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Effects of asthma severity on cardiac remodeling in patients with arterial hypertension

A. A. Odegova [1], E. I. Tarlovskaya [2]

[1] Kirov State Medical Academy, Kirov, Russia
[2] Nizhny Novgorod State Medical Academy, Nizhny Novgorod, Russia

Corresponding author:
Alla A. Odegova,
Kirov State Medical Academy,
112 K. Marksa street, Kirov,
610027 Russia.
Phone: +7(8332)56–03–01,
+7(8332)62–77–28,
+7(8332)62–77–28.
E-mail: all.odegova@yandex.ru

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Abstract

Background. Bronchial asthma (BA) is a serious medical problem. The number of patients with the first manifestations at age older than 40–50 years has increased. The frequency of arterial hypertension (HTN) in BA patients was found to be about 30%. **Objective.** To reveal the characteristics of cardiac remodeling at different severity degrees of BA in hypertensive patients. **Design and methods.** Altogether 91 patients were enrolled in the study in 2008–2015 years, they presented with controlled BA of varying severity associated with 1–2 degree HTN. Of these, 26 patients (29%) had mild BA, 34 (37%) — moderate BA severity, 31 (34%) — severe BA. All examined subjects were outpatients and received adequately chosen basic BA therapy by inhaled corticosteroids. On demand, they used a β_2-agonists of short action. A group of patients with 1–2 degree HTN (n = 30), and a group of patients with different severity of controlled BA (n = 32) served as controls. All patients underwent echocardioscopy (Acuson 128XP/10c, USA). **Results and conclusions.** Left ventricular hypertrophy and diastolic dysfunction were found in all groups, but their number was greater in the group with comorbidities. The number of patients with hypertrophy and diastolic dysfunction was greater with the higher severity of bronchial obstruction in groups with 1–2 degree HTN. Systolic dysfunction was registered in none of the groups.

Key words: arterial hypertension, bronchial asthma, heart remodeling

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Влияние степени тяжести бронхиальной астмы на ремоделирование сердца у пациентов с артериальной гипертензией

А. А. Одегова[1], Е. И. Тарловская[2]

[1] Государственное бюджетное образовательное учреждение высшего профессионального образования «Кировская государственная медицинская академия» Министерства здравоохранения Российской Федерации, Киров, Россия
[2] Государственное бюджетное образовательное учреждение высшего профессионального образования «Нижегородская государственная медицинская академия» Министерства здравоохранения Российской Федерации, Нижний Новгород, Россия

Контактная информация:
Одегова Алла Андреевна,
ГБОУ ВПО «Кировская ГМА» МЗ РФ,
ул. К. Маркса, д. 112, г. Киров, Россия,
610027.
Тел.: +7(8332)56–03–01,
+7(8332)62–77–28,
+7(8332)62–77–28.
E-mail: all.odegova@yandex.ru

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Резюме

Актуальность. Бронхиальная астма (БА) является серьезной медицинской проблемой. В большинстве стран распространенность БА возрастает. Установлено, что частота обнаружения артериальной гипертензии (АГ) у лиц, страдающих БА, составляет около 30%. Ухудшение функции легких является столь же сильным предиктором сердечно-сосудистой летальности, как и основные кардиоваскулярные факторы риска. **Цель исследования** — выявление особенностей ремоделирования сердца при разной степени тяжести БА на фоне АГ. **Материалы и методы.** В исследовании 2008–2015 гг. участвовал 91 пациент с различной степенью тяжести БА в стадии контролируемости в сочетании с АГ 1–2 степени тяжести, из них 26 пациентов (29%) с легкой степенью тяжести БА, 34 (37%) со средней степенью тяжести БА, 31 (34%) — с тяжелой БА. Все обследованные пациенты были амбулаторными, получали адекватно подобранную базисную терапию БА ингаляционными глюкокортикостероидами. Испытуемые использовали β_2-адреномиметики короткого действия по потребности. Для сравнительного анализа была взята группа пациентов с АГ 1–2 степени (n = 30) и группа лиц с БА различных степеней тяжести в стадии контроля (n = 32). Всем испытуемым проводилась эхокардиоскопия на аппарате «Acuson 128XP/10c» (США). **Результаты и выводы.** Гипертрофия миокарда левого желудочка и диастолическая дисфункция были выявлены у пациентов всех исследуемых групп, но число их было больше в группе с сочетанной патологией. Доли испытуемых с гипертрофией и диастолической дисфункцией были тем больше, чем выше была тяжесть бронхообструктивного синдрома в группах при неизменной АГ 1–2 степени. Систолическая дисфункция не зарегистрирована ни в одной из групп данного исследования.

