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AMBULATORY ARTERIAL STIFFNESS MONITORING IN PATIENTS WITH ASTHMA

Abstract

Cardiovascular disease is one of the major causes of death throughout the world. Early detection of target organ damage is important for more successful prevention of cardiovascular diseases and improvement of patient outcomes. One of these target organs is the vascular wall, and its damage consists in loss of elastic properties and increase in stiffness. Many studies have shown that the increasing stiffness of the vascular wall is an independent predictor of cardiovascular risk. **Objective:** To evaluate parameters of the ambulatory arterial stiffness monitoring in patients with bronchial asthma. **Materials and methods.** The study enrolled 119 patients with asthma. The group of control included 30 practically healthy volunteers comparable by gender and age with the patients of the main group. Each patient underwent ambulatory blood pressure and arterial stiffness monitoring using BPLab MnSDP-2 device (Petr Telegin, Russia). **Results:** In patients with asthma higher values of augmentation index, arterial stiffness index and pulse wave velocity in the aorta in comparison with patients of the control group, as well as violation of the 24-hour profile of arterial stiffness were noted. **Conclusion.** Patients with asthma demonstrated significantly increased arterial stiffness in comparison with control group.

Key words: *arterial stiffness, asthma, augmentation index, pulse wave velocity*

Conflict of interests

The authors declare no conflict of interests

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ASI — arterial stiffness index, AASI — ambulatory arterial stiffness index, HT — hypertension, BA — bronchial asthma, DBP — diastolic blood pressure, AI — augmentation index, CHD — coronary heart disease, PP — pulse pressure, SBP — systolic blood pressure, ABPM — ambulatory blood pressure monitoring, AASM — ambulatory arterial stiffness monitoring, DND — degree of night decrease, PWV — pulse wave velocity, CVD — cardiovascular disease, RF — risk factors, HR — heart rate

Introduction

Cardiovascular disease (CVD) is one of the leading causes of death worldwide. The development of CVD is a cardiovascular continuum from exposure to risk factors, progression of atherosclerosis to the development of its complications and death [1]. Accordingly, interrupting the chain of events at various stages of this continuum can prevent or delay CVD development. Risk factors (RF)

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are at the very beginning of the chain, being the trigger for the development of pathological conditions [2, 3]. Along with the main factors (hypertension, age, smoking, hypercholesterolemia, diabetes mellitus), vascular wall stiffness is considered as an independent cardiovascular risk factor [4].

Determination of stiffness is a study recommended by the Russian Medical Society for Hypertension to assess the state of the vascular wall as a target organ in CVD [5]. Increased stiffness is the culmination of pathological processes in the vascular wall, such as calcium deposition, fragmentation and reduction of elastin, increased amount of collagen. Calcification of major arteries results in increased pulse wave velocity (PWV) and, therefore, an earlier return of the reflected wave to the aorta. With rising stiffness systolic blood pressure (SBP) in the aorta increases but diastolic pressure (DBP) decreases. This leads to an increase in postload on the left ventricle, followed by the development of hypertrophy, deterioration of coronary perfusion [4].

Bronchial asthma (BA) remains a global health problem due to the annual increase in morbidity and mortality [5]. The frequent combination of BA and CVD raises interest in the study of this problem. In local medicine, studies were conducted with a single measurement of vascular stiffness in patients with asthma, and an increased PWV in patients with severe disease was revealed [6, 7]. The emergence of the possibility of measuring stiffness during the day makes it possible to identify the initial changes in the vascular wall, to assess the circadian rhythm of indicators [8, 9].

The **objective** was to study the daily profile of vascular wall stiffness in patients with BA.

Materials and methods

The open-label study enrolled 119 patients with BA (44 men and 75 women), whose mean age was 56.7 ± 8.23 years. The disease duration was 6 [4.0; 8.0] years. The diagnosis of BA was established in accordance with the generally accepted clinical, laboratory and functional criteria (GINA 2016) after spirometry. In accordance with the objective, we excluded patients with clinical signs of coronary heart disease, peripheral vascular atherosclerosis, other severe chronic diseases at the acute stage, bronchial and lung diseases of other etiology from the analysis.

The control group included 30 healthy individuals without hypertension (HT), chronic respiratory diseases, or family history of coronary heart disease (CHD). The examined persons of the control group were comparable by gender and age with the patients of the main group (Table 1).

All examined persons signed an informed consent to participate in the study.

