

Elastic properties of pulmonary artery in chronic obstructive pulmonary disease

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Today chronic obstructive pulmonary disease (COPD) is one of the most common diseases with specific pulmonary vascular changes.

The aim – to evaluate elastic properties of pulmonary artery (PA) and pathogenic mechanisms of disorders in COPD.

Materials and methods. Participants were 50 patients with COPD stages 1–3 without comorbidities (32 men and 18 women, average age was 49.8 ± 1.0 years). Control group included 30 healthy people (19 men and 11 women, aged 50.1 ± 1.2 years). PA elastic properties was researched by ultrasound method. Statistical analysis was performed by means of the Statistica® 6.0 for Windows (StatSoft Inc.) software using parametric and nonparametric methods.

Results. Study data showed that pulmonary arterial pressure (PAP) and PA elastic properties were significantly different between subjects with COPD and control group. Thus, pulsatility, compliance and distensibility in COPD were decreased ($39.23 \pm 1.6\%$, 6.4 ± 0.4 mm²/mm Hg and $1.71 \pm 0.10\%$ mm Hg versus $51.4 \pm 1.9\%$, 11.1 ± 0.5 mm²/mm Hg and $3.30 \pm 0.12\%$ mm Hg in control group, respectively, $p < 0.05$), and elastic modulus and index stiffness B were increased (65.7 ± 3.7 mm Hg and 2.91 ± 0.17 to 31.6 ± 1.2 mm Hg and 2.05 ± 0.08 , versus 31.6 ± 1.2 mm Hg and 2.05 ± 0.08 in control group, respectively, $p < 0.05$). Analysis in groups divided by severity of COPD showed that PA elastic properties was not different significantly between subjects with COPD stage-1 and control group. However, several significant differences in PAP and PA elastic properties between subjects with COPD stage-2, COPD stage-3 and control group were found ($p < 0.05$). Pearson correlation analysis was showed significant relationships between indexes of PA elastic properties and FEV₁, indexes of PAP.

Conclusions. The changes of PA elastic properties in COPD are accompany by increasing stiffness, thus reduce pulsatility, compliance and elasticity of vascular wall. Detected changes of PA elastic properties were associated with disease progression, airflow limitation and pulmonary hypertension, most significantly in COPD stage-3.

Ключові слова:

хронічне обструктивне захворювання легень, легенева артерія, пружно-еластичні властивості.

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Пружно-еластичні властивості легеневої артерії при хронічному обструктивному захворюванні легень

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Хронічне обструктивне захворювання легень (ХОЗЛ) залишається одним із поширених захворювань, що супроводжується змінами легеневих судин.

Мета роботи – оцінити пружно-еластичні властивості легеневої артерії та патогенетичні механізми їхніх порушень у хворих на ХОЗЛ.

Матеріали та методи. Обстежили 50 хворих на ХОЗЛ 1–3 стадії без супутніх захворювань (32 чоловіки та 18 жінок), середній вік яких – $49,8 \pm 1,0$ року. До групи контролю увійшли 30 здорових осіб (19 чоловіків, 11 жінок) віком $50,1 \pm 1,2$ року. Визначення показників пружно-еластичних властивостей легеневої артерії здійснювали ультразвуковим методом. Статистичне опрацювання результатів виконували з використанням програми «Statistica® 6.0 for Windows» (StatSoft Inc.) із застосуванням параметричних і непараметричних методів.

Результати. У хворих на ХОЗЛ спостерігали суттєві порушення пружно-еластичних властивостей легеневої артерії у вигляді зменшення індексу пульсації, піддатливості, розтяжності (до $39,2 \pm 1,6\%$, $6,4 \pm 0,4$ мм²/мм рт. ст. та $1,71 \pm 0,10\%$ мм рт. ст. проти $51,4 \pm 1,9\%$, $11,1 \pm 0,5$ мм²/мм рт. ст. і $3,30 \pm 0,12\%$ мм рт. ст. у здорових осіб відповідно, $p < 0,05$) і збільшення еластичного модуля, індексу жорсткості В (до $65,7 \pm 3,7$ мм рт. ст. і $2,91 \pm 0,17$ проти $31,6 \pm 1,2$ мм рт. ст. і $2,05 \pm 0,08$ у здорових осіб відповідно, $p < 0,05$). Аналіз пружно-еластичних властивостей ЛА залежно від стадії ХОЗЛ довів збільшення подібних змін поряд із прогресуванням бронхообструктивних порушень найсуттєвіше у групі ХОЗЛ 3 стадії. За даними кореляційного аналізу виявлені щільні зв'язки між показниками пружно-еластичних властивостей легеневої артерії та величинами артеріального тиску в легеневій артерії, більш суттєві з боку систолічного та пульсового артеріального тиску.