Ключевые слова: артериальная гипертензия, бронхиальная астма, ремоделирование сердца

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Introduction

Bronchial asthma (BA) is a serious global problem [1]. In most countries, the prevalence of BA is increasing [1]. BA is a significant burden associated with both high costs for treatment and mortality and disability [1]. The estimates show that 250,000 people die from BA annually, and in the Russian Federation (RF) mortality resulting from BA remains one of the highest worldwide [2]. While the average BA-related mortality, according to the data from 48 countries, amounts to 7.9 per 100,000 of the population, this ratio is 4-fold higher in the RF [2].

Myocardium remodeling in patients with coexistent HTN and bronchopulmonary pathology has high priority. The diagnostics of structural and functional cardiovascular changes in these patients is important to find the most efficient approaches to antihypertensive therapy and to evaluate their prognostic relevance regarding the risk of sudden death, arrhythmia, coronary heart disease and chronic heart failure [4, 5]. Remodeling of the left ventricle (LV) results from blood pressure (BP) elevation, changes in other hemodynamic, neurohumoral and genetic factors, and acts as an independent prognostic factor in arterial hypertension (HTN) [4, 10]. Despite the sufficient data on the prognostic significance of LV remodeling, current understanding of structural and functional remodeling of cardiovascular system in patients with coexistent HTN and BA is incomplete. The data on cardiac remodeling in patients receiving different treatment for bronchial obstructive syndrome in BA of various severity with concomitant HTN are insufficient. Optimization of treatment choice, assessment of its efficacy and safety are especially important in concomitant diseases [1, 4].

Objective of our study was to identify peculiarities of cardiac remodeling in patients with HTN and concomitant BA of different severity.

Design and methods

We examined 91 patients with controlled BA of different severity accompanied by HTN stage 1–2. Mild BA with coexistent HTN (MiBAHTN) was diagnosed in 26 patients (29%), moderate BA (MoBAHTN) was diagnosed in 34 patients (37%), and severe BA (SBAHTN) was diagnosed in 31 (34%) patients.

All examined subjects were outpatients, and during at least 6 months after BA exacerbation they received adequately selected BA therapy with inhaled glucocorticosteroids (IGCSs), including short-term β_2-adrenoceptor agonists prior to IGCSs and on request. The patients were given recommendations for non-pharmacological treatment of HTN and correction of risk factors. All patients signed written informed consent and could withdraw from the study at any time.

The following exclusion criteria were applied: secondary or malignant HTN; acute coronary syndrome; atrial fibrillation; sick sinus syndrome, sinoatrial or atrioventricular block of II–III degree; cerebral stroke; chronic heart failure NYHA functional class III–IV; uncontrolled BA; severe concomitant diseases, including cancer; severe depression; history of alcohol and drug abuse; pregnancy and lactation.

As a control group primary referred patients with HTN stage 1–2, who had not previously received antihypertensive drugs and had no clinically significant comorbidity. They composed the HTN group (n = 30); and the patients with controlled BA of various severity without significant comorbidity composed the BA group (n = 32).

BA was diagnosed according to the GINA 2014 guidelines. The diagnosis of HTN and BP increase were defined based on the recommendations by the expert group of the World Health Organization, 2013.

