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Review article

The echocardiographic features of left ventricular dysfunction in men with chronic obstructive pulmonary disease

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Abstract

Introduction: Left ventricular dysfunction (LVD) in chronic obstructive pulmonary disease (COPD) increases the risk of mortality. The coexistence of LVD and COPD has substantial number of diagnostic difficulties. It is still not clear how prevalent is it and what causes LVD in COPD patients without cardiovascular diseases. The aim of our study was to assess the prevalence of LVD and its association with the severity of the COPD.

Methods: The prospective cohort study included 120 patients with previously diagnosed stable COPD. In all patients spirometry and transthoracic echocardiography were performed. Patients were divided into four stages of COPD, according to Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria.

Results: The prevalence of LVDD across the cohort was 65.8%. The first degree of DD was found in 42.5% of COPD patients, second degree in 20.8% and third degree DD in 2.5% of patinets. LVDD was present in all COPD stages, but was not associated with the severity of the COPD disease ($p = 0.8$). The parameters of systolic function are decreasing with COPD stage, with significant differences between groups (SVI ($p=0.01$), EF $p<0.001$). Decreased EF≤49% was found in the 16.6% of cochort, in the first COPD stage 13.3% vs. 10% in GOLD II vs. 20% in GOLD III and 23.3% of patients in GOLD IV.

Conclusion: Undiagnosed LVD in COPD patients without previously diagnosed CVD was highly prevalent. These results highlight the need for regular echocardiography screening to reduce morbidity and mortality in these patients.

Kew words chronic obstructive pulmonary disease, left ventricular dysfunctions, echocardiography

Introduction

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of mortality globaly[1]. COPD and cardiovascular (CV) disorders are very common combination, as they share the same risk factors. Small airway obstruction plays the most important patophysiological role in COPD and represents an important risk factor for cardiac impairment[2]. Patients with mild airflow limitation have more chance to die from CV event than respiratory failure[3]. The forced expiratory volume in the first second (FEV1) is a well established predictor of cardiovascular morbidity and mortality[4]. Hypoxia and systemic inflammation go along with COPD and may lead to ventricular dysfunction[5]. The coexistence of COPD and heart failure (HF) has a number of diagnostic and therapeutic pitfalls and increases the mortality risk[6,7].

The association between left ventricular dysfunction (LVD) and COPD has not been well studied. Conflicting results suggest a prevalence of LVD between 9-52% in COPD patients[8]. The presence of even subclinical diastolic LV dysfunction raises the risk of all cause mortality in COPD[9]. Importantly, when assessing the LV function large amount of studies did not exclude patients with cardiovascular comorbidities such as ischemic heart disease or hypertension that influence the left ventricular function regardless of COPD. The aim of our study was to asses the prevalence of left ventricular dysfunction in stable COPD patients without formerly diagnosed cardiovascular disorders that may influence ventricular function. We also aimed to investigate weather left ventricular dysfunction is associated with COPD severity.

Methods

We performed a prospective cohort study that was conducted at the Institut of pulmonary diseases diseases of Vojvodina, Sremska Kamenica, Serbia. We included 120 male patients who were previously diagnosed with

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COPD, according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria. Patients were divided into four stages of COPD considering the FEV1 and arterial blood gas values. We excluded patients with any serious illness previously diagnosed or treated: pulmonary diseases (interstitial, tromboembolic, bronchial carcinoma), pulmonary artery hypertension, CV disorders (coronary artery disease, significant valvular stenosis or regurgitation, prior cardiac surgery, history of heart failure, arterial hypertension, atrial flutter and fibrillation, signs of left ventricular hypertrophy or left brunch bundle block in electrocardiogram).

All patients underwent clinical history assessment, anthropometric measurements (body weight, body height), blood pressure measurement, pulmonary function testing, electrocardiography, and transthoracic echocardiography. The study was approved by the Institutional Ethics committees, according to the Declaration of Helsinki, and informed consent was obtained from all participants. Assasment of the pulmonary function testing included spirometry, total body plethysmography and arterial blood gas analysis. Spirometry was performed according to 2005 ATS/ERS criteria[10]. A postbronchodilator values for forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), and the FEV1/FVC ratio were used according to GOLD recomendations.