Висновки. Перебіг ХОЗЛ супроводжується порушеннями пружно-еластичних властивостей легеневої артерії у вигляді зменшення пульсативності, піддатливості, розтяжності та збільшення жорсткості, ступінь яких пов'язаний із величинами бронхообструктивних порушень і виразністю легеневої гіпертензії.

Ключевые слова:

лёгочная артерия, упруго-эластические свойства, хроническое обструктивное заболевание лёгких.

Упруго-эластичные свойства лёгочной артерии при хроническом обструктивном заболевании лёгких

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Хроническая обструктивная болезнь лёгких (ХОБЛ) остаётся одним из распространённых заболеваний, которое сопровождается изменениями лёгочных сосудов.

Цель работы – оценить упруго-эластические свойства лёгочной артерии и патогенетические механизмы их нарушений у больных ХОБЛ.

Материалы и методы. Обследовано 50 больных ХОБЛ 1–3 стадии без сопутствующих заболеваний (32 мужчины и 18 женщин), средний возраст которых составил $49,8 \pm 1,0$ года. В группу контроля вошли 30 здоровых человек (19 мужчин и 11 женщин) в возрасте $50,1 \pm 1,2$ года. Определение показателей упруго-эластичных свойств лёгочной артерии проводили методом эхокардиографии. Статистическая обработка полученных результатов проводилась с использованием программы «Statistica® 6.0 for Windows» (StatSoft Inc.) с применением параметрических и непараметрических методов.

Результаты. У больных ХОБЛ наблюдались существенные нарушения упруго-эластичных свойств лёгочной артерии в виде уменьшения индекса пульсации, податливости, растяжимости (до $39,2 \pm 1,6\%$, $6,4 \pm 0,4$ мм²/мм рт. ст. и $1,7 \pm 0,10\%$ /мм рт. ст. против $51,4 \pm 1,9\%$, $11,1 \pm 0,5$ мм²/мм рт. ст. и $3,30 \pm 0,12\%$ /мм рт. ст. у здоровых людей соответственно, $p < 0,05$) и увеличение эластичного модуля и индекса жёсткости (до $65,7 \pm 3,7$ мм рт. ст. и $2,91 \pm 0,17$ против $31,6 \pm 1,2$ мм рт.ст. и $2,05 \pm 0,08$ у здоровых людей соответственно, $p < 0,05$). Анализ упруго-эластичных свойств ЛА в зависимости от стадии ХОБЛ показал увеличение подобных изменений наряду с прогрессированием бронхообструктивных нарушений, при этом более значимо в группах ХОБЛ 2 степени и ХОБЛ 3 степени. По данным корреляционного анализа выявлены тесные связи между показателями упруго-эластичных свойств лёгочной артерии и величинами артериального давления в лёгочной артерии, наиболее существенные со стороны показателей систолического и пульсового давления.

Выводы. Течение ХОБЛ сопровождается нарушениями упруго-эластичных свойств лёгочной артерии в виде уменьшения пульсативности, податливости, растяжимости и увеличения жёсткости, степень которых связана с величинами бронхообструктивных нарушений и выраженностью лёгочной гипертензии.

Today chronic obstructive pulmonary disease (COPD) is one of the common diseases and observed in 9–10% adult in many countries, including Ukraine [1]. COPD characterized by progressive, complicated clinical course and is the fourth leading cause in mortality. The main pathogenic factors of adverse course in COPD are airflow limitation, and pulmonary vascular changes. Functional decline is often associated with vascular remodeling and reduction of the pulmonary vascular bed leading to pulmonary hypertension and pulmonary heart progression with heart failure [2,3].

In the last decade, the elastic properties of the pulmonary artery (PA) and their impact on the course of COPD attracted the attention of researchers. Obvious information about PA stiffness associated with the severity of COPD [4], obstructive respiratory disorders and pulmonary hypertension [5]. It is proved, that the violation of elastic properties of PA adversely affects the structural and functional condition of pulmonary vessels and leads to right ventricular overload [6]. Furthermore, Bhatt SP. et al. observed prognostic changes of PA pulsatility in COPD significantly associated with pulmonary hypertension and adversely clinical course [7]. So, the study of PA elastic properties in COPD remains relevant for both scientific and practical medicine.

Aim of the study

To evaluate the elastic properties of the pulmonary artery and pathogenetic mechanisms of disorders in patients with COPD.

Materials and Methods

Participants were 50 patients with COPD stages 1–3 without comorbidities (32 men and 18 women, average age was 49.8 ± 1.0 years). Control group included 30 healthy people (19 men and 11 women, aged 50.1 ± 1.2 year).