Echocardioscopy (Echo-CS) was performed on Acuson 128XP/10c device (USA). According to the R. Devereux (1983) formula, the left ventricular mass (LVM) was calculated: $LVM = 1.04 \times [EDD + LVPWT + IVST)^3 — EDD^3] — 13.6$, where EDD is end-diastolic diameter, LVPWT is left ventricular posterior wall thickness, IVST is interventricular septum thickness; myocardial mass index (LVMI) was calculated according to the formula: LVM/body surface area (g/m^2). Systolic pressure in the pulmonary artery (SPPA) and total pulmonary vascular resistance (TPVR) were also evaluated.

The data were processed by variation statistics methods using program package Statistica 6.0 (StatSoft Inc.) Mean sampled values and standard

Table 1

CHARACTERISTICS OF PATIENTS

Parameter	MiBAHTN (n = 26)	MoBAHTN (n = 34)	SBAHTN (n = 31)	BA (n = 32)	HTN (n = 30)	p
Age, years (18-70)	46.35 ± 8.2	56.2 ± 5.7	54.8 ± 7.2	48.18 ± 9.4	53.8 ± 7.5	p > 0.05
Male/Female, n	11/15	12/22	12/19	11/20	18/12	p > 0.05
Duration of BA, years	11.32 ± 6.84	14.35 ± 7.63	14.52 ± 4.32	13.27 ± 9.12	–	p > 0.05
Duration of HTN, years	6.4 ± 4.6	9.3 ± 3.6	7.2 ± 5.1	–	4.5 ± 2.8	p > 0.05
SBP, mm Hg	144.2 ± 2.8	143.2 ± 3.2	145.4 ± 4.7	134.6 ± 3.7	147.9 ± 2.1	p > 0.05
DBP, mm Hg	89.4 ± 3.1	90.4 ± 2.1	91.2 ± 5.3	83.7 ± 2.5	86.2 ± 3.1	p > 0.05
HR, bts/min	68 ± 6.2	74 ± 5.4	76 ± 5.6	74 ± 7.2	69 ± 6.4	p > 0.05

Note: MiBAHTN — mild BA accompanied with grade 1–2 hypertension; MoBAHTN — moderate BA accompanied with grade 1–2 hypertension; SBAHTN — severe BA accompanied with grade 1–2 hypertension; BA — bronchial asthma; HTN — arterial hypertension; SBP — systolic blood pressure; DBP — diastolic blood pressure; HR — heart rate.

Table 2

ECHOCARDIOGRAPHY DATA (M ± m)

Parameter	MiBAHTN (n = 26)	MoBAHTN (n = 34)	SBAHTN (n = 31)	BA (n = 32)	HTN (n = 30)
LA, cm	2.92 ± 0.04	3.12 ± 0.03*	3.47 ± 0.05*	2.92 ± 0.05	3.02 ± 0.04
EDD LV, cm	5.01 ± 0.06	5.19 ± 0.06	5.65 ± 0.06*	4.90 ± 0.06	5.01 ± 0.06
PWT LV, cm	0.98 ± 0.01*	1.13 ± 0.03*#	1.27 ± 0.03*	0.93 ± 0.02	1.08 ± 0.03
IVST LV, cm	0.79 ± 0.03	1.02 ± 0.04	1.05 ± 0.03*	0.88 ± 0.03	0.93 ± 0.04
LVMI, g/m^2	109.18 ± 1.16*	123.1 ± 1.42*#	128.45 ± 1.91*	87.55 ± 2.65	112.1 ± 1.97
LVDD, cm	0.88 ± 0.02	1.06 ± 0.03*	1.16 ± 0.03*	0.90 ± 0.02	1.02 ± 0.02
LV/RV	2.90 ± 0.11	2.78 ± 0.10	2.47 ± 0.06	2.58 ± 0.09	2.42 ± 0.09
RWT	0.39 ± 0.03	0.44 ± 0.02	0.45 ± 0.01	0.38 ± 0.01	0.41 ± 0.02
RV, cm	3.83 ± 0.16*	5.2 ± 0.12*#	10.45 ± 0.49	1.9 ± 0.07	1.94 ± 0.05
Ve, m/s	0.7 ± 0.02	0.6 ± 0.04	0.6 ± 0.02	0.7 ± 0.02	0.6 ± 0.02
Va, m/s	0.7 ± 0.03	0.7 ± 0.03	0.7 ± 0.01	0.7 ± 0.04	0.7 ± 0.01
E/A	0.96 ± 0.05	0.88 ± 0.05*#	0.86 ± 0.07*#	1.04 ± 0.07	0.98 ± 0.03
IVRT, ms	103.4 ± 5.8*	124.7 ± 5.8*#	131.3 ± 4.1*#	93.2 ± 4.5	98.5 ± 5.3
PASP, mm Hg	26.12 ± 0.43	27.28 ± 0.57*	28.54 ± 0.32*	22.09 ± 0.39	26.21 ± 0.43
TPR, dyne×s×cm^{-5}	201.4 ± 8.25	225.16 ± 13.24*#	234.8 ± 11.00*#	177.44 ± 7.56	203.54 ± 11.64

