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Influence of Patern and Degree of Left Ventricular Hypertrophy on Cardiac Arrhythmias

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1. Introduction

1.1 Ventricular arrhythmias

A large number of clinical and epidemiological studies [1,2] have reported a correlation between the mass increase of the left ventricle (LV) and the risk of disease or death. The prevalence of left ventricular hypertrophy (LVH) in patients with essential hypertension may be as high as 40% (12%-70%) [3]. It has been suggested [4] that in patients with LVH sudden death may be associated with an increased number of ventricular extrasystoles (VPB), which is frequently observed in this population. In effect, a review by Lombardi et al. [5] indicated that nonsustained ventricular arrhythmias are an independent predictor of cardiac death in hypertensive patients.

Most frequently it concerns concentric hypertrophy with no enlargement of ventricular cavities, interpreted by multiplication of sarcomeras in a parallel arrangement. This type of hypertrophy is characterised by increased mass and increased relative wall thickness. In a smaller number of hypertensives LVH is initially eccentric, with a multiplication of sarcomeras in sequence in the process of which enlargement of the ventricular cavity is prevalent and thickening of the wall is only proportional or less marked. Although many hypertensive patients develop isolated septum hypertrophy, data on the structure and function of the myocardium and arrhythmias in hypertensive patients with this type of LVH are limited [6]. It would appear that concentric hypertrophy carries the greatest and eccentric hypertrophy a moderate risk of cardiovascular (CV) events [7]. Published data regarding arrhythmias are still conflicting and unconvincing [6,8]. It is unclear whether the greatest risk of CV events in the concentric type is due to arrhythmias or something else (ishaemia for inst.). According to some authors [9] the correlation between left ventricular mass (LVM) and ventricular arrhythmias is graded and permanent. Arrhythmias described in hypertensive patients with LVH are usually single premature ventricular contractions, frequently bigeminal or multiform, and more rarely ventricular tachycardia. [10].

1.2 Supraventricular arrhythmias

Risk of AF (and other supraventricular arrhythmias) is also increased in patients with left ventricular hypertrophy (LVH) [11]. Concentric hypertrophy appears to hold the highest

risk [12]. Earlier investigations reported that LVH leads to diastolic dysfunction, decreased coronary blood flow reserve and the occurrence of ventricular and atrial arrhythmias. It appears that in the development of atrial arrhythmias, atrial volume overloading and the distension and dilatation of the myofibrils have a greater impact. This results from ventricular diastolic dysfunction (particularly of the left ventricle), due to hypertrophy and subsequent decreased compliance [13]. From the pathophysiological point of view, this is caused by the hypertrophy of cardiac myocytes, interstitial fibrosis and media hypertrophy of the arterioles. Microangiopathy can be diagnosed as the earliest sign of hypertensive heart disease, with diastolic dysfunction also being found as an early change. [14]. More recently, an increasing number of investigations conducted on supraventricular premature beats (SVPB) have documented enlarged atria in hypertrophic hypertensive hearts.

1.3 QT interval and QT dispersion

Marked left ventricular hypertrophy (LVH) is associated with potentially arrhythmogenic ventricular repolarization abnormalities and may generate conditions for QT interval (QTi) prolongation and increase QT dispersion (QTd)) [15,16]. Prolongation of QTc interval and QTd are risk markers for malignant ventricular arrhythmias (VA) and sudden cardiac death [17,18]. QT prolongation and dispersion are indicators for abnormalities in ventricular repolarization. This could suggest the presence of functional reentrant proarrhythmic circuits [19]. Defined as the difference between the longest and shortest QTi measured in any lead of the 12-lead electrocardiogram (ECG), QTd reflects the inhomogeneity in ventricular repolarisation. Both parameters include also depolarisation. Increased QTd has been shown to correlate positively to complex VA in many clinical conditions [17,20]. QTd and QTi correlate with the left ventricular mass index (LVMI) determined echocardiographically in a group of selected patients with essential hypertension [19,21,22]. Normal QTd values vary extensively from 10 to 71 ms. QTd is higher in cardiac patients in comparison to normal subjects. The probability is that only explicitly abnormal values (i.e., those >100 ms) outside error margins may potentially have a practical value, suggesting a markedly abnormal repolarisation [23]. Scarce data was published regarding QTc interval prolongation/QTd and complex ventricular arrhythmias in hypertensive patients with LVH [24,25], but which type of LVH has the greatest influence has been understudied (especially for the asymmetric type).

1.4 Discussion

Examined patients included in such studies should have essential hypertension and LVH confirmed by echocardiography. For that reason one must exclude congestive heart failure, known coronary disease (angina pectoris, previous myocardial infarction, percutaneous coronary interventions), heart surgery, valvular diseases, other cardiac diseases (previous myocarditis and hypertrophic obstructive cardiomyopathy in the absance of systemic hypertension), diabetes mellitus, alcoholics, mental disorders, overuse of non-antihypertensive drugs (psychiatric drugs: sedatives, psychopharmacs etc; antiparkinsonics, antirheumatics, analgesics and hormones), malignant or accelerated hypertension, stroke in the previous six months, patients with cancer, abnormal electrolytes, anemia, cardiopulmonary diseases, serum creatinine >140 μmol/L and abnormal thyroid function.

Study subjects usualy have long-term hypertension (average duration of 17 years in our sample) and excessive body weight. The majority subjects are physically inactive with elevated values of lipids and urea in serum [26].