PA elastic properties were researched by ultrasound method [4] and calculated following indexes: pulsatility (Puls, %), compliance (Comp, мм²/мм Hg), distensibility (Dist, %/мм Hg), elasticity module (EM, мм Hg) and stiffness index B (SI–B). Mean pulmonary arterial pressure (PAP) (mPAP, мм Hg) was calculated by Kitabatake equation, systolic PAP (sPAP, мм Hg) was calculated by tricuspid regurgitation, diastolic PAP (dPAP, мм Hg) and pulse PAP

(pPAP, мм Hg) counted as derived from mPAP and sPAP. Forced expiratory volume in 1 s (FEV₁) was measured by spirometry after inhalation bronchodilator tests.

Data were analyzed using “Statistica” (v. 6.0, StatSoft Inc, USA). Comparison of variables between groups was established using independent samples t-test or Mann-Whitney U test (for data with normal or abnormal distribution, respectively). Pearson correlation was used for variable pairs with linear distribution. Statistical significance among variables was considered a two-tailed p value of less than 0.05.

The results and discussion

Study data showed that PAP and PA elastic properties between subjects with COPD and control group were materially differing (Table 1). So, all PAP indexes were significantly higher in COPD ($p < 0.05$). Simultaneously, EM and IS–B were increased significantly to 65.7 ± 3.7 мм Hg and 2.91 ± 0.17 in group with COPD versus 31.6 ± 1.2 мм Hg and 2.05 ± 0.08 in control group, respectively ($p < 0.05$). Conversely, Puls, Comp and Dist were decreased significantly to $39.23 \pm 1.6\%$, 6.4 ± 0.4 мм²/мм Hg and $1.71 \pm 0.10\%$ /мм Hg in COPD versus $51.4 \pm 1.9\%$, 11.1 ± 0.5 мм²/мм Hg and $3.30 \pm 0.12\%$ /мм Hg in control group, respectively ($p < 0.05$).

Analysis in groups divided by severity of COPD (Table 2) was showed FEV₁ naturally decreased in groups with COPD stage-1, COPD stage-2 and COPD stage-3 to $88.3 \pm 2.1\%$, $60.4 \pm 1.6\%$ and $39.7 \pm 1.9\%$, respectively,

Table 1. PAP and PA elastic properties in the examined groups (M ± m)

Indexes	Control group n=30	COPD n=50
sPAP, mm Hg	24.4 ± 0.7	$39.1 \pm 0.9^*$
mPAP, mm Hg	13.7 ± 0.3	$22.7 \pm 0.9^*$
dPAP, mm Hg	8.2 ± 0.3	$14.5 \pm 0.5^*$
pPAP, mm Hg	16.1 ± 0.8	$24.6 \pm 1.0^*$
Puls, %	51.4 ± 1.9	$39.23 \pm 1.6^*$
Comp, мм ² /мм Hg	11.1 ± 0.5	$6.4 \pm 0.4^*$
Dist, %/мм Hg	3.30 ± 0.12	$1.71 \pm 0.10^*$
EM, мм Hg	31.6 ± 1.2	$65.7 \pm 3.7^*$
IS–B	2.05 ± 0.08	$2.91 \pm 0.17^*$

*: likely differences between comparison group ($p < 0.05$).

Table 2. PAP and PA elastic properties distributed by the severity of COPD

Indexes	Control, n=30	COPD stage-1, n=12	COPD stage-2, n=20	COPD stage-3, n=18
FEV ₁ , %	98.8±1.2	88.3±2.1*	60.4±1.6**	39.7±1.9**
sPAP, mm Hg	24.1 (22.98;25.6)	26.1 (24.4;27.9)	37.8 (35.3;40.9)**	47.1(41.4;53.6)**
mPAP, mm Hg	13.8 (13;14.7)	15.2 (14.6;16.8)	23.1 (21.7;25.6)**	25.3(22.9;28.3)**
dPAP, mm Hg	8.2 (7.1;9.6)	8.8 (6.9;9.80)	15.1 (13.7;17.9)**	17.2 (15.2;20.8)**
pPAP, mm Hg	15.3 (13.4;17.7)	18 (16.7;20.3)	27.5 (25.3;28.8)**	30.1 (28.6;33.4)**
Puls, %	49.8 (45.5;55.3)	46.5 (42.7;50.3)	39.9 (35.4;44.5)**	35.5 (31.6;40.1)**
Comp, mm ² /mm Hg	10.8 (9.5;12.3)	8.7 (7.9;9.7)	6.44 (5.7;7.4)**	4.99 (3.9;5.5)**
Dist, %/mm Hg	3.2 (2.6;3.7)	2.8 (2.3;3.2)	1.7 (1.4;1.9)**	1.2 (0.9;1.4)**
EM, mm Hg	31.7 (27.5;36)	45.0 (39.7;51.2)	58.6 (51.1;66.8)**	80.2 (69.2;98.3)**
IS-B	2.1 (1.7;2.5)	2.2 (1.8;2.5)	2.3 (1.9;2.4)**	3.3 (2.3;3.4)**

*: likely differences compared with the control group (p<0.05); *: likely differences compared with COPD stage-1 (p<0.05); †: likely differences compared with COPD stage-2 (p<0.05).