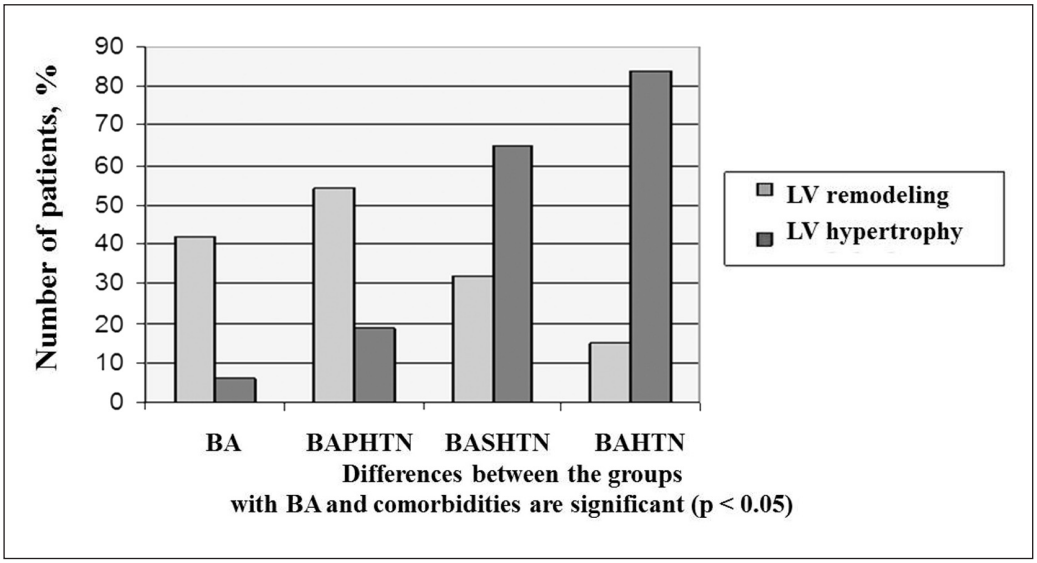
Note: MiBAHTN — mild BA accompanied with grade 1–2 hypertension; MoBAHTN — moderate BA accompanied with grade 1–2 hypertension; SBAHTN — severe BA accompanied with grade 1–2 hypertension; BA — bronchial asthma; HTN — arterial hypertension; LA — left atrium; LV — left ventricular; EDD LV — end diastolic diameter LV; PWT LV — posterior wall end-diastolic thickness LV; IVST LV — interventricular septum thickness LV; LVMI — left ventricular mass index; LVDD — left ventricular diastolic dysfunction; LV/RV — left ventricular/right ventricular; RWT — relative wall thickness; RV — right ventricular; IVRT — isovolumic relaxation time; PASP — pulmonary artery systolic pressure; TPR — total pulmonary resistance; * — significant differences between groups BA + HTN and BA ($p < 0.05$); # — significant differences between groups BA + HTN and HTN ($p < 0.05$).

deviation were calculated (M ± SD). The Student's t-test was used for normally distributed indicators. The differences between mean values were considered significant at $p < 0.05$.

Results

Overall characteristics of patients enrolled in the study are provided in Table 1. The groups were matched by gender, age, and clinical data.

