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ПОКАЗАТЕЛИ ЭХОКАРДИОГРАФИИ У ПРЕДСТАВИТЕЛЕЙ УРБАНИЗИРОВАННОЙ ЭТНИЧЕСКОЙ ГРУППЫ ШОРЦЕВ С АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ

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Основные положения

• Этническая принадлежность влияет на эхокардиографические показатели пациентов с гипертонией, поэтому необходимо использовать референсные значения, характерные для конкретной популяции, а не евроамериканские стандарты. У городских шорцев с гипертонией наблюдаются иные паттерны ремоделирования левого желудочка и диастолической функции по сравнению с представителями других этнических групп, что подчеркивает роль этнической принадлежности в адаптации сердца к гипертонии. Метаболические и гемодинамические факторы, вызывающие гипертрофию левого желудочка, также различаются у шорцев и представителей других этнических групп, что указывает на этнические различия в патофизиологии изменений сердца, связанных с гипертонией.

Цель	Сравнение показателей эхокардиографии у урбанизированных пациентов с артериальной гипертензией шорцев и представителей некоренных этнических групп, проживающих в регионе Горная Шория.
Материалы и методы	В исследование были включены пациенты с артериальной гипертензией: 58 шорцев (20 мужчин и 38 женщин) и 50 некоренных жителей (15 мужчин и 35 женщин) Горной Шории. Всем пациентам было проведено эхокардиографическое исследование с оценкой параметров левых отделов сердца. Оценивались выраженность гипертрофии левого желудочка (ГЛЖ), геометрия левого желудочка (ЛЖ) и диастолическая функция в этнических и половых группах.
Результаты	У шорцев с гипертензией не выявлено достоверной разницы в размерах левого желудочка по сравнению с лицами европеоидной расы. Выраженная патологическая ГЛЖ у шорцев была связана с уровнем глюкозы (ОШ 3,24; 95% ДИ 1,56–6,7; p = 0,001), а у европеоидов – с систолическим (p = 0,021) и диастолическим (p = 0,011) артериальным давлением, а также частотой сердечных сокращений (p = 0,033). У шорцев чаще наблюдалась нормальная диастолическая функция ЛЖ (p = 0,029) и концентрическое ремоделирование ЛЖ (p = 0,023), а также реже – нормальная геометрия ЛЖ (p = 0,015) по сравнению с мужчинами некоренного населения.
Заключение	Таким образом, данное исследование может быть полезным для оценки влияния развития артериальной гипертензии, а также изменения образа жизни шорцев при переезде из сельской местности в город на этнические различия в эхокардиографии.
Ключевые слова	Артериальная гипертензия • Эхокардиография • Гипертрофия левого желудочка • Шорцы

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ECHOCARDIOGRAPHY INDICES IN URBANIZED SHOR ETHNIC GROUP REPRESENTATIVES WITH ARTERIAL HYPERTENSION

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Highlights

• Ethnic specificity matters in echocardiographic parameters of hypertensive patients, necessitating population specific reference values beyond Euro American standards. Urbanized Shors with hypertension demonstrate distinct patterns of left ventricular remodeling and diastolic function compared to non indigenous residents, highlighting the role of ethnicity in cardiac adaptation to hypertension. Metabolic and hemodynamic determinants of left ventricular hypertrophy differ between Shors and non indigenous groups, suggesting ethnic variations in the pathophysiology of hypertension related cardiac changes.

Aim	To compare echocardiography indicators in urbanized patients with arterial hypertension of the Shor and non-indigenous ethnic groups living in the Gornaya Shoria region.
Methods	The study included patients with arterial hypertension: 58 Shors (20 men and 38 women) and 50 non-indigenous residents (15 men and 35 women) of Gornaya Shoria. All underwent an echocardiographic study with an assessment of the left heart parameters. We assessed the left ventricular hypertrophy (LVH) severity, left ventricular (LV) geometry and diastolic function in the ethnic and sex groups.
Results	Shors with hypertension no showed a significant difference in left ventricular dimensions compared to Caucasian. Severe abnormal LVH was associated in the Shors with glucose levels (OR 3.24, 95% CI 1.56–6.7, $p = 0,001$), and in Caucasians with systolic ($p = 0.021$) and diastolic ($p = 0.011$) blood pressure, and heart rate ($p = 0.033$). The Shor men were more likely to have normal LV diastolic function ($p = 0.029$) and LV concentric remodeling ($p = 0.023$), and less often to have normal LV geometry ($p = 0.015$) compared to non-indigenous men.
Conclusion	In this study, no significant differences in left ventricular size were observed in urban Shors with hypertension compared to Caucasians. Severe abnormal LVH and concentric LV remodeling were specific to Shors males, which should be taken into account when assessing the impact of hypertension in this ethnic group.

Keywords	Arterial hypertension • Echocardiography • Left ventricular hypertrophy • Shors

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Список сокращений

a'	– late diastolic mitral annular tissue velocity	e'	– early diastolic mitral annular tissue velocity
A	– peak left ventricular filling velocity at atrial contraction	LVH	– left ventricular hypertrophy
BSA	– body surface area	LVM	– left ventricular mass
DT	– deceleration time of early diastolic mitral flow	LVMi	– left ventricular mass index
E	– peak early diastolic left ventricular filling velocity	s'	– systolic mitral annular tissue velocity

Introduction

Detection of left ventricular hypertrophy (LVH) has important clinical and prognostic significance in patients with arterial hypertension [1, 2]. Echocardiography is widely used and is considered the best method for this purpose. However, most data, on which the current worldwide-used ASE and EACVI recommended echocardiographic quantification values for the left ventricle are based, were obtained from Caucasian subjects in the United States and Europe [3]. Therefore, the use of published regulatory "control limits" remains limited as extending them to other racial and ethnic groups may not be applicable. For example, a systematic review has shown that left ventricular mass (LVM) reference ranges cannot be easily applied to the world's diverse multiracial population with cardiovascular disease [4]. If current ASE guidelines are used to define LVM as "normal", then some patients may be misclassified [5–7]. This misclassification can lead to an underestimation of LVH and, ultimately, to inadequate treatment and underestimation of the predictive value of LVH in certain patients [8, 9].

