ±0.152	1.480 ±0.345	-6.044	34	0.01
PFR (mm)	4.350 ±0.711	6.060 ±0.843	-6.288	34	0.01

wave in standard leads II,III and aVF.

- Pulmonary function test: The diagnostic criteria for COPD are FEV1 less than 75% and FEV1/FVC ratio less than 80% of the value predicated from measurement of pulmonary function in asymptomatic non-smoker.

Such selected patients were subjected to echocardiographic examination with colour Doppler echocardiographic machine complete M-mode. 2-Dimensional and pulsed wave Doppler echocardiographic studies with other relevant parameters.

Statistical Methods and Analysis

Such collected data was tabulated and reported as mean value ± standard deviation. Student's 't' test was used to assess the significance of the difference in pulmonary function and echocardiographic data between the COPD and control subjects.

Correlation coefficients were obtained from standard regression equation between the PAP and various echocardiographic parameters. A 'p' value < 0.05 was considered statistically significant.

Observations

The present study consists of detection of RV systolic and diastolic dysfunction in patients of COPD by 2-dimensional echocardiography and its correlation with pulmonary artery pressure (PAP)

In the present study PAP and following systolic and diastolic functions in patients of COPD were measured and correlated with control group. The mean ± SD value of PAP in COPD and control subjects was 29.520 ± 7.180 and 14.820 ± 2.680 respectively. The 't' value was 6.546 and 'p' value was significant (p < 0.01). The RVEF in both subjects was 39.760 ± 6.670 and 57.820 ± 3.950 respectively. The t value was -8.324 and p value was significant

(p < 0.01). The RVWtd in both subjects was 0.970 ± 0.226 and 0.550 ± 0.180 respectively. The 't' value was 5.390 and 'p' value was significant (p < 0.01) (Table 1).

To study the diastolic functions following echocardiographic parameters were measured in control and COPD subjects- 'E' wave, 'A' wave, E/A ratio and peak filling rates. The 'E' wave in both subjects was 0.540 ± 0.068 and 0.550 ± 0.090 respectively. 't' value was 0.448 and 'p' value was insignificant. The 'A' wave in both subjects was 0.550 ± 0.073 and 0.580 ± 0.071 respectively. 't' value and 'p' values in both subjects was insignificant. E/A ratio in COPD and control subjects was 0.980 ± 0.152 and 1.480 ± 0.345 respectively. The 't' value was -6.044 and 'p' value was significant (p < 0.01). Peak filling rates normalized to tricuspid stroke volume was calculated and compared in both COPD and control. Subjects. The PFR in both subjects was 4.350 ± 0.711 and 6.060 ± 0.843 respectively. The 't' value was -6.288 and 'p' value was significant (p < 0.01) (Table 1).

PAP has a strong negative linear correlation with RVEF (r = -0.92, p < 0.002), E/A (r = -0.74, p < 0.002) and PFR (r = -0.75, p < 0.002) (Table 2).

PAP has a strong linear correlation with RVWtd (r = 0.83, p < 0.002) (Table 2).

Discussion

Because of high compliance of RV, the ability of the RV to increase wall tension and systolic ejection pressure is limited. An absolute rise in RV afterload increases RVEDP and decreases RVEF and may lead to a fall in RV output. When pulmonary hypertension develops gradually as in patients with COPD, the RV is able to adapt to the increase in workload. As PVR rises, there is an increase in the density and the number of mitochondria in the myocytes of the RV free wall. In early stages, hypertrophied RV becomes less compliant and wall

Table 2: Correlation between PAP and right ventricular systolic and diastolic dysfunctions measured by 2D ECHO

Sample size	Variables	Correlation coefficients	P value
135 (100+35)	PAP and RVEF	-0.92	0.002
135 (100+35)	PAP and RVWtd	0.83	0.002
135 (100+35)	PAP and E/A	-0.74	0.002
135 (100+35)	PAP and PFR	-0.75	0.002

tension and contractility increases. The structural and functional adaptation of the RV to increase in afterload caused by disorders of the respiratory systems and commonly referred to as cor-pulmonale.

The prevalence of RV dysfunction increases with severity of pulmonary artery hypertension. As PAP increases, RVEF falls and ratio of early/ late peak atrial filling is reduced.