Standard two-dimensional echocardiography were performed using echocardiographic instrument GE Vivid, using a probe of 2.5 MHz included a long parasternal, short parasternal apical and subcostal echocardiographic view. Results were analyzed by an echo-cardiologist blinded to all clinical data. We used a two-dimensional (2D), M-mode, Continuous Doppler (CW), Pulsed Doppler (PW), color and tissue Doppler according to the American Society of Echocardiography guidelines[11]. Patients were placed in the position of left lateral decubitus and detailed evaluation of the cardiac chambers were done. The following measurements were made in 2D echocardiography from a parasternal long-axis view: interventricular septum in diastole thickness (IVS), left ventricular end-diastolic diameter (LVEDD), left ventricule end-sistolic diametar (LVESD), posterior wall in diastole thickness (PWT), right ventricular diameter (RVD) and left atrium (LA) diametar in end sistoly.

From apical four, two, and three-chamber views we calculated left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), and left atrial maximal volume (LAVS) using the biplane summation of disks method. The left ventricular ejection fraction (EF) was calculated using the modified Simpson's method. Cardiac dimensions were indexed for BSA. Pulsed wave Doppler parameters were obtained from the apical 4-chamber view at the tip of the mitral leaflets. The peak early diastolic (E) and late diastolic (A) mitral flow velocities and E/A ratio was calculated. Pulsed tissue Doppler of the septal and lateral mitral annulus were acquired from the apical four-chamber view. Peak early diastolic mitral annular velocities (e') were measured both at the septal and lateral annulus. The ratio for E/e' was calculated, both for the septal and the lateral wall, and with e' averaged from the septal and lateral wall. A comprehensive approach was used to diagnose and grade the left ventricular diastolic dysfunction (LVDD) according to the recommendations of the ASE 2016[12]. The pulmonary artery systolic pressure (PASP) was assessed using Bernoulli equation from the jet of the tricuspid regurgitation (TR), while right atrial pressure was obtained from imaging of the inferior vena cava in the subcostal view.

Statistical analysis

Data were collected through a standardized questionnaire and verified by the author, coded and entered into a specialy designed database.

We calculated frequencies, percentages, mean, standard deviation. For variables with normal distribution comparison between the COPD group was performed using analysis of variance, followed by, if necessary, Tukey's multiple comparison test. For variables with normal distribution comparison was performed using the Kruskal Valis test, followed by multiple comparison medium rang test. Rate of correlation was calculated by Spearman's correlation. The statistical software Statistica (Statistica 13.5, The Ultimate Academic Bundle, StatSoft Europe GmbH, Hamburg, Germany; university license for the University of Novi Sad) was used for all analyses.

Results

The study included 120 men, mean age 59.78 ± 7.46 years old. Patients were classified according to GOLD classification to GOLD I, II, III and IV stage. The details of characteristics among different stages are shown in Table 1. Age did not differ statistically between the groups. The BSA decreased in severe COPD, with a significant difference between stage I and IV (p=0.02).

The details of echocardiographic parametars in all groups are presented in Table 2. There is a trend of diasto-

Table 1. Anthropometric and clinical characteristics between groups

Variables	COPD I (*n* = 30)	COPD II (*n* = 30)	COPD III (*n* = 30)	COPD IV (*n* = 30)	*p*-Value
Age . years Mean ± SD	56.90 ± 8.9	57.90 ± 7.4	62.53 ± 6.9	61.76 ± 4.6	0.09
BSA	1.98 ± 0.28	1.94 ± 0.20	1.85 ± 0.17	1.82 ± 0.16	**0.002**
BMI	25.70 ± 4.3	26.33 ± 6.6	23.56 ± 4.2	23.4 ± 4.9	0.09
SBP	123.17±7.25	120.50±10.53	121.17±9.97	121.33±9.00	0.53
DBP	76.33±5.56	76.33±8.80	75.67±6.66	75.83±6.44	0.93
FEV1	93.87± 10.1	62.8 ± 7.7	33.32 ± 7.3	28.13 ± 5.9	**<0.001**