Moreover, even multicenter studies involving other regions and countries do not take the ethnic diversity of the inhabitants of these states into account. Some regions were even underrepresented in the largest-scale study of recent times – WASE-despite the inclusion of the countries of Africa, Asia and the Middle East [10]. A complete representation of the diversity of ethnic groups in each country was also not achieved in this study. Accordingly, further regional studies will be needed to address the issue of ethnic diversity within the country. Therefore, it becomes necessary to study ethnically different groups of the population, often characterized by differences in living conditions, dietary habits and lifestyle of both healthy persons and patients. Ultimately, this could allow the development of standards or correction factors for important indicators of echocardiography in each ethnic group [13, 14]. This was the basis for this study of one of the indigenous people's natives to the Gornaya Shoria region. Previous studies have shown differences in echocardiographic parameters of healthy individuals of the Shor nationality compared to Caucasians [11, 12]. The **aim of this study** was to study the echocardiographic parameters in urbanized patients with arterial hypertension of the Shor and non-indigenous ethnic groups living in the Gornaya Shoria region.

Methods

Patients

The study was conducted at medical facilities in Myski, including both indigenous (Shor) and non-indigenous subjects. The study design and inclusion and exclusion algorithms are described in detail in a previously published paper [12]. A total of 108 patients with arterial hypertension (aged 35 to 60 years) were included in the study: 58 Shors and 50 non-indigenous subjects (Figure 1). Anthropometry included measurement of height, weight, waist and hip circumference, calculation of body surface area (BSA), body mass index. The lipid profile was analyzed, studies such as electrocardiography, B-mode ultrasound examination of the carotid and femoral arteries were carried out. The exclusion criteria included a history or instrumental data of other cardiovascular diseases (ischemic heart disease, heart failure, peripheral atherosclerosis, transient ischemic attacks and previous stroke). Written informed consent was obtained from each patient. The study protocol was in accordance with the ethical principles of the 1975 Declaration of Helsinki. The study protocol was approved (date of approval: 9 February 2017) by the Local Ethical Committee of Research Institute for Complex Issues of Cardiovascular Diseases (Protocol No. 20170209) (Kemerovo, Russian Federation).

Echocardiography

All subjects underwent echocardiography on a Vivid S5 ultrasound system (GE Healthcare, Chicago, Illinois, USA) using a phased array sector probe. All measurements were carried out according to the current guidelines [15]. All echocardiographic examinations were performed by the same examiner (OA). All structural indicators were indexed to the BSA. We evaluated the structural parameters of the left heart: the diameter of the aorta and left ventricular chambers, the thickness of its walls, the volumes of the left ventricular chambers, the mass of the left ventricular myocardium, its ejection fraction by the Simpson method. To assess the diastolic function of the left ventricle, a pulse-wave Doppler mode was used (indicators E, A, DT, IVRT, E/A ratio). Spectral tissue Doppler sonography assessed indicators of regional function of the mitral valve ring related to diastolic (e', a', e'/a') and systolic parts of the spectrum (s'). The Tei index (a measure of overall LV performance) was also calculated, and early mitral flow velocity was measured in color M-scan mode.

Left ventricular hypertrophy was defined as LVMi > 95 g/m^2 for women and > 115 g/m^2 for men [15]. Relative wall thickness (RWT) was calculated using (IVSDd + PWDd) / LVEDd); concentric geometry was present when RWT > 0.42 [16]. LVMi and RWT were used to differentiate four types of left ventricular geometry: normal geometry (RWT ≤ 0.42 and normal LVMi), concentric remodeling (RWT > 0.42 and normal LVMi), concentric hypertrophy (RWT > 0.42 and increased LVMi), and eccentric hypertrophy (RWT ≤ 0.42 and increased LVMi). The severity of left ventricular hypertrophy was assessed according to the recommendations of Marwick TH et al. [16]. Mildly abnormal LVH was determined in women with LVMi

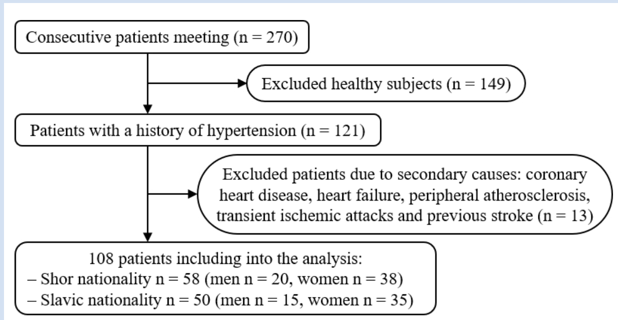

Figure 1. Protocol flowchart

within the range of 96–108 g/m², in men – at 116–131 g/m²; moderately abnormal LVH – at 109–121 g/m² and 132–148 g/m²; severely abnormal LVH – with LVMi ≥ 122 g/m² and ≥ 149 g/m², respectively.

The left ventricle diastolic function was assessed according to the ASE and EACVI recommendations [17]. Normal LV diastolic function was considered at values of e ≥ 8 cm/s, lateral e ≥ 10 cm/s and LAVi < 34 mL/m². In patients with e < 8 cm/s or lateral e < 10 cm/s and LAVi ≥ 34 mL/m², the grade of diastolic dysfunction from 1 to 3 was determined based on at least two indicators among the E/e (E ≤ 8, between 9 and 12 and ≥ 13 cm/s, respectively), the ratio E/A of mitral inflow (< 0.8, between 0.8 and 1.2 and > 2, respectively) and DT (> 200, between 160 and 200 and < 160 ms, respectively).