Ambulatory blood pressure (ABPM) and arterial stiffness (AASM) monitoring was carried out using the BPLab MnSDP-2 device (Petr Telegin LLC, Nizhny Novgorod, Russia), stiffness parameters

Table 1. *Clinical pattern of patients with asthma and of the control group*

Parameters	Patients with BA, n=119	Control group, n=30	p_1
Age (years)	56.7 ± 8.23	57.14 ± 6.5	ns
Body mass index (BMI) (kg/m^2)	27.43 ± 5.12	26.79 ± 5.05	ns
Smokers, %	33.6	36	ns
Mild bronchial asthma, %	6.7	–	–
Moderate bronchial asthma, %	33.6	–	–
Severe bronchial asthma, %	59.7	–	–
Hypertension, %	69.7	–	–
Duration of hypertension (years)	5.0 [4.0; 7.5]	–	–
Systolic BP, mm Hg	134.6 ± 15.22	118.2 ± 5.55	<0.001
Diastolic BP, mm Hg	85.3 ± 8.9	75.04 ± 4.3	<0.001
FEV_1, %	67.0 [38.0; 84.0]	99.0 [78.0; 102.0]	<0.001

Note: ns — not statistically significant

were estimated by means of the Vasotens application program based on mathematical processing of pressure oscillogram records.

Among the parameters of arterial stiffness, arterial stiffness index (ASI), ambulatory arterial stiffness index (AASI), pulse wave velocity in the aorta (PWV), and augmentation index (AI) were assessed. To solve the problem of dependence of arterial stiffness on BP value and heart rate (HR), BPLab software calculates values reduced to SBP of 100 mm Hg and HR of 60 and 75 beats/min: $PWV_{100\text{-}60}$, $ASI_{100\text{-}60}$, AI_{75}.

To assess the circadian rhythm we proposed the "Method of estimating the circadian rhythm of pulse wave velocity in aorta" (rationalization proposal No. 2993 dated 20.03.2018) based on the determination of the degree of night decrease (DND in PWV) by the formula: the ratio of the difference between the average daily and average night parameters of PWV to the average daily parameters, expressed as a percentage:

$$\text{DND in PWV} = \{[\text{PWV (d)} - \text{PWV (n)}] / \text{PWV (d)}\} \times 100\ \%$$

Normal values were determined based on the study results for arterial stiffness in healthy individuals. The range of normal values was defined as the range of measurements consisting of two quartiles above and below the median. Thus, the range includes the central 50 % of all measurements and determines the "normal" boundaries of DND in PWV in the aorta from 11 to 18 %.

Statistical analysis was performed using STATISTICA 10.0 (StatSoft) software package. To check the conformity of parameter distribution with normal distribution, the Shapiro — Wilk test was used, and normal distribution was considered with $p>0.05$. For the description of normally distributed quantitative parameters, the mean value of parameter and standard deviation (M ± SD) were used; for the description of parameter distribution different from the normal, the median (Me), upper and lower quartiles were indicated [Q25; Q75]. To compare two groups with normal distribution of the quantitative parameter, Student's t-test for independent groups was determined. The correlation of two normally distributed quantitative parameters was studied using the Pearson method. When the distribution deviated from normal distribution, the Mann — Whitney test was used to compare the significance of intergroup differences in quantitative values, and the Spearman method was used to analyze the association of qualitative parameters. The differences were considered as statistically significant at $p<0.05$.

Results

In the study of the daily vascular wall stiffness parameters, their statistically significant increases were found in patients with BA (Table 2). According to guidelines of European Society of Cardiology and European Society of Hypertension, PWV >10 m/s is a risk factor for cardiovascular events [4]. Our study revealed an increase in the average daily, daily and night PWV, and values of more than 10 m/s were reported in 43 (36.1 %) patients with BA and only in 6 (5 %) patients of the control group ($p<0.001$).

AI is the ratio of direct and reflected pulse waves passing through the vascular wall. Normally, the reflected component is always less than a direct one, and AI value is negative. In the case of increased vascular stiffness, the reflected component may exceed the direct one, and AI value becomes positive [10]. The increase in the reflected wave velocity leads to its early return to the aorta, during the systole period, which results in increased systolic BP (SBP), pulse pressure (PP), post-loading on the left ventricle, decreased diastolic BP (DBP), and, therefore, a deterioration in coronary perfusion [11]. Our study found 24-hour, day and night increase in AI in patients with BA compared to the values of similar parameters in healthy individuals. The given parameters of AI_{75} (24-hour, day, night), $ASI_{100\text{-}60}$ (24-hour, day), $PWV_{100\text{-}60}$ in the aorta (24-hour, night) in the patients keep increasing compared with the values of these parameters in healthy individuals, which confirms the higher arterial stiffness in patients with BA regardless of BP and HR values.

