

1. Introduction

One of the pressing problems of modern cardiology is the study of the etiological and risk factors for SCD. Arrhythmias, which in most cases correlate with signs of myocardial hypertrophy, are important for its occurrence. The most common cause of LVH is hypertension. However, an equally important trigger mechanism for malignant arrhythmias is CHD.

In CHD, myocardium is affected; metabolic processes and energy metabolism in cardiomyocytes are disrupted, which in turn leads to arrhythmias [1].

LVH is known to be considered as an independent risk factor for cardiovascular complications (myocardial infarction, heart failure (HF), arrhythmias, etc.) [2]. Among the mechanisms of the development of LVH may be considered an episode of hypoxia and ischemia, which in turn leads to the development of post-ischemic cardiomyocyte dysfunction and an increase in energy deficiency in them [2].

Studies have shown that there is some pattern between the mass of the left ventricle (LV) myocardium and the occurrence of cardiovascular catastrophes, including SCD. It has been proved that the myocardial need for oxygen increases and its ectopic activity increases with LVH [3].

It is known that in concentric LVH the thickness of the walls of the myocardium increases and the volume of the cavity of the LV decreases, while in the eccentric, on the contrary, the normal thickness of the walls of the LV decreases and the volume of its cavity increases. Literary evidence indicates that concentric LVH is associated with the highest risk of cardiovascular complications and the worst prognosis [4].

Studies have shown that the larger the LV myocardial mass index (LV MMI), the greater the size of the LA. It is proved that with increasing size of LA for every 5 mm the relative risk of atrial fibrillation increases 4 times [5].

Not all arrhythmias are equally significant for the prognosis of the patient's disease and life. In this regard, the clinical and prognostic classification of arrhythmias and cardiac conduction disorders, depending on their severity, is of some clinical importance for predicting the occurrence of various cardiovascular complications and their potential for prevention [6].

The literature describes the importance of LVH in the development of cardiac arrhythmias, which in turn lead to cases of SCD. However, it should be noted that there are insufficient re-

LEFT VENTRICULAR HYPERTROPHY AND LEFT ATRIAL DILATATION AS MARKERS OF MALIGNANT ARRHYTHMIAS DEVELOPMENT

Yuliia Shushkovska

Associate Professor¹

suskovskaaulia@gmail.com

Oksana Afanasiuk

PhD, Associate Professor¹

o.afanasiuk@gmail.com

¹*Department of Internal Medicine No. 3*

Pirogov National Memorial Medical University

56 Pirogova str., Vinnytsya, Ukraine, 21018

Abstract: One of the pressing problems of modern cardiology is the study of the etiological and risk factors for sudden cardiac death (SCD). Arrhythmias, which in most cases correlate with signs of myocardial hypertrophy, are important for its occurrence. The most common cause of left ventricular hypertrophy (LVH) is hypertension. However, an equally important trigger mechanism for malignant arrhythmias is coronary heart disease (CHD). It is known that LVH is considered as an independent risk factor for cardiovascular complications.

Methods. The study design included an assessment of the effect of cardiac remodelling, the degree of LVH, and the degree of dilatation of left atrium (LA) on the development of malignant disorders of rhythm and conduction of the heart (DRCH). 78 patients with II degree of hypertension were examined. Patients were divided into two groups depending on the presence of CHD. In each group, there were malignant rhythm and conduction disorders. The control group consisted of 20 healthy people.

Results and discussion. The stages of LVH were equally represented in both groups, but the concentric LVH, the initial degree of LVH, and the dilatation of LA predominated. In patients with malignant DRCH, a moderate degree of LVH predominated. Significantly, more likely malignant DRCH occurred in the second group and presented with high-grade ventricular extrasystoles, prolonged QT interval, and sinus node weakness syndrome.

Keywords: malignant arrhythmias of the heart, hypertension, coronary heart disease, left ventricular hypertrophy.

ports on the features of the types and degrees of LVH, dilatation of the LA, which would most likely lead to malignant arrhythmias.

The purpose of the study was to investigate the possible effects of different types and degrees of LVH and LA dilatation on the development of malignant arrhythmias in arterial hypertension, depending on the presence or absence of CHD.

2. Material and methods

The study was conducted at the Vinnytsa Regional Clinical Medical Diagnostic Center for Cardiovascular Pathology during 2014–2017.

The criteria for inclusion in the study were patients with hypertension, various DRCH with or without CHD. Exclusion criteria were acute coronary syndrome, HF of IV functional class (FC) according to NYHA classification, presence of permanent atrial fibrillation, previously diagnosed idiopathic cardiomyopathies, myocarditis, severe kidney disease, liver, lung, and neoplasm. The study design included an assessment of the effect of cardiac remodelling, the degree of LVH, and the degree of LA dilatation on the development of malignant DRCH. 78 patients were examined, including 47 men and 31 women, with an average age (62.18 ± 0.64) years. Systolic blood pressure was, on average, determined (154.97 ± 2.20) mm Hg. art., diastolic blood pressure – (90.18 ± 1.91) mm Hg. art.