Statistical analysis

Statistical processing was performed using the standard Statistica 10.0 software package. The normality of the distribution was verified using the Shapiro–Wilk test. Since most of the quantitative characteristics were distributed asymmetrically, all quantitative variables were presented as the median, low and upper quartiles (ME [LQ, UQ]). Comparison of quantitative traits was carried out using the Kruskal–Wallis's test. Qualitative and binary characteristics were compared using the χ^2 (chi-square) test with Yates' correction for small samples. Intergroup differences were assessed using the Mann–Whitney test with Bonferroni's correction. Using logistic regression analysis, we studied the relationship of possible factors with severely abnormal LVH in all patients, and then separately in Shors and Caucasians. Multivariate regression analysis included variables for which the statistical significance in the univariate analysis was less than 0.1. After a preliminary assessment of the correlation between the identified predictors, several regression models were formed with the inclusion of independent factors. The level of statistical significance was taken as $p < 0.05$.

Results

The groups were comparable in age (Table 1). According to the results of anthropometry, both body weight and BSA were lower in men and women of Shor ethnicity than in men and women of non-indigenous ethnicity (p = 0.0014 and p = 0.004). Among the representatives of the Shor urbanized population, persons with secondary specialized education (p = 0.013) were significantly less common, especially among men (p < 0.01), while no significant differences between educational level of men and women within the Shor ethnic group was noted. Among the Shor men, there were significantly more people engaged in heavy physical and, generally, low-skilled labor than in all other groups (p = 0.01). Smoking was more prevalent among indigenous people (p = 0.003). The groups did not have significant differences in blood lipid spectrum parameters and in the level of office systolic and diastolic blood pressure, however, there was a tendency for the parameters to increase beyond the reference values in both groups. The Shor men and women had

ОРИГИНАЛЬНЫЕ ИССЛЕДОВАНИЯ

Table 1. General characteristics of hypertensive patients of various ethnic groups

	Patients of Shor nationality (n = 58)		Patients with non-indigenous ethnicity (n = 50)		H	p
	Men (n = 20)	Women (n = 38)	Men (n = 15)	Women (n = 35)		
Age, years	51.5 [45.0–55.0]	48.0 [43.0–53.0]	52.0 [41.0–55.0]	51.0 [44.0–55.0]	2.1	0.27
Weight, kg	65.0 [60.0–69.0]	71.0 [64.0–80.0]^	75.0 [65.0–90.0]*	79.0 [67.0–88.0]	10.8	0.005
Height, cm	161.0 [157.5–166.5]	156.5 [154.0–162.0]^	169.0 [166.0–171.0]*	160.0 [157.0–164.0]+	28.9	0.04
BSA, m²	1.7 [1.6–1.8]	1.8 [1.7–1.8]^	1.8 [1.7–1.9]*	1.8 [1.7–1.9]	9.6	0.004
BMI, kg/m²	24.61 [22.29–27.36]	29.14 [26.48–32.79]^	26.89 [23.22–31.24]	31.16 [26.98–33.79]	15.5	0.001
Obesity	0 (0%)	2 (5.3%)	2 (13.3%)	3 (8.6%)	2.9	0.41
Elementaryeducation	2 (10%)	2 (5.3%)	0 (0%)	1 (2.9%)	0.4	0.5
Secondaryeducation	9 (45.0%)	14 (36.8%)	1 (6.7%)*	11 (31.4%)	6.3	0.09
Vocationaleducation	8 (40.0%)	20 (52.6%)	14 (93.3%)*	20 (57.14%)+	10.7	0.013
Highereducation	1 (5.0%)	2 (5.3%)	0 (0%)	3 (8.6%)	1.5	0.68
Hardphysicallabor	16 (80.0%)	20 (52.6%)^	8 (53.3%)	16 (45.7%)	0.5	0.5
Smoking	14 (70.0%)	9 (23.7%)^	4 (26.6%)*	5 (14.3%)	11.5	0.003
Cholesterol, mmol/L	6.65 [5.2–7.3]	6.4 [5.9–6.9]	6.4 [5.4–6.9]	6.4 [5.9–7.4]	1.5	0.73
LDL, mmol/L	2.5 [2.0–3.9]	2.9 [2.3–3.7]	2.9 [2.5–4.1]	3.8 [3.1–4.1]	14.9	0.83
HDL, mmol/L	1.2 [1.1–1.5]	1.12 [0.96–1.59]	1.05 [0.92–1.2]	1.06 [0.94–1.2]	6.3	0.21
Glucose, mmol/L	5.14 [4.37–5.9]	4.7 [4.2–5.5]	5.8 [5.1–6.2]	5.9 [5.1–6.1]	12.9	0.33
SBP, mmHg	140.0 [125.0–150.0]	140.0 [130.0–150.0]	150.0 [130.0–160.0]	140.0 [130.0–150.0]	4.2	0.97
DBP, mmHg	80.0 [80.0–80.0]	80.0 [80.0–80.0]	80.0 [80.0–100.0]	80.0 [80.0–90.0]	6.9	0.44

Note: *Continuous data are presented as median (lower quartile, upper quartile). ^ – p < 0.05 between 1, 2 group; * – p < 0.05 between 3, 4 group, + – p < 0.05 between 3, 4 group. BMI – body mass index; BSA – body surface area; DBP – diastolic blood pressure; HDL – high-density lipoproteins; LDL – low-density lipoproteins; SBP – systolic blood pressure.*

significantly lower urea and creatinine levels than the corresponding non-indigenous groups.