Figure 1. Rate of left ventricular remodeling and hypertrophy



Note: MiBAHTN — mild BA accompanied with grade 1–2 hypertension; MoBAHTN — moderate BA accompanied with grade 1–2 hypertension; SBAHTN — severe BA accompanied with grade 1–2 hypertension; BA — bronchial asthma; HTN — arterial hypertension; LV — left ventricle.

ECHO-CS was performed at baseline (Table 2). SPPA and TPVR increase was detected, being more pronounced in the groups with cardiovascular pathology as compared to isolated BA: BA group — 22.09 ± 0.39 mmHg, HTN group — 26.21 ± 0.43 mmHg, MiBAHTN — 26.12 ± 0.43 mmHg, MoBAHTN — 27.28 ± 0.57 mmHg (p = 0.003), SBAHTN — 28.54 ± 0.32 mmHg (p = 0.0001). The SPPA index was similar in the groups with isolated HTN and in patients with comorbidity. TPVR increase was detected in the examined subjects with HTN, and, in patients with the combination of HTN and BA, PVR was higher: BA group — 177.44 ± 7.56 dyn × s × cm^{-5}, HTN group — 203.54 ± 11.64 dyn × s × cm^{-5}, MiBAHTN — 201.4 ± 8.25 dyn × s × cm^{-5}, MoBAHTN — 225.16 ± 13.24 dyn × s × cm^{-5} (p = 0.005), SBAHTN — 234.8 ± 11 dyn × s × cm^{-5} (p = 0.001).

The right ventricular (RV) remodeling had similar pattern in BA patients with and without HTN, and was more unfavorable nature in case of comorbid diseases. Thus, the frequency of RV hypertrophy in patients with the combination of BA and HTN was significantly higher: BA group — 1.9 ± 0.07, HTN group — 1.94 ± 0.05, MiBAHTN — 3.83 ± 0.16 (p = 0.01), MoBAHTN — 5.2 ± 0.12 (p = 0.002, p = 0.005), SBAHTN — 10.45 ± 0.49 (p = 0.0015, p = 0.0001).

The most significant differences in the compared groups concerned the LV. Particularly, LVMMI and LVPWT in all groups with HTN were significantly ($p < 0.05$) higher. The differences were also detected regarding ventricular functions, primarily those of the LV. LVMMI in patients with comorbidity exceeded the similar values of the BA and HTN groups: BA group — 87.55 ± 2.65 g/m^2, HTN group — 112.1 ± 1.97 g/m^2, MiBAHTN — 109.18 ± 1.16 g/m^2 (p = 0.004), MoBAHTN — 123.1 ± 1.42 g/m^2 (p = 0.001, p = 0.0015), SBAHTN — 128.45 ± 1.91 g/m^2 (p = 0.001, p = 0.0001). LVPWT in patients with comorbidity exceeded the similar values of the BA and HTN groups, specially starting from the group with moderate HTN: BA group — 0.93 ± 0.02 cm, HTN group — 1.08 ± 0.03 cm, MiBAHTN — 0.98 ± 0.01 cm, MoBAHTN — 1.13 ± 0.03 cm (p = 0.004, p = 0.001), SBAHTN — 1.27 ± 0.03 cm (p = 0.001, p = 0.0001).

EDD was similar in subjects with comorbidity and in the BA and HTN groups, although worsening of the bronchial obstructive syndrome was associated with a clear tendency to EDD increase: BA group — 4.90 ± 0.06 cm, HTN group — 5.01 ± 0.06 cm, MiBAHTN — 5.01 ± 0.06 cm, MoBAHTN — 5.19 ± 0.06 cm, SBAHTN — 5.65 ± 0.06 cm.

LV remodeling developed simultaneously with the changes in the right ventricle, but in case of the

LV structural changes it was different depending on presence or absence of comorbidity. LV concentric remodeling was found in 13 patients (42 %) with isolated BA, while the cases of concentric LV hypertrophy were diagnosed in 2 patients (6 %). In HTN groups, the specific weight of LV remodeling decreased in proportion to severity of the bronchial obstructive syndrome: in the MiBAHTN group, LV remodeling was found in 14 examined patients (54 %), in the MoBAHTN group — in 11 patients (32 %), in the SBAHTN group — in 4 patients (13 %), yielding to LV hypertrophy (LVH): MiBAHTN — in 5 patients (19 %), MoBAHTN — in 22 patients (65 %), SBAHTN — in 25 patients (84 %) (Fig. 1).

