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# Formerly Larboard Ventricular Hypertrophy in Arterial Hypertension and Cardiopathy Jeopardy

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**ANNOTATION:** Left ventricular hypertrophy (LVH) is an important, independent risk factor for not only total and cardiovascular mortality, but also for sudden cardiac death. Following blood pressure increase in hypertrophied LV, due to myocardial diastolic dysfunction, left atrium dilates very fast. That results in supraventricular extrasystolia, atrial fibrillation and flutter in 25750% of the patients with arterial hypertension (AH). The link between LVH and ventricular arrhythmia incidence and severity depends on LVH stage, and might be absent at mild to moderate, close to physiological, stages. Association between LVH and spontaneously induced ventricular arrhythmia has been demonstrated in adequately controlled experimental studies. In AH and LVH patients, arrhythmogenic risk factors include the following: late ventricular potentials, decreased heart rate variability, prolonged QRS duration, increased T interval dispersion and T wave alteration. Assessing arrhythmia risk in asymptomatic patients is a difficult task, that could be solved with an algorrhytm proposed.

**KEYWORD:** Arterial hypertension, left ventricular hypertrophy, arrhythmogenic risk.

Arterial hypertension (AH) is one of the leading risk factors for cardiovascular diseases (CVD) and mortality [8]. The presence of hypertension doubles the risk of coronary heart disease and more than triples the risk of developing chronic heart failure (CHF) and stroke [17]. In patients with high blood pressure (BP), disorders of the structure and function of the myocardium often occur: left ventricular hypertrophy (LVH), systolic and diastolic myocardial dysfunction, supraventricular and ventricular arrhythmias, sometimes LVH is an important independent FR of not only general and cardiovascular mortality, but also sudden cardiac death (SCD) [18].

The earliest change in the hypertensive heart is an increase in myocytes with an expansion of their transverse diameter, which is difficult to assess using routine microscopy. In the future, the increase in the nuclei and the cells themselves becomes partially irregular, adjacent myocytes of different sizes appear, which have lost myofibrils, and interstitial fibrosis develops [19]. The progression of LV remodeling and the formation of LV occurs under the influence of a huge number of factors, among which we can distinguish the main, hemodynamic and non-hemodynamic. Using the echocardiographic (EchoCG) method, LV architectonics in patients with hypertension is divided into four geometric models using indicators of myocardial mass (MM) and relative wall thickness (OTC)



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of the LV. The LV OTC index is a sensitive indicator of the geometric model in LVH and is determined by the ratio of LV TC to the transverse diameter of its cavity at the end of the diastole.

Various geometric models of LV remodeling according to EchoCG data:

- ✓ concentric hypertrophy – increase in MM and LV OTC;
- ✓ eccentric hypertrophy – an increase in MM with normal OTC;
- ✓ concentric remodeling – normal MM and increased OTC;
- ✓ Normal LV geometry.

Observation of 253 patients with initially uncomplicated essential hypertension for 10 years confirmed that the frequency of cardiovascular complications (CVD) and mortality are quite strictly dependent on the geometric model of LV. The worst prognosis for MTR (31%) and mortality (21%) was observed in the group of patients with concentric LVH. The most favorable prognosis is the absence of fatal outcomes and 11% MTR in the group of patients with normal LV geometry [12].

Differences in the LV structural and geometric model in patients with hypertension are closely related to the pathophysiology of the heart and blood circulation. Patients with concentric LVH are characterized by almost normal end systolic myocardial stress, normal LV size and shape, increased total peripheral vascular resistance (OPSS)and a slight increase in the cardiac index (SI).

Patients with concentric remodeling are also characterized by a normal level of end systolic myocardial stress and an increase in OPSS. At the same time, they have a reduced impact index (CI) and SI. The reason for the increase in LV OTC in this group is not fully understood. In part, this increase can be explained by a decrease in the elasticity of the arteries, as indicated by a subnormal shock volume with a slight increase in pulse blood pressure. Patients with eccentric LVH are characterized by high SI, normal OPSS, an increase in the LV cavity, end systolic myocardial stress, indicating the inadequacy of LVH. As hemodynamic prerequisites for the formation of this geometric model, a predominant increase in venous tone or the volume of circulating blood is given. The absolute majority of patients with hypertension has normal LV geometry and is characterized by a slight increase in OPSS, systolic and diastolic blood pressure. Following an increase in pressure in the hypertrophied LV against the background of diastolic myocardial dysfunction, the left atrium (LP) expands very quickly. This leads to the occurrence of supraventricular extrasystole (NE), fluttering and atrial fibrillation (TP and AF) in 25%750% of patients with hypertension. According to the results of the Manitoba FollowUp Study, it is known that the frequency of AF increases by 1.42 times in men with a history of hypertension [14]. Patients with LVH suffered supraventricular tachycardia (LVT) in 27.3% of cases ($p<0.05$), and the amount of LVH correlated with the size of LP [11]. When observing 2482 patients with essential hypertension without endocrine or other CVD for 16 years, it was concluded that in untreated patients with hypertension with sinus rhythm, age and MMLF are independent predictors of the development of AF, and the size of LP is an additional factor in the subsequent chronization of AF [15].

The development of ventricular arrhythmias is an important mechanism determining high cardiovascular mortality and SCD in patients with LVH [13]. The relationship between LVH, frequency and severity of ventricular arrhythmias depends on the degree of hypertrophy and may be absent in the early and middle stages of LVH closer to physiological.