In the Table 2 presents data on medication use in patients with hypertension across ethnic groups. Approximately half of the patients were unaware of their hypertension before the study. This explains the large number of patients who did not receive adequate therapy or were not treated at all. Overall, the groups were comparable in terms of medication use.

From the absolute echocardiographic parameters of the left heart (Table 3), no significant differences were found between the groups. The data of the longitudinal LV systolic function (Table 4) in the form of a systolic excursion of the septum (s'sept) were the highest in Shor men (p = 0.069), and the LV performance Tei index was the lowest in them (p = 0.034). Also in this group, the minimum isovolumic relaxation time (p = 0.065) was noted.

The degree of LVH in patients is shown in Figure 2. No statistically significant difference was observed among men and women in both ethnic groups with different severity of LVH. At the same time, pronounced LVH was somewhat more common among Shors than among non-indigenous residents (27.6% and 16.0%, respectively, p = 0.03).

The normal geometry of the left ventricle in the

Table 2. Medical therapy in patients with hypertension of different ethnic groups

	Patients of Shor nationality (n = 58)		Patients with non–indigenous ethnicity (n = 50)		p
	Men (n = 20)	Women (n = 38)	Men (n = 15)	Women (n = 35)	
Complete absence of antihypertensive therapy or occasional use	14 (70%)	21 (55,2%)	9 (60%)	19 (54,3%)	P_{1-3} = 0,537 P_{2-4} = 0,933
Beta-blockers	–	1 (2,6 %)	–	–	–
ACE inhibitors	4 (20 %)	4 (10,5 %)	5 (33,3 %)	6 (17,1 %)	P_{1-3} = 0,372 P_{2-4} = 0,964
Calcium channel blockers (amlodipine)	2 (10 %)	5 (13 %)	1 (6,7 %)	3 (8,6 %)	P_{1-3} = 0,727 P_{2-4} = 0,964
Diuretic monotherapy	–	3 (7,9 %)	–	1 (2,9 %)	P_{2-4} = 0,964
Combination therapy: ACE inhibitor + diuretic	–	4 (10,5 %)	–	6 (17,1 %)	P_{1-3} = 0,788 P_{2-4} = 0,964

Table 3. Structural parameters of echocardiography in hypertensive patients of various ethnic groups

	Patients of Shor nationality (n = 58)		Patients with non-indigenous ethnicity (n = 50)		H	p
	Men (n = 20)	Women (n = 38)	Men (n = 15)	Women (n = 35)		
Aorta, mm	35.5 [33.0–37.0]	37.0 [36.0–38.0]	38.0 [37.0–38.0]	37.0 [35.0–38.0]	8.14	0.06
Aorta index, mm/m^2	29.5 [25.4–31.0]	26.0 [24.0–30.0]	28.3 [22.9–31.2]	25.6 [22.2–30.0]	4.14	0.1
TPW, mm	11.0 [11.0–12.0]	12.0 [11.0–13.0]	11.0 [11.0–12.0]	11.0 [11.0–13.0]	1.25	0.38
TPWi, mm/m^2	9.1 [8.5–10.3]	8.6 [7.2–10.3]	8.8 [6.6–9.7]	8.5 [7.5–9.6]	3.7	0.15
TIVS, mm	12.0 [11.0–13.0]	12.5 [11.0–13.0]	11 [11.0–12.0]	12.0 [10.0–13.0]	3.99	0.18
TIVSi, mm/m^2	9.8 [7.9–10.7]	9.08 [8.09–10.4]	8.8 [6.6–9.7]	8.3 [7.2–9.5]	8.7	0.39
LVM, g	173.5 [155.5–193.0]	176.0 [159.0–214.0]	182.0 [167.0–196.0]	185.0 [161.0–205.0]	0.9	0.53
LVMi, g/m^2	141.9 [119.7–164.2]	132.0 [114.0–162.0]	127.4 [105.0–147.0]	134.0 [108.4–154.0]	1.7	0.37
EDD, mm	50.0 [48.0–53.0]	53.0 [48.0–54.0]	55.5 [53.0–56.0]	51.0 [49.0–55.0]	4.7	0.30
EDV, mL	109.5 [103.0–122.0]	132.0 [100.0–142.0]	135.0 [127.0–150.0]	124.0 [104.0–150.0]	6.1	0.15
LVEDVi, mL/m^2	89.4 [84.0–98.0]	86.7 [73.5–110]	102.0 [77.9–120.0]	89.8 [79.5–104.6]	1.5	0.46
LVEF, %	66.0 [62.0–69.0]	65.0 [63.0–68.0]	67.0 [64.0–70.0]	67.0[64.0–70.0]	3.2	0.52
LA, mm	38.0 [35.0–39.0]	38.0 [37.0–40.0]	39.0 [37.0–40.0]	39.0 [37.0–40.0]	4.4	0.26
LAV, mL	81.0 [50.0–89.0]	79.0 [57.0–88.0]	89.0 [68.0–91.0]	81.0 [66.0–89.0]	4.1	0.68
LAVi, mL/m^2	63.2 [41.9–72.5]	54.5 [44.5–62.5]	64.3 [47.8–69.7]	53.1 [40.4–66.4]	3.5	0.14
PA, mm	21.0 [20.0–21.0]	21.0 [20.0–22.0]	22.0 [20.0–22.0]	21.0 [20.0–22.0]	3.9	0.16
PAi, mm/m^2	17.2 [15.0–18.0]	15.7 [13.9–17.9]^	15.3 [12.6–17.6]	15.4 [13–17.4]	5.5	0.05
RA, mm	32.0 [31.5–35.0]	35.0 [32.0–36.0]^	35.0 [33.0–38.0]*	35.0 [32.0–38.0]	7.6	0.04
RAS, cm^2	11.5 [11.5–14.4]	14.4 [11.5–16.3]	15.4 [12.0–17.5]	14.2 [11.5–17.0]	5.4	0.07
RASi, cm^2/m^2	10.0 [8.4–11.7]	10.3 [8.7–11.7]	10.7 [9.3–12.0]	9.3 [7.6–11.5]	2.6	0.78