The inclusion of algorithms for determining arterial stiffness in ABPM devices makes it possible to estimate 24-hour changes of parameters [12]. It was found that ASI, $ASI_{100\text{-}60}$, AI and AI_{75} at night were higher than in the daytime ($p<0.05$), which indicates an increase in arterial stiffness at night.

Table 2. *Parameters of ambulatory arterial stiffness monitoring in patients with asthma and of the control group*

Parameters	Patients with BA, n=119	Control group, n=30	p_1
	24 hours		
PWVao, m/s	9.74 ± 1.73	9.12 ± 1.23	=0.0023
$PWVao_{100-60}$, m/s	9.14 ± 1.87	8.59 ± 1.55	=0.014
AI, %	–21.14 ± 22.54	–25.62 ± 17.23	=0.03
AI_{75}, %	–24.32 ± 20.16	–28.31 ± 21.71	=0.024
ASI	129.4 [124.0; 159.0]	118.5 [108; 124]	=0.012
ASI_{100-60}	119.0 [105.0; 159.0]	109.0 [95.0; 112.0]	=0.02
AASI	0.30 ± 0.18	0.31 ± 0.18	ns
DND in PWVao, %	8.5 [5.5; 11.5]	11.5 [9.5; 14.5]	=0.032
	Awake		
PWVao, m/s	9.86 ± 1.76	9.25 ± 1.22	<0.001
$PWVao_{100-60}$, m/s	9.02 ± 2.49	8.72 ± 1.46	ns
AI, %	–20.56 ± 19.53	–28.26 ± 18.63	<0.001
AI_{75}, %	–24.38 ± 21.35	–31.35 ± 19.43	=0.0041
ASI	119.0 [121.0; 155.0]	109.0 [91.0; 131.0]	=0.021
ASI_{100-60}	107.0 [97.0; 142.0]	98.5 [89.0; 122.0]	ns
	Asleep		
PWVao, m/s	9.34 ± 1.73	8.98 ± 1.19	=0.045
$PWVao_{100-60}$, m/s	8.97 ± 2.16	8.46 ± 1.28	=0.028
AI, %	–17.52 ± 24.5	–25.46 ± 28.34	=0.012
AI_{75}, %	–23.46 ± 23.15	–33.96 ± 37.46	=0.0023
ASI	132.0 [123.0; 181.0]	122.5 [106.5; 138.5]	=0.042
ASI_{100-60}	117.0 [95.0; 166.0]	109.0 [90.0; 128.0]	ns

Note: ns — not statistically significant

This is confirmed by the value of the degree of night decrease in PWV, which in patients with BA (8.5 [5.5; 11.5]) was significantly lower than in the control group (11.5 [9.5; 14.5], p=0.032). Abnormal DND in PWV in the aorta was found in 48 (40.3 %) patients with BA and only in 5 (16.6 %) people in the control group.

In this study, it was found that the increase in vascular wall stiffness is associated with clinical and functional parameters. Thus, BA severity correlated with PWV per day (r=0.46, p=0.001), PWV_{100-60} per day (r=0.36, p<0.001), ASI per night (r=0.3, p=0.008) and ASI_{100-60} per day (r=0.35, p=0.023). Increase in PWV at night (r=0.33, p=0.003) was associated with the increase in the number of BA exacerbations over the past 12 months. The number of points obtained in the AST questionnaire had a moderate negative correlation with PWV and PWV_{100-60} per day (r=–0.56, p<0.001 and r=–0.62, p<0.001, respectively) and in daytime hours (r=–0.5, p=0.001 and r=–0.43, p=0.001, respectively).

The dependence of vascular stiffness parameters on age, BMI, cholesterol levels and smoking has been proven in numerous studies. Patients with asthma also showed an increase in PWV per 24 hours (r=0.32, p=0.021) and day (r=0.34, p=0.014), PWV_{100-60} per day (r=0.39, p=0.009) with increasing age. The relationship between body mass index (BMI) and AI per day (r=0.36, p=0.001), PWV per 24 hours (r=0.68, p<0.001), day (r=0.66, p<0.001) and night (r=0.73, p<0.001) was revealed. Average night AI_{75} values were correlated with an index in pack-years (r=0.32, p=0.004). With an increase in cholesterol levels, there was an increase in the average daily (r=0.39, p<0.001) and average night (r=0.37, p=0.001) values of ASI_{100-60}.