As for LV diastolic dysfunction, the maximal velocity of active filling (A) exceeded the maximal velocity of rapid filling (E) was found in all groups. The E/A ratio was the following: BA group — 1.04 ± 0.07, HTN group — 0.98 ± 0.03, MiBAHTN — 0.96 ± 0.05, MoBAHTN — 0.88 ± 0.05 (p = 0.001, p = 0.0015), SBAHTN — 0.86 ± 0.07 (p = 0.001, p = 0.0001), and it was lower in the groups with bronchopulmonary pathology than in the BA and HTN groups, most likely due to more pronounced redistribution of blood flow at the phase of active filling in subjects with comorbidity (Table 2). LV isovolumic relaxation time was the following: BA group — 93.2 ± 4.5 ms, HTN group — 98.5 ± 5.3 ms, MiBAHTN — 103.4 ± 5.8 ms (p = 0.002), MoBAHTN — 124.7 ± 5.8 ms (p = 0.005, p = 0.001), SBAHTN — 131.3 ± 4.1 ms (p = 0.0015, p = 0.001). The presented data indicate an increase in stiffness and rigidity of the myocardium in patients with comorbidity compared to isolated HTN and BA.

Systolic dysfunction was not registered in either of the study groups, although there is a clear tendency to decrease in ejection fraction (EF) by Simpson according to the worsening of the bronchial obstructive syndrome in patients with coexistent HTN.

Ejection fraction in patients with isolated HTN and comorbidity are presented in Table 3.

Discussion

Thus, our data show a definite pattern in the development of structural and functional cardiac changes in patients with HTN depending on the severity of concomitant BA. These values significantly differ from those in isolated HTN and BA given the fact that the groups were matched by age, sex and history of BA and HTN.

A. Yu. Ryabova et all (2010) showed similar remodeling of the right heart in patients with BA and cardiovascular diseases and without them and the tendency to an increase in the RV anterior wall thickness and diastolic diameter. RV structural and functional changes in patients with BA and HTN develop earlier than in subjects without HTN. As a result, RV remodeling in patients with concomitant HTN is diagnosed in patients with BA of lower severity, i. e. already in mild BA accompanied by HTN, whereas, in isolated BA it was diagnosed in BA of moderate severity and more severe (while other authors found it only in severe BA). LV remodeling demonstrates similar unidirectional tendency with "one step ahead" changes [5]. Regarding LV diastolic function, a decrease of the ratio between its early and late filling was found. In patients with comorbidity, LV diastolic dysfunction was already found in group with mild BA, and in case of comorbid pathology, along with asymptomatic systolic function, adaptive diastolic LV remodeling was found with the most severe forms in patients with severe BA [5, 10, 14, 20].

N. A. Karoli et all (2009) showed LV diastolic dysfunction in patients with cardiovascular

Table 3

THE SYSTOLIC FUNCTION IN PATIENTS ENROLLED IN THE STUDY (M ± m)

Parameter	MiBAHTN (n = 26)	MoBAHTN (n = 34)	SBAHTN (n = 31)	BA (n = 32)	HTN (n = 30)
EF LV (Simpson), %	67.7 ± 1.8	62.9 ± 1.5	61.3 ± 2.8	67.1 ± 2.4	64.5 ± 1.6

Note: MiBAHTN — mild BA accompanied with grade 1–2 hypertension; MoBAHTN — moderate BA accompanied with grade 1–2 hypertension; SBAHTN — severe BA accompanied with grade 1–2 hypertension; BA — bronchial asthma; HTN — arterial hypertension; EF LV — left ventricular ejection fraction.

pathology even with mild bronchial obstructive syndrome. LV changes were worse with the increase of bronchial obstruction severity [3].