Note: *^ – p < 0.05 between 1, 2 group; * – p < 0.05 between 3, 4 group. EDD – end-diastolic diameters; EDV – end diastolic volume; i – indices of structural indica-tors to body surface area; LA – diameter of the left atrium; LAV – left atrium volume; LVEF – left ventricular ejection fraction; LVM – left ventricular mass; PA – pulmonary artery diameter; RA – diastolic dimension of the right atrium; RAS – right atrial square; TIVS – interventricular septum thickness; TPW – posterior wall thickness.*

whole cohort was equally common in the Shors and in the non-indigenous population ($p = 0.163$), and only in the Shors men the normal geometry of the left ventricle was less common compared to non-indigenous men ($p = 0.023$) (Figure 3). Accordingly, concentric LV remodeling was more common among Shors in general and among Shor men, although these differences did not reach statistical significance ($p = 0.364$ and $p = 0.255$, respectively). No significant ethnic differences in LV geometry were found in women.

Normal diastolic function of the left ventricle (Figure 4) was detected more often in Shor men than in men of non-indigenous nationality ($p = 0.029$). Accordingly, grade 1 diastolic dysfunction was detected more often in non-indigenous men than in Shors ($p = 0.002$). We did not reveal differences in the LV diastolic dysfunction prevalence in women of different ethnicities.

In univariate logistic regression analysis (Supplementary Table 1), the factor associated with severe abnormal LVH in the total sample was glucose (OR = 1.83, 95% CI 1.1–3.03, $p = 0.017$), which remained an independent factor in the multiple logistic regression model (OR = 1.79, 95% CI 1.08–2.97, $p = 0.020$). When carrying out univariate and multiple logistic regression analysis, the probable predictor associated with severe abnormal LVH in Shor patients was glucose level (OR = 3.31, 95% CI 1.6–6.85, $p = 0.001$ and OR = 3.24, 95% CI 1.56–6.70, $p = 0.001$, respectively). When carrying out univariate logistic regression analysis, the likely predictors associated with severe abnormal LVH in the group of Caucasian patients were SBP (OR = 1.04, 95% CI 1.0–1.08, $p = 0.021$), DBP (OR = 1.09, 95% CI 1.01–1.17, $p = 0.011$), heart rate (OR = 1.06, 95% CI 1.0–1.12, $p = 0.033$), and LDL cholesterol (OR = 1.99, 95% CI 1.04–3.79, $p = 0.034$). But in the multiple logistic regression model,

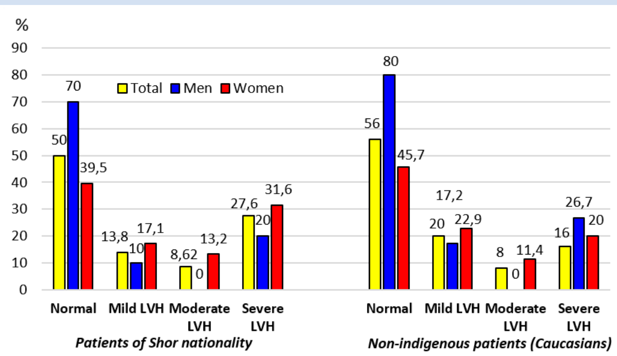

Figure 2. The severity of LVH in patients of different ethnic groups
Note: *LVH – left ventricular hypertrophy.*

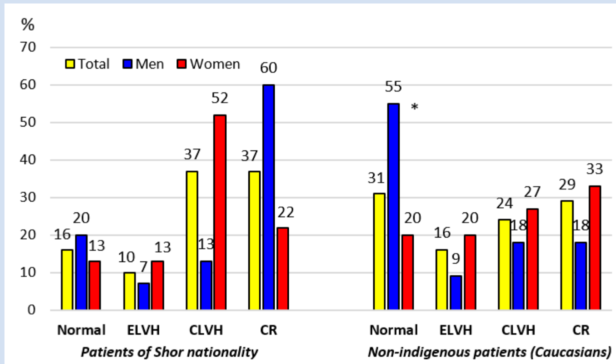

Figure 3. Left ventricular remodeling in patients of different ethnic groups
Note: ** – p < 0.05 compared with Shor men. CLVH – concentric left ventricular hypertrophy; CR – concentric left ventricular remodeling; ELVH – eccentric left ventricular hypertrophy; Normal – normal left ventricular geometry.*

Table 4. Indicators of diastolic function in patients with arterial hypertension of various ethnic groups