BSA - body surface area, BMI - body mass index, SBP - systolic blood pressure, DBP - diastolic blood pressure, FEV1 - forced expiratory volumen in 1 second

Table 2. Echocardiographic characteristics of patients across the COPD groups

Variables	**COPD I (*n* = 30)**	**COPD II (*n* = 30)**	**COPDIII (*n* = 30)**	**COPD IV (*n* = 30)**	***p*-Value**
LAVSI (ml)	32.87 ± 5.51	33.93 ± 4.98	33.47 ± 5.06	34.69 ± 5.38	0.66
E (m/sec)	0.79 ± 0.21	0.65 ± 0.19	0.58 ±0.15	0.60 ± 0.16	0.00
A (m/sec)	0.80 ±0.18	0.81 ± 0.19	0.83 ± 0.26	0.90 ± 0.25	0.72
E/A	1.07 ± 0.27	0.75 ± 0.13	0.69 ± 0.11	0.71 ± 0.09	0.00
E'(m/sec) average	0.10 ± 0.03	0.08 ± 0.03	0.09 ±0.03	0.09 ± 0.02	0.14
A'(m/sec) average	0.12 ±0.06	0.12 ± 0.04	0.13 ± 0.13	0.12 ± 0.05	**0.94**
E'/A' average	0.99 ± 0.55	0.71 ± 0.19	0.88 ± 0.33	0.78 ± 0.27	**0.02**
E/E' ratio	7.09±2.71	7.56±2.66	7.95±3.51	8.19±3.28	**0.05**
LVESd (mm)	31 ± 6.19	31.07 ± 4.68	32.33 ± 4.96	35.83 ± 8.47	**0.02**
LVEDd (mm)	43.60 ± 6.16	47.57 ± 4.74	47.10 ± 4.25	49.47 ± 7.86	**0.01**
IVSd (mm)	10.77 ± 1.36	10.12 ± 1.08	10.27 ± 1.06	10.5 ± 1.31	0.15
PWd (mm)	10.98 ± 1.29	10.48 ± 1.53	10.32 ± 0.97	10.43 ± 1.25	0.09
EDVI (ml/m2)	56.50 ± 15.62	52.35 ± 12.99	55.34 ± 11.31	58.53 ± 15.60	0.39
ESVI (ml/m2)	23.91 ± 8.93	23.74 ± 6.83	24.13 ± 5.65	31.57 ± 13.38	**0.03**
SVI(ml/m2)	32.58 ± 7.98	28.62 ± 7.00	31.21 ± 7.51	26.96 ± 6.88	**0.01**
EF %	60.03 ± 5.42	55.40 ± 5.35	56.50 ± 5.82	50.03 ± 9.25	**<0.001**
RV (mm)	27.30 ± 3.01	26.57±3.43	29.06±3.72	30.07±4.21	**<0.001**
RVSP	23.31 ± 7.08	26.24 ± 6.72	29.64 ± 10.31	39.20 ± 15.93	**<0.01**
TAPSE	2.03 ±0.32	2.07±0.31	1.79±0.36	1.7±0.34	**<0.001**