The most common concomitant disease in patients with BA is HT. The combination of the two diseases was found in 35 % of patients. In order to analyze the effect of concomitant hypertension on vascular wall stiffness, patients were divided into 2 groups depending on the presence of HT. The first group

Table 3. *Clinical pattern of patients with asthma with and without hypertension*

Parameters	BA and HT, n=83	BA without HT, n=36	Control group, n=30
Age (years)	57.54 ± 9.18	56.82 ± 9.54	57.14 ± 6.5
Body mass index (BMI) (kg/m²)	28.26 ± 4.34*#	26.92 ± 5.02	26.79 ± 5.05
Mild bronchial asthma, %	3.6	13.9	–
Moderate bronchial asthma, %	24	55.6	–
Severe bronchial asthma, %	72.4	30.5	–
Hypertension, %	100	–	–
Duration of hypertension, years	6.32 ± 8.2#	–	–
Systolic BP, mm Hg	134.8 ± 10.4**##	123.4 ± 6.1	118.2 ± 5.55
Diastolic BP, mm Hg	83.6 ± 9.1**#	78.12 ± 4.7	75.04 ± 4.3

Note: Statistically significant differences with the control group: * — p<0.05, ** — p<0.01, *** — p<0.001; with group 2: # — p<0.05, ##### — p<0.01, ### — p<0.001

Table 4. *Parameters of ambulatory arterial stiffness monitoring in patients with asthma with and without hypertension*

Parameters	BA and HT, n=83	BA without HT, n=36	Control group, n=30
	24 hours		
PWVao, m/s	10.09 ± 1.82*#	9.28 ± 1.82	9.12 ± 1.23
$PWVao_{100-60}$, m/s	9.38 ± 1.96*#	8.78 ± 1.73	8.59 ± 1.55
AI, %	–15.84 ± 16.94***##	–23.61 ± 17.6	–25.62 ± 17.23
AI_{75}, %	–22.78 ± 18.43*#	–27.45 ± 18.52	–28.31 ± 21.71
ASI	134.0 [118.0; 148.0]**#	124.0 [116.0; 132.0]*	118.5 [108; 124]
ASI_{100-60}	120.5 [108.0; 139.0]**#	114.0 [101.0; 129.0]	109.0 [95.0; 112.0]
AASI	0.3 ± 0.17	0.29 ± 0.19	0.31 ± 0.18
DND in PWVao, %	8.0 [5.5; 10.0]***##	10.5 [7.0; 12.5]*	11.5 [9.5; 14.5]
	Awake		
PWVao, m/s	10.13 ± 1.81*#	9.36 ± 2.1	9.25 ± 1.22
$PWVao_{100-60}$, m/s	9.65 ± 2.09*#	8.92 ± 1.93	8.72 ± 1.46
AI, %	–17.11 ± 17.4***##	–24.92 ± 18.2*	–28.26 ± 18.63
AI_{75}, %	–23.38 ± 19.52**#	–28.36 ± 19.1*	–31.35 ± 19.43
ASI	126.0 [120.0; 149.0]*#	118.0 [112.0; 141.0]*	109.0 [91.0; 131.0]
ASI_{100-60}	117 [97.0; 124.0]*#	109 [91.0; 124.0]*	98.5 [89.0; 122.0]
	Asleep		
PWVao, m/s	9.87 ± 1.86*#	9.14 ± 1.92*	8.98 ± 1.19
$PWVao_{100-60}$, m/s	9.24 ± 1.56*#	8.54 ± 2.42	8.46 ± 1.28
AI, %	–14.73 ± 15.41*#	–21.72 ± 21.14*	–25.46 ± 28.34
AI_{75}, %	–21.82 ± 20.1*#	–26.53 ± 22.72*	–33.96 ± 37.46
ASI	135.5 [122.0; 152.0]**#	128.0 [121.0; 149.0]*	122.5 [106.5; 138.5]
ASI_{100-60}	126.0 [97.0; 146.0]*#	114.0 [99.0; 136.0]	109.0 [90.0; 128.0]

Note: Statistically significant differences with the control group: * — p<0.05, ** — p<0.01, *** — p<0.001; with BA patients without HT: # — p<0.05, ##### — p<0.01, ### — p<0.001

consisted of 83 patients with BA with HT, the second one — 36 patients without HT (Table 3).

It was noted that vascular wall stiffness increased in patients with BA regardless of the presence or absence of HT, but changes in PWV and AI per 24 hours, day and night were more significant in patients with HT (Table 4).

DND in PWV was calculated for all of the subjects. In patients with a combination of BA and HT, pathological DND in PWV in the aorta was revealed

in 41 (49.4 %) patients, in 7 (19.4 %) patients with BA without HT and in 5 (16.6 %) patients of the control group.