	Patients of Shor nationality (n = 58)		Patients with non-indigenous ethnicity (n = 50)		H	p
	Men (n = 20)	Women (n = 38)	Men (n = 15)	Women (n = 35)		
IVRT, msec	68.0 [60.0–81.0]	81.0 [74.0–90.0]	80.0 [67.0–104.0]	89.0 [70.0–100.0]	7.23	0.065
E, cm/sec	76.0 [64.0–89.5]	62.5 [56.0–76.0]	69.0 [57.0–94.0]	61.5 [53–76]	4.74	0.112
A, cm/sec	59.1 [47.1–73.1]	71.3 [57.5–82.0]	68.5 [48.2–83.0]	72.7 [58.7–82.0]	4.50	0.213
E/A	1.3 [1.1–1.54]	0.87 [0.75–1.15]	0.99 [0.63–1.28]	0.81 [0.71–1.27]	4.70	0.016
DT, msec	171.5 [148–200]	203.5 [163.0–222.0]	163.0 [115.0–222.0]	177.0 [137.0–214.0]	3.71	0.295
Vf, cm/sec	47.0 [42–50.5]	47.0 [35.0–53.0]	42.0 [39.0–54.0]	48.0 [39.0–60.0]	1.94	0.585
Lateral e’, cm/sec	12.0 [10.0–13.0]	11.5 [9.0–13.0]	11.0 [10.0–14.0]	11.0 [9.0–14.0]	0.58	0.901
Lateral a’, cm/sec	9.4 [6.5–12.0]	10.8 [8.2–12.8]	9.4 [6.7–12.03]	10.0 [9.2–12.6]	2.38	0.497
Lateral e’/ a’	1.36 [0.77–1.7]	1.0 [0.87–1.33]	1.04 [0.79–1.73]	1.2 [0.9–1.6]	1.72	0.634
Lateral s’, cm/sec	9.5 [9.0–12.0]	9.0 [7.0–10.7]	9.0 [7.8–13.0]	10.0 [8.0–12.0]	2.06	0.559
Septal e’sept, cm/sec	9.5 [9.0–12.0]	9.8 [7.0–12.0]	9.0 [8.0–11.0]	10.0 [7.8–11.0]	1.37	0.712
Septal e’ sept/ a’ sept	1.0 [0.73–1.2]	0.97 [0.7–1.1]	1.06 [0.6–1.3]	0.95 [0.73–1.3]	0.91	0.822
Septal s’ sept, cm/sec	10.3 [10.0–11.3]	9.0 [8.0–10.0] ^	9.0 [8.0–11.0]	9.0 [8.0–11.0]	7.11	0.069
E/e’	0.37 [0.30–0.44]	0.30 [0.26–0.41] ^	0.37 [0.29–0.47]	0.32 [0.28–0.47]	2.68	0.44
Tei, index	0.37 [0.29–0.44]	0.49 [0.40–0.58] ^	0.49 [0.42–0.73] *	0.50 [0.39–0.64]	8.65	0.034

Note: *^ – p < 0.05 between 1, 2 group; * – p < 0.05 between 3, 4 group. A – peak left ventricular filling velocity at atrial contraction; DT – deceleration time of early diastolic mitral flow; E – peak early diastolic left ventricular filling velocity; IVRT – isovolumic relaxation time; s’ – systolic mitral annular tissue velocity; Vf – flow propagation velocity; a’ – late diastolic mitral annular tissue velocity; e’ – early diastolic mitral annular tissue velocity.*

Suppl. Table 1. Factors Associated with severely abnormal LVH in hypertensive patients (Logistic Regression Analysis)

Parameters	OR (95% CI)	p
Model 1 All patients		
Univariate analysis		
Male	2.11 (0.71–6.31)	0.176
Age	1.03 (1.02–0.96)	0.366
Smoking	0.628 (0.24–1.65)	0.341
Smoking Experience	1.0 (0.96–1.05)	0.688
Physical activity	3.32 (0.62–17.64)	0.152
BMI	0.96 (0.89–1.06)	0.418
Waist circumference	0.99 (0.97–1.02)	0.941
Thigh circumference	0.99 (0.97–1.02)	0.991
Systolic blood pressure	1.02 (0.99–1.05)	0.117
Diastolic blood pressure	1.02 (0.97–1.07)	0.411
Heart rate	1.0 (0.68–1.0)	0.684
Glucose	1.83 (1.1–3.03)	0.017
Total cholesterol	1.05 (0.68–1.63)	0.800
Triglyceride	0.93 (0.55–1.58)	0.806
LDL cholesterol	1.12 (0.74–1.69)	0.591
HDL cholesterol	0.98 (0.49–1.96)	0.961
Urea	1.0 (0.98–1.03)	0.691
Creatinine	0.87 (0.67–1.13)	0.296
Multivariate analysis		
Glucose	1.79 (1.08–2.97)	0.020
Model 2 Patients of Shor nationality		
Male	1.52 (0.44–5.19)	0.495
Age	1.01 (0.95–1.08)	0.624
Smoking	0.47 (1.16–1.44)	0.186
Smoking Experience	1.01 (0.96–1.07)	0.463
Physical activity	8.93 (0.83–95.2)	0.066
BMI	0.9 (0.8–1.01)	0.098
Waist circumference	0.98 (0.95–1.01)	0.386
Thigh circumference	0.99 (0.94–1.04)	0.721
Systolic blood pressure	1.0 (0.97–1.03)	0.958
Diastolic blood pressure	0.96 (0.89–1.03)	0.281
Heart rate	0.97 (0.92–1.02)	0.250
Glucose	3.31 (1.6–6.85)	0.001
Total cholesterol	0.79 (0.48–1.29)	0.347
Triglyceride	0.9 (0.48–1.67)	0.749
LDL cholesterol	0.77 (0.45–1.29)	0.32
HDL cholesterol	1.18 (0.62–2.26)	0.598
Urea	1.0 (0.98–1.03)	0.509
Creatinine	078 (0.57–1.06)	0.110
Multivariate analysis		
Glucose	3.24 (1.56–6.7)	0.001
Model 3 Patients with non-indigenous ethnicity		
Male	3.6 (0.41–31.2)	0.239
Age	1.03 (0.94–1.14)	0.595
Smoking	1.28 (0.24–6.87)	0.766
Experience of Smoking	0.98 (0.91–1.06)	0.718
Physical activity	0.76 (0.1–5.5)	0.784
BMI	1.06 (0.93–1.22)	0.332
Waist circumference	1.02 (0.98–1.06)	0.281
Thigh circumference	1.01 (0.95–1.07)	0.632
Systolic blood pressure	1.04 (1.0–1.08)	0.021
Diastolic blood pressure	1.09 (1.01–1.17)	0.011
Heart rate	1.06 (1.0–1.12)	0.033
Glucose	1.06 (0.69–1.62)	0.782
Total cholesterol	1.9 (0.89–4.11)	0.095
Triglyceride	1.01 (0.45–2.29)	0.963
LDL cholesterol	1.99 (1.04–3.79)	0.034
HDL cholesterol	0.26 (0.1–3.7)	0.324
Urea	0.99 (0.96–1.03)	0.792
Creatinine	1.12 (0.73–1.71)	0.59

Note: *BMI – body mass index; CI – confidence interval; HDL – high-density lipoproteins; LDL – low-density lipoproteins; OR – odds ratio.*

none of these factors were independently associated with severe abnormal LVH.