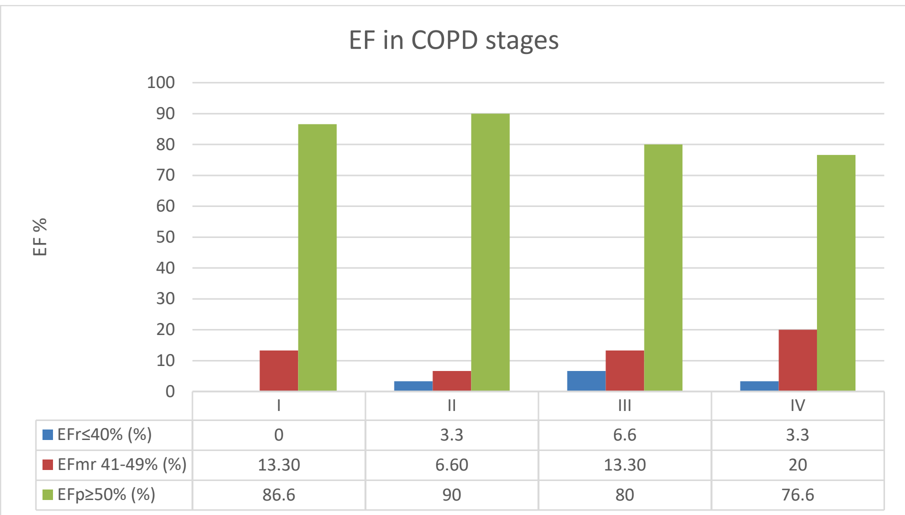
COPD - chronic obstructive pulmonary disease, LAVSI - left atrial volume systolic index, LVEDd - left ventricular end-diastolic diameter, LVESd - left ventricule end-sistolic diametar, IVSd - interventricular septum in diastole thickness, PWd - posterior wall in diastole thickness, EDVi - left ventricular end-diastolic volume index, ESVI - left ventricular end-systolic volume index, SVI - stroke volume index, EF - ejection fraction, RV - right ventricle

lic dysfunction imparment with COPD stage. There were stasticaly significant differences between group in the E vawe (p <0.001), E/A ratio p<0.001, E'/A' ratio p=0.02 and E/E' ratio p=0.05.

There are statistically significant differences between groups in left ventricular systolic (LVESd p=0.02) and diastolic diameters (LVEDd p=0.01) with trend of chamber dilatation increasing with COPD stage. The parameters of systolic function are decreasing with COPD stage, with significant differences between groups (SVI (p=0.01), EF p<0.001). Decreased EF≤49% was found in the 16.6% of cochort, in the first COPD stage 13.3% vs. 10% in GOLD II vs. 20% in GOLD III and 23.3% of patients in GOLD IV.

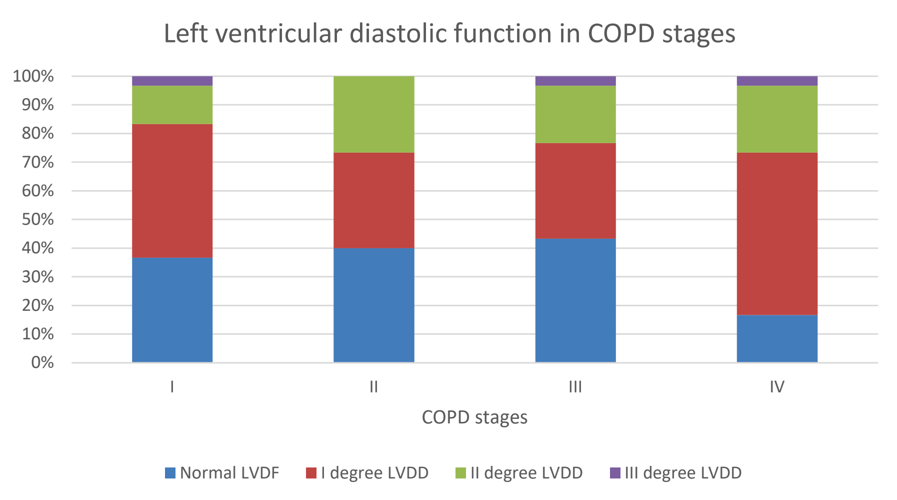
Grading of LVDD across COPD stages is presented in figure 2. The prevalence of LVDD across the cohort was 65.8%. The first degree of DD was found in 42.5% of COPD patients, second degree in 20.8% and third degree DD in 2.5% of patinets. LVDD was present in all COPD stages, but was not associated with the severity of the COPD disease (p = 0.8).