The association between LVH and spontaneous induced ventricular arrhythmia has been confirmed in well-controlled, experimental studies [16]. There is evidence that LVH regression leads to the restoration of normal electrical and structural properties of the myocardium, to a decrease in the


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frequency and complexity of ventricular arrhythmias [4,11]. In part, the risk is associated with the pro-arrhythmogenic electrophysiological phenotype of hypertrophied myocardium, as well as with the presence of myocardial ischemia and a number of other factors.

Arrhythmogenic risk factors in patients with hypertension with LVH [8]:

- ✓ Reduction of heart rate variability (HRV)
- ✓ Increase in the duration of the action potential (PD)
- ✓ Increase in the dispersion of the QT interval (formation of a long QT interval)
- ✓ T Wave Alternations
- ✓ Identification of late ventricular potentials (PPV).

PPJ are low–amplitude signals that can be registered with the help of special equipment at the end of the QRS complex. The presence of PPV in 10% of patients with hypertension is associated with a violation of the contractile function of the ventricles [15]. Indeed, patients with AH with LVH are more often diagnosed with PPV than patients without it [14]. The prognostic significance of these changes as an indicator of the occurrence of arrhythmias is still being discussed, since the correlation between the presence of PPV and total cardiovascular mortality or SCD remains not fully confirmed [9]. HRV is a very important independent predictor of overall mortality and SCD in various clinical conditions – from acute myocardial infarction (AMI) to CHF [1]. As a result of several studies, it became possible to conclude that the presence of AH and LVH is associated with a significant decrease in HRV [1,5]. It is believed that this reflects a shift in the physiological sympathoparasympathetic balance in the direction of sympathicotonia and a decrease in vagal influence on the sinus node. It is quite difficult to analyze the relationship between a decrease in HRV, LVH and cardiovascular mortality, since the influence of neurohumoral and structural factors involved in the formation of hypertension is much more pronounced than specific arrhythmogenic ones. As a result of recent studies, it was found that in patients with hypertension, an increased variance of the QT interval is associated with an increased risk of ventricular arrhythmias and SCD [6,9,15]; a correlation with the presence and severity of LVH was revealed [6,15]. An increase in the duration of PD is an important diagnostic sign of the risk of arrhythmias. This can cause early post-depolarization and trigger activity. And even in an intact heart, an increased dispersion of repolarization can support arrhythmia. And in the hypertrophied myocardium, disorganization of myofibrils, heterogeneity of the distribution of intercellular gaps and channels, interstitial fibrosis significantly increase the number of potential arrhythmogenic components.

Numerous studies have revealed an increase in the number of isolated, premature ventricular contractions and ventricular arrhythmias in patients with hypertension with LVH, regardless of the presence or absence of heart vascular lesions detected by coronary angiography [10]. These data have been repeatedly confirmed both in the reports of individual groups of researchers [2] and in the Fremingen study [11]. This is explained by the development of hypertrophy of the media of arterioles against the background of LVH and an increase in the ratio of wall thickness to vessel size. As a result, coronary microangiopathy occurs. Due to hypertrophy of the media, the ability to regulate vascular resistance decreases and leads to a decrease in the reserve of coronary blood flow. These changes in patients with LVH can be detected by using argon before and after the maximum dilation of the heart vessels induced by dipyridamole; the reserve of coronary blood flow in essential hypertension is reduced to 30% [15]. Patients with such a decrease in blood flow reserves complain of shortness of breath and suffer from angina pectoris. In studies with Holter electrocardiogram (ECG) monitoring, it has been proven that episodes of ischemia serve as triggers of arrhythmias [16].


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Similar disorders in patients with LVH are observed relatively early, at the asymptomatic stages of the disease [18]. Changes in microvessels and endothelial dysfunction also contribute to coronary insufficiency, pain-free ischemia and the formation of myocardial fibrosis [2]. The combination of hemodynamic overload and relative insufficiency of coronary blood flow in hypertension and LVH predisposes to the development of fatal rhythm disturbances and SCD [7].

The use of such classes of drugs as angiotensin converting enzyme inhibitors (ACE inhibitors), AT receptor blockers (ARBs) and aldosterone receptor antagonists may become a new direction in the development of antiarrhythmic therapy, as was proposed in the treatment of AF [7]. Assessing the risk of arrhythmias in asymptomatic patients is a difficult task that can be solved using the developed algorithm [19]. After a thorough survey and physical examination of patients, it is necessary to examine the systolic and diastolic functions of the heart, as well as to evaluate the MM index (IMM). The next important point is the exclusion of pain–free myocardial ischemia - the absence of stable or unstable ventricular tachycardia (VT) according to the Holter ECG monitoring data suggests that these patients are at very low risk. A much more detailed examination with the study of the arrhythmogenic substrate and the profile of the autonomic nervous system is necessary for patients with frequent FE, reduced ejection fraction (EF) and increased MMLF [19]. For this purpose, non-invasive methods can be used: analysis of micro-wave and HRV alternations, as well as intracardiac electrophysiological examination with fixed VT. Antiarrhythmic therapy, apparently, should be prescribed to individual patients who are already receiving adrenoblockers, ace inhibitors, spironolactone, and diuretics and adequately control blood pressure levels.

Thus, the mechanisms of arrhythmogenesis in LVH are extremely diverse. On the one hand, they include changes in the important electrophysiological properties of individual myocytes and extracellular matrix in various areas of the heart muscle, on the other, they are associated with external proarrhythmic factors that affect the hypertrophied myocardium. The presence of structural changes in the atria and ventricles at various degrees of hypertension may explain the high frequency of AF and ventricular arrhythmias in these patients. Improving blood pressure control, as well as the correct assessment of arrhythmogenic risk in patients with hypertension and LVH, will slow down those structural changes that create an ideal substrate for electrical instability of the myocardium.

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