The pathogenesis of RV dysfunction in COPD is multifactorial but studies demonstrating a close inverse correlation between PaO₂ and mean PAP indicate that hypoxic pulmonary vasoconstriction (HPV) and subsequent remodelling of the pulmonary vascular bed undoubtedly play the greatest role. Normally HPV diverts blood flow from areas of regional alveolar hypoxia to better ventilated areas of the lung, thereby optimizing ventilation perfusion relationship. When alveolar hypoxia involves the entire lung, however as occurs during hypoventilation or exposure to high altitude, HPV results in increased PAP and PVR. In COPD, HPV is followed by medial hypertrophy of muscular pulmonary arteries and proliferation of vascular smooth muscle into normally non-muscular vessels of the pulmonary circulation. This remodelling of the vascular bed rises PVR and contributes to the development of PHT.

COPD affects RV functions through mechanisms other than HPV and vascular remodelling. Destruction of lung parenchyma decreases the cross sectional area of the pulmonary capillary bed. Patients with COPD may have increased serum viscosity from secondary polycythemia and prove to develop chronic pulmonary thromboembolic disease. Finally, increased intrathoracic pressure related to air trapping in the lower lobe may

increase RV afterload by compressing pulmonary vessels.

Determinants of right ventricular systolic functions based on the concept that ventricular functions are determined by preload, afterload and contractility. A physiologically correct definition for afterload is systolic wall tension. Component of afterload are PAP, total pulmonary resistance, RV wall thickness and ventricular volume.

Correlation of RVEF and PAP: Our data showed a strong negative linear correlation ($r=-0.92$, $p<0.002$) between pulmonary artery pressure and RVEF that closely parallels the findings of other recent observations of RV functions. Steven M. Kawut et al 2009 found RVEF was inversely correlated with pulmonary vascular resistance ($r=-0.51$; $p<0.001$). Schuler et al, conducted a study in 1978, and obtained a comparable correlation ($r=-0.75$) between RVEF and PAP. Similarly, Ellis et al¹ and Lewis M D et al observed a significant decrease in PAP and a concomitant increase in RVEF after low flow oxygen therapy. Similarly, Korr K. K. et al² in 1981, conducted a study and observed a significant negative linear correlation between RVEF and mean PAP ($r=-0.82$).

Correlation of PAP and RVWT: RVWT was significantly higher (0.970 ± 0.226) in patients COPD and it was significantly correlated with PAP ($r=0.83$, $p<0.002$). In COPD when PAP and PVR rises it leads to increase afterload on the RV, this chamber dilates and hypertrophies. To maintain cardiac output, the RV, by Frank starling mechanism, enlarges and hypertrophy develops. Our study closely parallels the study conducted by Cacho A. et al who concluded that 2D ECHO is useful in diagnosing RVH.

The mechanisms for the abnormalities of RV diastolic filling in COPD have not been clearly delineated. In patients with normal to

mild increased in Right ventricular free wall thickness (RVFWT), the relaxation process may be altered to affect calcium fluxes or to cause non uniformity between contraction and relaxation of myocardial cells, resulting is delayed early ventricular filling and prominent filling with atrial contraction as a compensatory mechanism in contrast to patients with moderate to severe increase in RVFWT leads to firm, poorly compliant Right ventricle, resulting to rapid early filling and then subsequent restriction to filling.

This study shows that RV diastolic function in altered in stable moderate to severe COPD, Peak filling rate was lower (4.350 ± 0.711) which indicates prolonged myocardial relaxation (mishimura RA et al 1989). Increase in peak velocity of atrial filling and reversal of E/A ratio in 15 patients (60%) indicates increased atrial contribution to ventricular filling. These parameters show that RV diastolic function is altered and is compensated by atrial filling.

Pulmonary artery pressure has a strong negative linear Correlation with E/A ($r=-0.74$, $p<0.002$) and PFR ($r=-0.75$, $p<0.002$).

Summary and Conclusion

RV Systolic function (RVEF and RVWT) are significantly abnormal in patients of stable compensated COPD and they are significantly correlated with PAP.

RV diastolic function i.e., E/A ratio and PFR are altered in 60% of patients of COPD studied against control subjects and significantly correlated with PAP.

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