Figure 1. Ejection fraction across the spectum of COPD stages



EF - ejection fraction, COPD - chronic obstructive pulmonary disease

Figure 2. Left ventricular diastolic function across COPD stages



COPD - chronic obstructive pulmonary disease, LVDF - left ventricular diastolic function, LVDD - left ventricular diastolic disfunction

Discussion

Our study showed a high prevalence of unknown ventricular dysfunction in patients with COPD in patient without history of cardiovascular diseases.
LVD in COPD has been described in earlier studies but the prevalence varies widely[9,13-18]. This wide variation is in part due to the dissimilar studied populations' characteristics, and in part due to the different echocardiographic methods used for the assessment of LV diastolic dysfunction[19]. Our study showed that early COPD stages are more prone to have left ventricular diastolic dysfunction, while severe COPD is associated with both left ventricular systolic and diastolic dysfunction. We found no correlation between the severity of COPD and LVDD. Similiar to our results the study from Freixa et al. showed that echocardiographic abnormalities in COPD patients were unrelated to COPD severity[16]. In the same way, there was no relation of pulmonary function parameters to pulmonary artery pressure and diastolic function parameters in study from Ozer et al[15].
The pathophysiology and relationship between LVD and COPD is complex and involves a lot of factors including hypoxia, neurohumoral activation, endothelial dysfunction, inflammation/oxidative stress, ventricular interdependence, hyperinflation/emphysema and coronary artery disease (CAD)[20].
Alveolar hypoxia and consequent hypoxemia causes endothelial dysfunction, arterial stiffness, blood pressure raise[21,22] and triggers LV relaxation impairment. Experimental research in healthy subjects submitted to long periods of altitude induced hypoxia has shown a marked decrease in LV preload, owing to a reduction in their plasma volume[23].
Left and right ventricle are anatomically and physiologicaly bonded, they share pericardium and ventricular septum. Changes in right ventricular (RV) functions may influence left ventricular functions. Ventricular interdependence is a significant phenomenon in patients with COPD[20]. RV structural changes and remodeling with RV subclinical dysfunction is seen even in mild COPD. Pulmonary vascular changes and intermittent increases in pulmonary artery pressure (PAPs) that occur during exercise and/or sleep in early stages of COPD cause RV wall stress, thickening and hypertrophy[24]. RV concentric hypertrophy decreases RV enddiastolic volume and alters right ventricular and left ventricular diastolic function. Dilating RV hypertrophy is usually seen in severe stages of COPD, that has also been seen in our study.
High prevalence of LVDD is described in pulmonary emphysema which is usually seen in severe COPD. Watz et al. found that abnormal LV filling is a consequence of chest hyperinflation in patients with COPD and showed that LVDD is not related to LV myocardial malfunction but rather to a reduced preload[25]. Lung volume reduction surgery showed improvement in the LVD function in patients with emphysema, supporting that the changes in the LVD function are not due to myocardial malfunction[26]. In study from Barr et al severe airflow obstruction were linearly related to impaired left ventricular filling, reduced stroke volume, and lower cardiac output without changes in the ejection fraction. Impared diastolic function is surrogate for decreased left ventricular preload. Reduced preload affect stroke volume and ejection fraction. There are multiple mechanisms affecting left ventricular systolic function in COPD patients[17].
Sistolic left ventricular dysfunction was only present in severe stages of COPD in our study. The LVSD prevalence differs significantly in patients with stable COPD. From 0%–16% in COPD patients without previously diagnosed cardiovascular diseases[7] and from 8-26.7% in all COPD population[9,16,27-28]. LVSD is associated with the severity of COPD i.e., more severe COPD is more likely to have LV systolic dysfunction[27,29], which is consistent with our results. Some investigators postulated that CAD which is not proved with conventional methods may be the cause for LVSD in COPD[30].
COPD is a progressive disease, and with slow chronic airflow limitation both left ventricle (LV) and right ventricle (RV) and their systolic and diastolic function[31] are afected. Signs and symptoms of LV failure can be difficult to differentiate from those of COPD. This is partly due to similar clinical manifestations but mainly due to lack appropriate diagnostic tests to diagnose HF and/or COPD[32].