The study was conducted according to basic bioethical standards Helsinki Declaration

of the World Medical Association on Ethics principles of scientific and medical research as amended (2008), Universal Declaration of Bioethics and Human Rights (1997), Convention Council of Europe for Human Rights and Biomedicine (1997).

The duration of hypertension in average was 12.46 ± 2.66 years, CHD – 7.35 ± 0.62 years. HF of II FC according to NYHA classification was determined in 62 (79.49 %), III FC – in 16 (20.51 %) patients. Patients were divided into two groups. The first group (n=40) consisted of patients with hypertension and a variety of DRCH, but without existing CHD. The second group (n=38) – with hypertension and available DRCH and CHD. In each group, there were malignant rhythm and conduction disorders. The control group consisted of 20 healthy people.

The patients underwent a standard comprehensive clinical laboratory and instrumental examination: ECG in 12 conventional leads, blood lipid spectrum, blood glucose, biochemical

analysis of blood, Echocardiography in M- and B-modes, Holter ECG monitoring, 6-minute stroke test angina or HF.

Determined the types of LV remodelling according to Genau (1992), and the degrees of LVH by V. Denesiuk, O. Denesiuk (Table 1) [6, 7].

Table 1

Criteria for the diagnosis of degrees of left ventricular hypertrophy in cardiovascular diseases according to Echocardiography

Degrees of LVH according to echocardiography	Left ventricular myocardial mass index, g/m ²	Thickness of the interventricular septum, cm
In healthy people	male ≤ 110	male ≤ 1.0
	woman ≤ 95	woman ≤ 0.9
I (initial)	male 111–170	male 1.01–1.19
	woman 96–160	woman 0.91–1.15
II (moderate)	male 171–205	male 1.20–1.34
	woman 161–190	woman 1.16–1.29
III (significant)	male ≥ 205	male ≥ 1.35
	woman ≥ 191	woman ≥ 1.30

Note: 1 – in cases where the parameters of LV MMI and the thickness of the interventricular septum (TIVS) are different and do not correspond to the specified degree of LVH, as a basis, it is advisable to take LV MMI, on which to determine its degree of expression; 2 – LV MMI is normal, and the index of TIVS corresponds to the II degree of LVH, then we expose the I degree of LVH

Along with this used the proposed prof. Denesiuk VI et al. criteria for the diagnosis of the degree of dilatation of LA [6]. So, the I degree of (initial) dilatation of the LA corresponded to 4.0–4.4 cm, the II (moderate) degree – 4.5–5.0 cm, the III (significant) degree – ≥ 5.1 cm.

Professor Denesiuk V.I. published an improved clinical-prognostic classification of life-threatening arrhythmias and heart blockade [2] in 1999 and in 2015 [6]. Thus, malignant DRCH included ventricular extrasystole 3–5 gradations according to Laun, complete AV blockade with syncopal states, ventricular paroxysmal tachycardia (especially “pyruvic tachycardia”) with increasing disorders of hemodynamic, sinus node weakness syndrome, QT prolonged syndrome, Brugada syndrome, and idioventricular rhythm.

All data obtained from the study were processed using SPSS software by methods of variational statistics minus the mean M, the standard deviation S, the mean error of the mean m, the confidence criterion t, the confidence value p. The difference at $p < 0.05$ was considered statistically significant. The investigated values are given in the form ($M \pm m$). Pairwise group comparisons were performed using the non-parametric Mann-Whitney method.

3. Results

Among patients of group I, without CHD, 5 people (12.5 %) had grade I of hypertension, 25 people (62.5 %) had grade II, 10 people (25 %) had grade III of hypertension. There was no significant difference between of hypertension rates in group II patients (with CHD). Myocardial infarction occurred in 3 (7.89 %) patients of group II.

Acute damage of cerebral circulation was anamnestic manifested in 1 (2.5 %) patient of group I and in 2 (5.26 %) – group II.

Each group was further divided into two subgroups depending on the presence of malignant arrhythmias. Thus, in group I (without CHD) malignancies occurred in 18 (45 %) patients who did not have a significant difference depending on the indicators of pressure, degrees and duration of hypertension. Malignant DRCH occurred in 19 (50 %) patients of group II (with CHD), but significantly ($p > 0.05$) occurred more frequently among patients with stable angina or existing HF of FC III, as confirmed by other authors [8]. However, they also had no significant difference depending on the indices of pressure, degrees and duration of hypertension, duration of CHD, anamnestic myocardial infarction and acute damage to the cerebral circulation. The second degree of hypertension was prevalent among all patients.