T. G. Shapovalova et all (2009) demonstrated significant structural changes of the left and right heart in patients with comorbid diseases: large values of systolic IVST, LVPWT and MMI. Structural LV changes were different. Particularly, the groups with isolated BA showed concentric LV remodeling in patients with moderate BA; its frequency increased with aggravation of BA. On the contrary, in HTN groups, concentric and eccentric LV hypertrophy were prevalent. In addition, LV hypertrophy was diagnosed in patients with mild bronchial obstructive syndrome, and frequency of cases increased significantly with aggravation of BA [8, 10, 14].

Our data show that structural and geometric changes were found in both RV and LV myocardium in patients with BA. Along with aggravation of pulmonary and cardiovascular pathology, these changes intensified [5].

A. V. Barsukov and his foreign colleagues (2005) confirmed that concentric LV hypertrophy was observed in all groups (patients with comorbidity and with isolated HTN), but patients with coexistent HTN and BA showed greater concentricity in comparison with the patients with isolated HTN. In addition, they had significantly thicker LV walls, smaller LV cavity and LVMMI, compared to the persons with less significant concentric LV hypertrophy [1, 11, 13, 19]. The evidence of higher LVM values in patients with coexistent HTN and BA as compared to patients with HTN without concomitant pathology of the bronchial tree is rather scarce [4, 11, 13, 18]. Persistent increased LV load is the main cause leading to the LV hypertrophy in patients with HTN [10, 19].

Thus, as a compensatory mechanism, LV hypertrophy at the very early stages adversely affects LV diastolic function. Thickened cardiac wall is characterized by greater rigidity leading to a decrease in relaxation properties [2, 12].

A. V. Barsukov (2005) as well as some other researchers showed a slight decrease in systolic function of the heart in patients with concomitant chronic bronchopulmonary diseases. It was also confirmed in our study. This fact is difficult to be explained, as various ratios of volume characteristics of LV, included in the formula for EF calculation are involved. Moreover, lower EF values can also indicate initial manifestations of LV pump dysfunction in patients with comorbidity [1, 12, 18, 19].

The available publications do not present any data on any significant changes in the heart structure in mild isolated BA, even in cases of long duration (more than 10–15 years) [1, 2, 4–6, 8, 10, 11, 13]. This may be indicative of the prevailing influence of cardiovascular pathology on LV remodeling. However, the majority of publications, describing isolated HTN stage 1–2, indicated the absence of significant changes in the myocardial structure [1, 12, 19] or showed LV remodeling [1, 19]. At the same time, in patients with coexistent HTN stage 1–2 and bronchial obstructive syndrome, LV changes are identified starting with mild chronic bronchial obstruction and are aggravated with the increase of its severity [2, 4, 5, 8, 13, 18].

Conclusions

1. Isolated BA was associated with LV remodeling; LV hypertrophy was occasional in severe BA. In coexistent BA and HTN the incidence of remodeling decreased, LVMMI and sizes of LVPWT increased with increasing severity of bronchial obstruction. The more severe bronchial obstruction at persisting HTN was, the more cases of LV hypertrophy were recorded. These changes can be considered a syndrome of mutual aggravation.

2. Pulmonary hypertension is moderate and is detected already in patients with moderate and severe BA.

3. Remodeling of the right heart is similar in patients with BA both with and without HTN. In isolated BA, RV hypertrophy was diagnosed in several patients in the group with severe BA, whereas, in comorbid states, hypertrophy was found already in mild bronchial obstruction. The severity of bronchial obstruction was associated with the more severe hypertrophy.

We conclude that structural and functional changes of the myocardium in patients with the coexistent HTN and BA have a number of peculiarities. We assume the syndrome of mutual aggravation of cardiovascular and pulmonary diseases. So the patients with different comorbidities represent a special group differing from patients

with isolated pathology, and they apparently need special treatment regimen and monitoring.

Conflict of interest

The authors declare no conflict of interest.

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Author information

Alla A. Odegova, MD, Physician, Kirov State Medical Academy;

Ekaterina I. Tarlovskaya, MD, PhD, DSc, Professor of Cardiology, Department of Internal Medicine, Nizhny Novgorod State Medical Academy.