Discussion

Our study has found no differences in the structural parameters of the left heart and the severity of left ventricular hypertrophy dependent on ethnicity of hypertensive patients. Normal geometry of the left ventricle was more common in non-indigenous men than in Shor men. At the same time, Shor men were more likely to have normal LV diastolic function compared to non-indigenous men. The LVH severity was associated with glucose levels in Shors, and with blood pressure and heart rate in Caucasians.

The obtained results were somewhat unexpected for us. In the previous study, when comparing healthy people of Shor ethnicity and Caucasians, the differences in structural parameters were more noticeable, in particular, there was a significant increase in the indices of the end-diastolic diameter of the LV, left atrium and diameter of the pulmonary artery in men and women of Shor nationality relative to Caucasians. Moreover, there was a significant increase in the indices of the aorta diameter, of the LV posterior wall and interventricular septum thickness, and the mass of the left ventricular myocardium among indigenous women [11]. Such differences can be partly explained by the younger age of the examined healthy persons. In our opinion, in this case, this does not indicate the presence of "hypertensive cardio-myopathy" in the absence of hypertension, since differences in LVMi in people of different ethnicity were noted earlier in multicenter registries [18–21]. In particular, it was shown that the values of LVMi in women from different regions of Asia were higher than in Caucasian women [21]. Apparently, development of arterial hypertension and age-related changes leveled most of the initial ethnic differences in the compared cohorts. Similar data were obtained in the study of rural residents of Gornaya Shoria; it was shown that the LV myocardial mass index in the groups of hypertensive patients of the indigenous and non-indigenous population did not differ, although the absolute values of the end-diastolic size and LV myocardial mass were higher in Caucasians [18].

In previous studies, the following patterns were noted: LV myocardial mass was lower in healthy representatives of South Asia than in Caucasians and Afro-Caribbean in the SABER study [19]. A meta-analysis by Poppe KK et al., noted a significant geographic variability of LV myocardial mass values in healthy individuals. Lower values of LVMM were found in residents of Asia and South America compared with representatives of Europe and North America. The review also found that BSA indexing does not improve the relevance of recommended reference ranges for either sex and does not seem to account for differences between populations [20]. That study was limited by the lack of LV myocardial mass analysis in individual ethnic groups, since ethnicity was limited to definition at the country level, and the details of ethnic composition depended on the focus of a separate study.

The results of the EchoNoRMAL meta-analysis led to a proposition for standards for left ventricular indices in various ethnic groups [21]. Thus, the normative values of LVMM in men were lower in the Middle East, and even lower in South Asians as compared to Caucasians. On the other hand, LVMM values were lower in Caucasian women than in residents of different regions of Asia [21]. At the same time, studies in healthy individuals cannot be extended to cohorts of patients with various pathologies; this requires additional studies [15]. Different results of the echocardiography parameters comparing Shors and Caucasians among healthy individuals and hypertensive patients in our study are consistent with this statement.

In the WASE study, the protocol for assessing left ventricular parameters was standardized, with a wider representation of Asian countries (41.8% of subjects). That work found significant cross-country variations for all LV parameters reflecting LV size (size, mass, and volume), even after indexing body surface area, with LV end-diastolic and end-systolic volumes having the greatest variation. For both sexes, the largest volumes were recorded in Australia and the smallest were found in India. This finding suggests that, in addition to gender and body surface area, country-specific assessments of LV volumes should be considered ($p < 0.05$ for all). While the differences in the main parameters of the LV were insignificant between Asians and Caucasians [10], the other races had the smallest values of the volumes and mass of the LV myocardium. One gets the impression that the representatives of the Shor nationality in this regard are closer to the indicators of the Asian ethnic groups.

Fewer studies have focused on comparing left ventricular diastolic function in individuals of different nationalities. Interestingly, the recently published results of the WASE study [22] have shown that Asians had lower e 'speeds, higher E/e' and lower LAVIs in almost all age groups compared to Caucasians. Similar findings were reported by Chahal et al. [23]: while comparing data from healthy Caucasians and Indian Asians living in London, Asians had a significantly lower e 'rate and a higher E/e' than whites with similar E and E / A rates. Lower indices of the main LV diastolic function parameters served as the basis for the development of separate standards for ethnic Asians in a recently published study [24]. Nevertheless, in our study, E/A rates were higher in Shors men than non-indigenous men of Gornaya Shoria with arterial hypertension, differences in diastolic function were noted in Shors and non-indigenous women. It is quite possible that the reason for this is leveling of the initial differences following the development of the pathological changes.

Unlike non-indigenous ethnicity in Shors with hypertension the only independent predictor of severe left ventricular hypertrophy was glucose levels, not blood pressure and heart rate. Indeed, structural myocardial changes characterized by the development of left ventricular hypertrophy have been observed in diabetes mellitus [25]. We did not include patients with diabetes mellitus in this study, but we did not conduct targeted screening for prediabetes. At the same time, prediabetes can also be accompanied by impaired left ventricular function, including the development of heart failure [26]. Patients with prediabetes (detected by HbA1c levels) also showed a decreased E/A ratio, higher left atrial volume, and sphericity index compared to the control group [27]. Furthermore, impaired glucose metabolism, including prediabetes, was shown to be associated with increased left ventricular wall thickness, independent of hypertension [28]. It appears that Shors with arterial hypertension should additionally be actively screened for carbohydrate metabolism disorders.