Echocardiography was performed on all patients. Comparative analysis was subjected to a control group (healthy patients) with each group of patients with hypertension. The likelihood of difference ($p > 0.05$) of LV MMI values, LA size, volume systolic and diastolic indices, and ejection fraction (EF) of patients without CHD and CHD compared with healthy people and the probability of difference ($p \leq 0.01$) values of the interventricular and posterior walls of the LV compared with healthy people. However, no validity was found between and without cohorts of patients with CHD, suggesting the same development of LVH in patients in both cohorts, which was not dependent on the development of changes in CHD. In addition, no significant differences were found in the comparative characteristics of patients with malignant arrhythmias in patients in both groups. The results of echocardiography indicated the development of different types of LVH with preserved systolic heart function. Such changes lead to impaired cardiac muscle relaxation and increase myocardial stiffness, promoting the development of HF and capable of provoking the onset of DRCH.

The conducted analysis of the Echocardiography study allowed dividing the patients by types of heart remodelling according to Genau, degrees of LVH by V. Denesiuk, O. Denesiuk and the degree of dilatation of LA of both groups and depending on the available malignant arrhythmias. No significant differences were found in patients from both groups and when comparing patients with malignant DRCH. However, it should be noted that all patients were dominated by concentric hypertrophy for Genau, which confirms a higher incidence of DRCH. This type of hypertrophy of the LV occurs as a compensatory mechanism, ensuring normal circulation with elevated blood pressure. Over time, the compensatory properties weaken, the incidence of HF increases, and the risk of SCD increases significantly [9]. The mean was (77.19 ± 2.49) % and eccentric hypertrophy was (15.5 ± 3.25) %. When comparing the rates of VH by V. Denesiuk, O. Denesiuk, the patients of both groups showed a predominance of the initial degree of LVH, which was already accompanied by the development of DRCH and the initial degree of dilatation of LA. In comparison of both groups with malignant arrhythmias – a moderate degree of LVH and a significant degree of predominance $p > 0.05$ there was a more frequent occurrence of LA dilatation in patients with hypertension in combination with CHD and existing malignant DRCH, which can be caused by the increase of paroxysmal and persistent forms of atrial fibrillation, which according to V. I. Denesiuk was not classified as malignant.

4. Discussion and conclusions

Comparative characteristics of the types of malignant DRCH in two groups were conducted. Thus, the most common type of malignant DRCH were ventricular extrasystoles 3–5 gradations according to Laun, which occurred significantly ($p>0.05$) more frequently in patients of group II (with coronary heart disease). In addition, in second group, 1 (2.63 %) patient experienced QT prolongation on the background of amiodarone administration and 2 (5.26 %) patients experienced sinus node weakness syndrome, which was absent in group I patients. It should be noted that all DRCH in both groups occurred only with hypertrophic LVH and the initial stages of LVH and dilatation of LA. The obtained data are confirmed by other authors [10], who indicate an increase in the frequency of QT prolongation on the background of arrhythmic drugs, with an increase in the degree of LVH.

In patients with hypertension and coronary heart disease, malignant rhythm and conduction of the heart are significantly more likely to occur with a greater degree of functional class of stable angina and heart failure, and regardless of the duration of coronary heart disease and hypertension.

The highest frequency of various malignant disorders occurs in concentric left ventricular hypertrophy, regardless of the presence of coronary heart disease.

Malignant abnormalities of rhythm and conduction of the heart occur even with the initial degree of left ventricular hy-

pertrophy and left atrial dilatation and increase with hypertrophy in patients in both groups.

Among the malignant disorders in the overwhelming number occur ventricular extrasystoles 3–5 gradations according to Laun in both groups, and significantly more often in combination with coronary heart disease, contributes to the development of the probability of acquired QT syndrome against the background of taking amiodarone, which may in turn develop malignant and fatal heart rhythm disorders.

Coronary heart disease may be an additional provocative risk factor for malignant heart rhythm disorders.

The proposed determination of the degree of left ventricular hypertrophy and left atrial dilatation in patients with hypertension allows to evaluate in more detail and specifically the cardiac remodelling and possibly to determine the gradual regression of left ventricular hypertrophy on the background of treatment.

Taking into account the revealed data on the dependence of the development of malignant arrhythmias on the functional class of stable angina and heart failure, it is necessary to evaluate the influence of indicators of lipid metabolism and the degree of damage of the coronary bed atherosclerotic process on the development of malignant disorders of rhythm and conduction.

Conflict of interests

No conflict of interest.

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Received date 06.09.2019

Accepted date 16.10.2019

Published date 30.10.2019

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