Table 3. Correlation between indexes of PA elastic properties, blood pressure and FEV₁ (Pearson r, P value)

Indexes	Puls	Comp	Dist	EM	IS-B
FEV ₁	+0.43 (0.03)*	+0.40 (0.05)*	+0.40 (0.05)*	-0.40 (0.05)*	-0.41 (0.05)*
sPAP	-0.56 (0.001)*	-0.42 (0.03)*	-0.55 (0.001)*	+0.52 (0.01)*	+0.50 (0.01)*
mPAP	-0.51 (0.03)*	-0.41 (0.03)*	-0.44 (0.02)*	+0.47 (0.02)*	+0.42 (0.03)*
dPAP	-0.40 (0.04)*	-0.38(0.06)	-0.41 (0.03)*	+0.42 (0.03)*	+0.37 (0.07)
pPAP	-0.55 (0.001)*	-0.43 (0.03)*	-0.51 (0.01)*	+0.53 (0.001)*	+0.50 (0.01)*

*: p<0.05.

versus 98.8±1.2% in control group (p<0.05). In its turn, PAP and PA elastic properties was not different significantly between subjects with COPD stage-1 and control group. However, several significant differences in PAP and PA elastic properties between subjects with COPD stage-2, COPD stage-3 and control group were found (p<0.05). Thus, EM and IS-B in groups with COPD stage-2, COPD stage-3 were increased to 58.6 (51.1; 66.8) mm Hg, 2,3 (1.9; 2.4) and 80.2 (69.2; 98.3) mm Hg, 3.3 (2.3; 3.4) versus 31.7 (27.5; 36.0) mm Hg and 2.1 (1.7; 2.5) in control group, respectively (p<0.05). Conversely, Puls, Comp and Dist were decreased to 39.9 (35.4; 44.5)%, 6.44 (5.7; 7.4) mm²/mm Hg, 1.7 (1.4; 1.9) %/mm Hg and 35.5 (31.6; 40.1)%, 4.99 (3.9; 5.5) mm²/mm Hg, 1.2 (0.9; 1.4) %/mm Hg versus 49.8 (45.5; 55.3)%, 10.8 (9.5; 12.3) mm²/mm Hg and 3.2 (2.6; 3.7) %/mm Hg in control group, respectively (p<0.05).

Pearson correlation analysis was used to identify factors linearly correlated to indexes of PA elastic properties (Table 3). Thus, FEV₁ was inversely related to EM (r=-0.49; p=0.02), IS-B (r=-0.49; p=0.02) and directly related to Puls (r=+0.43; p=0.03), Comp (r=+0.40; p=0.05), Dist (r=+0.40; p=0.05). Conversely, all PAP indexes were inversely related to Puls, Comp and Dist, but directly related to EM and IS-B. Most significant relationships were founded between sPAP, pPAP and Puls (r=-0.56; p=0.001; r=-0.55; p=0.001, respectively), Dist (r=-0.55; p=0.001; r=-0.51; p=0.01, respectively). dPAP had least related to indexes of PA elastic properties.

Our received data coincide with previous studies [8] which had demonstrated that PA stiffness and distensibility were worsened in COPD patients and correlated with decreased functional capacity. It should be noted, that the most significant PA elastic properties changes we are observed in the later stages of COPD. In addition, the PA elastic properties were correlated well with the presence of pulmonary hypertension. Relationship between PA elastic properties and sPAP, pPAP suggests PA stiffness as a promising biomarker for early detection of pulmonary vascular disease, and, may be, to play a role in right ventricular failure in COPD. Similar relationships marked by Thenappan T. et al. [9] as early change in the disease process even when pulmonary artery pressure and pulmonary vascular resistance are normal.

Thus, present studies demonstrated violation of PA elastic properties in COPD with increased stiffness, thus reduce pulsatility, compliance and elasticity of vascular wall. Detected changes of PA elastic properties were associated with disease progression, airflow limitation and pulmonary hypertension, most significantly in COPD stage-3. More significant relationships between PA elastic properties violations and systolic, pulse pulmonary arterial pressure were founded.

Conclusion

1. Violation of pulmonary artery elastic properties in COPD characterized by increasing stiffness and thus reduce pulsatility, compliance and elasticity of vascular wall.
2. Changes of PA elastic properties in COPD is associated with disease progression, airflow limitation and pulmonary hypertension, most significantly in COPD stages-3.
3. Systolic and pulse pulmonary arterial pressure had most significant relationships with violations of PA elastic properties.

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