In rural residents of Gornaya Shoria, the LV hypertrophy prevalence in hypertensive patients was higher in the Shor group than in the non-indigenous (Caucasian) group. At the same time, various genetic associations with LV hypertrophy were established in different ethnic groups. Among the Shors, these were the MTHFR gene according to the log-additive mode of inheritance, the A/G genotype of the ADRB1 gene among persons with normal body weight, the I/D genotype of the ACE gene among men; among Caucasians, the D/D genotype of the ACE gene for the codominant mode of inheritance, the NOS3 gene for the log-additive mode of inheritance [18]. In addition to genetic factors, many factors affect LV myocardial mass, including age, sex, body size and composition, level of physical activity, blood pressure [29], and presence of other diseases (for example, diabetes mellitus). In healthy representatives of the Shor nationality living in rural areas, in addition to genetic factors, LVMM is also influenced by the traditional way of life, which includes hunting, gathering, and high physical activity in the middle mountains. In our sample, living in a city significantly changes the lifestyle of the Shors, and we have not identified any differences in the severity of LVH, only noting differences in the factors associated with the LVH presence. Our data are consistent with the opinion that the influence of environment seems to be critical in explaining most of the ethnic differences in LV mass when compared with genetic factors [29].

What is the clinical significance of this study? While previous studies have found ethnic differences in geographically diverse populations, the impact of changes in environment and socioeconomic status remains unknown. Consequently, it remains unclear whether the normal performance for a particular ethnic group living in their home area will be the same compared to people of the same ethnic group living in a culturally and economically different environment? Changes in the habitat often entail changes in diet, lifestyle and, as a result, in physique, which can affect the norms of echocardiography values based on indexing body surface area [29]. In this light, the fact of leveling of ethnic differences in the frequency of detection of LV hypertrophy in arterial hypertension when examining a cohort of urban residents in comparison with previously identified patterns in rural residents is of undoubted interest. This can be a useful addition to studying of adaptation to changing environmental conditions due to the intensive industrial development of the Gornaya Shoria region, urbanization and food system transformation [14], as well as the possibility of influencing these mechanisms in order to improve the quality of life and prevent cardiovascular disease risk factors.

It would be interesting to study the effect of drug therapy on echocardiographic parameters in this ethnic group. For example, in previous studies in patients with CHF, conflicting results have been obtained. So, in patients with dilated cardiomyopathy, drug therapy causes favorable remodeling of the heart, regardless of ethnicity (south or north of Europe) [30]. At the same time, the use of sacubitril/valsartan in patients with symptomatic HF-rEF improvement in LV EF was less pronounced in black patients compared with Caucasians [31]. However, the cross-sectional design of this study does not allow us to investigate this issue, which requires prospective studies.

Although the Shors are a small people (the total number of which is about 14 thou-sand people), however, they are close to other Turkic-speaking peoples living in Siberia (Altaians, Khakasses, Chulyms, etc.). Therefore, the scientific results obtained by us can be useful for studying representatives of both other small peoples of Siberia and other representatives of the Mongoloid race in Asia.

Study limitation

A limitation of the study is the relatively small number of patients included. This was due to the relatively small number of Shors living in urban areas. The data obtained in this study, due to the small size of the sample, cannot be generalized to the entire Shor population and give not a global, but a preliminary idea. Also, the effect of obesity on the structural and functional parameters of the heart was not considered due to the small number of Shor men with increased body weight. There was a gender imbalance in our sample (only 35 from 108 patients were male), which was one of the limitations of the study. Age at menopause in women was not considered, which may have influenced the study results in female ethnic groups. In this work, we did not compare the age indicators of the structure and function of the ventricles, since the study groups were comparable in age. Furthermore, the present study did not conduct screening carbohydrate metabolism disorders, which prevented the identification of prediabetes in the examined cohort. Also, we did not use more modern methods for assessing the state of the left heart (for example, 3D-echocardiography), but our results are quite consistent with real clinical practice, where patients with arterial hypertension are usually examined using routine transthoracic echocardiography. Conducting echocardiography by one operator, on the one hand, allowed us to avoid the need to assess reproducibility between operators. On the other hand, it becomes impossible to determine to what extent the results can be reproduced by different people. Therefore, the use of a single operator in this study is another of its limitations, potentially leading to biased or less reproducible results. Also, the cross-sectional design of the study does not allow for establishing causal relationships in the identified associations.

Conclusion

In the present study, hypertensive urbanized Shors had no significant difference in left ventricular dimensions compared to Caucasian. The LVMM index did not differ between the ethnic groups; however, the severe abnormal LVH was associated with glucose level in the Shors, and with SBP, DBP, and heart rate in Caucasians. The Shor men were more likely to have normal LV diastolic function and LV concentric remodeling, while non-indigenous men had normal LV geometry more often. This study can be useful in assessing the impact of the development of arterial hypertension, as well as the impact of changing the lifestyle of the Shors when moving to the city from rural areas on ethnic differences in echocardiography.

Conflict of interest

N.S. Gomozova declares no conflict of interest. A.N. Sumin is a member of the editorial board of the journal “Complex Issues of Cardiovascular Diseases”. A.V. Shcheglova declares no conflict of interest. O.G. Arkhipov declares no conflict of interest.

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GNS – data collection, analysis and interpretation, contribution to the concept of the study, editing, approval of the final version, fully responsible for the content

SAN – contribution to the concept and design of the study, data collection and interpretation, manuscript writing, approval of the final version, fully responsible for the content

ShchAV – data collection and interpretation, manuscript writing, approval of the final version, fully responsible for the content

AOG – data collection, editing, approval of the final version, fully responsible for the content

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