In the study of the above parameters in patients with BA and HT elevated values of AI_{75} (24 hours, day, night), ASI_{100-60} (24 hours, day, night), $PWVao_{100-60}$ (24 hours, day, night) remained in comparison with patients with BA without HT and individuals in the control group.

The correlation of ASI_{100-60} per 24 hours ($r=0.37$, $p=0.001$) and night time ($r=0.32$, $p=0.004$) with the duration of HT was noted.

Discussion

Despite significant advances in understanding the pathogenesis of BA and the development of new drugs, in real clinical practice, challenges in the achievement of complete disease control remain. One of the reasons hindering control is the presence of concomitant diseases, including of the cardiovascular system. The results of recent studies show that the combination of BA and CVD is increasingly common in routine clinical practice, due to both increased incidence of CVD and BA, and an increase in the geriatric population of patients.

According to meta-analysis data, including 11 studies (117,548 patients with BA and 443,948 persons of the control group), cardiovascular and cerebrovascular diseases, including CHD, myocardial infarction, HT, stroke, arrhythmias, atrial fibrillation and heart failure are more common in patients with asthma [13].

The modern concept of CVD development, which is understood as the continuous development of CVD from risk factors to the development of chronic heart failure, implies the possibility of correcting changes already at the stage of identifying risk factors. Arteries are one of the main organs that are affected by CVD risk factors. In the literature, there is a large number of works devoted to the study of changes in the structure and metabolism of the arterial wall under the influence of CVD risk factors. However, most of them were carried out in patients with existing CVD, so the question on the state of the walls of the major arteries in the presence/absence of CVD risk factors in practically healthy (with respect to the cardiovascular system) persons remains relevant. Based on the requirements for CVD prevention, modern non-invasive diagnosis of the state of artery walls should facilitate screening studies. The methods used should be reproducible, independent of the researcher, allow comparison of data obtained in different research centers, and ensure the fullest possible compliance of the obtained arterial wall stiffness parameters with histological data. Automated systems for determining the biomechanical properties of arteries based on the principles of sphygmography are of great interest to researchers. One of the most commonly used parameters of arterial wall stiffness is PWV.

Earlier studies have demonstrated an increase in the stiffness of the vascular wall in patients with BA in the case of single measurement. In the study by Demko I. V. (2013), 204 patients with moderate and severe BA in the exacerbation period and over the time after 12 months were evaluated for arterial stiffness, and increase in PWV and AI values was revealed in patients with BA both in the period of exacerbation and in remission. It was noted that stiffness parameters correlated with oxygen saturation, disease severity, and clinical symptom severity [6]. The results of our study confirm the previously obtained data in patients with BA; an increase in vascular wall stiffness was found in patients with BA, and correlation of disease severity with PWV was also noted.

In previous studies, it was found that obesity contributes to vascular wall stiffness [6]. In our study, a positive correlation between arterial stiffness and BMI and other classical CVD factors (cholesterol level, age) was found.

Analyzing the status of arterial mechanical properties in patients with BA depending on the presence or absence of HT, it was found that the presence of HT worsens the elastic properties of the vascular wall: in hypertensive patients, changes in PWV and AI were more significant than in patients without hypertension. Correlation of vascular stiffness with duration of HT was revealed.

Sobko E. A. (2012), in her study with a single measurement of stiffness parameters, found that an increase in arterial stiffness is observed regardless of the presence of concomitant HT in patients with BA, but the greatest changes were detected in patients with high blood pressure [14]. The advantage of ambulatory vascular stiffness monitoring is the ability to assess circadian rhythm of parameters. In the patients examined by us, there was a

tendency of night increase in vascular stiffness, accompanied by insufficient DND in PWV, which may be the result of the negative effect of night bronchial obstruction attacks.

In the study by Sobko E. A. et al. (2012), the severity of systemic inflammation was a predictor of increased vascular stiffness [14]. The data obtained by the authors indicate that an increase in serum levels of proinflammatory cytokines (sPECAM-1/sCD31) during the period of BA exacerbation leads to an increase in PWV (r=0.517, p=0.008) [13]. Our study revealed a positive correlation between the number of asthma exacerbations and PWV, which indirectly confirms the relationship between systemic inflammation and increased vascular stiffness.

Conclusions

In patients with bronchial asthma, there was an increase in vascular wall stiffness, an increase in PWV and AI values for 24 hours, day and night, and AI values per night were higher than daily values, which indicates an increase in arterial stiffness at night. The presence of concomitant HT leads to an increase in vascular wall stiffness. Given the proven prognostic value of arterial stiffness parameters, their study in patients with BA will allow to assess cardiovascular risk earlier.

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