HF in COPD is an independent predictor of hospitalizations and death. Subclinical diastolic dysfunction can be an early predictor of left ventricular dysfunction and heart failure[9]. Both LVDD and LVSD have increased mortality. So even mild stages can be predictor of poor prognosis[9,28]. The results of our study demonstarted that significant portion of COPD patients with no prior history of cardiovascular diseases have LVDD, even among mild COPD stages, suggesting that LVDD is vastly underdiagnosed in COPD which can have a detrimental effect on long term outcomes of these patients. Still, it is not clear which COPD patients are at risk for LVDD. In the same line, about 16.6% of COPD patients had EF<50%, and no previous history of CVD, however high symptom burden could likely obscured the diagnosis.

Our study has several limitations. First, we included a relatively small number of patients which may preclude some statistical analysis. Second, we compared echocardiographic features with spirometric classification of COPD, as the study was designed before the updated simptom/risk GOLD classification of COPD. Finally, patients treated in University hospitals may not represent the general population.

Conclusion

High prevalence of unrecognized ventricular dysfunction in COPD patients with no history of CVD highlights the need for echocardiography in these patients. We may postulate that in mild COPD stages intermitent hypoxia and sistemic inflamation play a major role in LVDD. Ventricular dysfunction may begin to develop from the begining in the progress of pulmonary disease and might stay sub-clinical for a long time.

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Sažetak

Ehokardiografske osobine disfunkcije leve komore kod muškaraca sa hroničnom opstruktivnom bolesti pluća

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Uvod: *Disfunkcija miokarda leve komore (LVD) kod bolesnika sa hroničnom obstruktivnom bolesti pluća (HOBP) povećava rizik od mortaliteta. Istovremeno postojanje LVD i HOBP nosi veliki broj dijagnostičkih poteškoća. Još uvek nije jasno kolika je učestalost i šta dovodi do LVD u HOBP kod bolesnika bez prethodne kardiovaskularne bolesti. Cilj studije je bio odrediti učestalost disfunkcije miokarda leve komomre i njenu povezanost sa stepenom težine HOBP.*
Metodologija: *Prospektivna kohortna studija je uključila 120 bolesnika sa prethodno dijagnostikovanom stabilnom hroničnom obstruktivnom bolesti pluća. Svim bolesnicima je urađena spirometrija i ehokardiografija. Bolesnici su podeljeni u četiri stadijuma HOBP prema kriterijumima globalne inicijative za HOBP (GOLD).*
Rezultati: *Prevalencija dijastolne LVD u celoj kohorti bila je 65.8%, prvi stepen nađen je kod 42.5% pacijenata sa HOBP, drugi stepen kod 20.8%, a treći stepen kod 2.5% pacijenata, bez statistički značajne povezanosti sa težinom HOBP bolesti (p = 0.8). Sistolna funkcija opada sa stadijumom HOBP, sa značajnim razlikama između grupa (SVI (p=0,01), EF p<0,001). Smanjenje EF≤49% nađeno je u 16.6% kohorte, u prvoj fazi HOBP 13.3% naspram 10% u GOLD II prema 20% u GOLD III i 23.3% pacijenata u GOLD IV.*
Zaključak: *Veliki broj bolesnika bez prethodne srčane bolesti je imao neprepoznatu disfunkciju leve komore. Ovi rezultati naglašavaju važnost ehokardiografije u skreeningu radi pravovremene dijagnostike i lečenja u cilju sprečavanja morbiditeta i mortaliteta ovih bolesnika.*

Ključne reči: hr*onična opstruktivna bolest pluća, disfunkcija leve komore, ehokardiografija*