Echocardiographic measurements confirme anthropological differences between genders [27]. Men have larger cardiac cavities and LVM. By indexing left ventricular mass according to the body surface (LVMI) this difference between genders is lost. Ejection fraction (EF) is most often very good (>60%). The values of mean LVMI, and in both genders are much higher than normal values (>170 g/m²) Concentric LVH is the most frequent (63% in [26]), which is not in agreement with some earlier investigations [28]. Eccentric LVH, usually of a mild degree, is more frequently observed in male patients. This could be explained by the larger diameter of the cardiac cavity in men. Mild LVH, according to some authors [29], does not carry increased risk either of complex neither of simple ventricular arrhythmias.

Complex *ventricular arrhythmias* on ECG are usualy found in small number (4%) of patients. During Holter monitoring this percentage increased to over 40% of patients, and during the stress test it increased by additional 7,4% (in proportion to heart rate and blood pressure). Ventricular tachycardia can be found in 7-18% [26]. Some authors [24] found that concentric hypertrophy carries the greatest risk, and the eccentric a moderate risk of death and of CV complications. Nunez et al. [6] found equal prevalence and complexity of ventricular arrhythmias in hypertensive patients with concentric and asymmetric LVH. Some earlier investigations [8] reported an equal incidence of ventricular arrhythmias with regard to the morphological type of LVH and LVMI, similar to our results. Devereux et al. [30] also obtained a negative correlation. Only a small number of studies in the literature (not written in the English language) have monitored this correlation and obtained a statistical significance.

Atrial fibrillation associated with atrial dilatation is often observed in patients with hypertension [31]. About 40% of patients have paroxysmal atrial fibrillation or paroxysmal supraventricular tachycardia during Holter monitoring and additional 4% during the exercise test [32]. Until now it is unable to conclude whether any of the LVH types had a greater effect on the appearance of these arrhythmias.

The left atrial size significantly positively correlates with left ventricular hypertrophy degree, and it seems that a larger number of *supraventricular premature beats* for moderate and severe concentric LVH compared to mild concentric LVH, can bee found [32]. Schannwell et al. [13] also found greater prevalence of supraventricular arrhythmias proportional to the degree of LVH.

The largest left atrial diameters can bee found in patients with eccentric LVH and the higher prevalence of supraventricular premature beats in the concentric and eccentric type [32]. Some outhors [6,33] found significantly greater prevalence and complexity of supraventricular arrhythmias in patients with asymmetric LVH. The relationship between arrhythmias and concentric geometry appears more coherent if we consider that a recent study [34] reported an independent association of impaired left ventricular relaxation with concentric LV geometry. The abnormal diastolic function of LV and the stretch in LA may be less expressed in the asymmetric tipe of hypertrophy. In the context of epidemiology, this means that in patients with asymmetric left ventricular hypertrophy (about 9% of subjects) a lower incidence of arrhythmias should be expected. Concentric LVH is most frequently found in hypertensive patients, and it appears to cause the most severe diastolic left

ventricular disfunction. Patients with eccentric LVH possibly have the largest LA dimension, and can have a slightly lower incidence of supraventricular premature beats then those with concentric LVH, meaning that the LA size is not the only factor that affecs the prevalence of supraventricular arrhythmias. Apart from mechanical remodeling, electrical remodeling that occures earlier should also be considered (change in the structure/function of ion channels and compounds, catecholamines, free oxygen radicals, angiotensin-converting enzyme, angiotensin II, cytokins and nitrogenous oxid), hypertrophy of media arteriola, dicrease in the coronary blood flow reserve and genetic factors (repeted expression of fetal genome isoforms, heme oxygenase-1[35]). These factors may differ in individual LVH forms.

Measurement of left atrial size from the parasternal long axis view using the 2-dimensional "M-mode" method is limited in accuracy for LA size quantification because of the irregular geometry of the LA and the angulations of the ultrasound beam [36]. Methods for measuring the LA volume are more appropriate for the assessment of the asymmetric remodelling of the LA chamber [37].

In patients with severe concentric and eccentric LVH higher values of the *QTc interval* can be find. Regarding the degree of LVH, a positive correlation with QTc length can be observed [38]. Not many articles investigating this correlation have been published in the literature [39,40]. In some [41] only 38 patients with essential hypertension and LVH were analyzed, and an attempt to classify them into groups with regard to LVH type was made. Their conclusion was that the QTc interval length correlates positively with LVMI and LVIDd. The longest QTc intervals were found in patients with LVH and complex arrhythmias. They also found that the incidence of complex ventricular arrhythmias was greater in patients with LVH, and that the prolonged QTc interval in these patients may be a good indicator for higher risk of arrhythmias. The mentioned authors also obtained the highest QTc interval values in patients with dilated (eccentric) LVH but the LVH type was not defined by calculating RWT, than by a simple addition of IVS and LVPW thickness. Patients with myocardial dilatation (with and without LVH) had the most severe arrhythmias. Measuring QTi only from the second lead represented a limitation to the study recognized by the authors. Other researchers also obtained the correlation between QTi and LVMI lengths [39,41]. QTc interval in patients with ventricular septal hypertrophy was significantly longer than in the normal group [42].

Higher values of the *QTdispersion* in severe concentric and eccentric LVH can also be find [38]. Manual assessment of the T-wave end is extremely unreliable. Regrettably, the existing automated methods have not proven to be advantageous. The main source of mistakes for readers and computers are the low amplitudes of T waves [43] and the border between the T and U wave or the P wave [44]. Increased QTc dispersion was associated with LVH, especially with its concentric variant in some studies [20,45]. In the LIFE study both concentric and eccentric LVH were associated with prolonged QTi and increased QTd [15]. In another study [46] QTd >60 ms and QTi >440 ms were associated with greater probability of LVH.

In our patients, the QTi length correlates with the VA incidence. QTc interval was also longer in patients having complex arrhythmias but the difference in relation to simple arrhythmias was not significant [38].

Several factors may influence the increased LV ectopic activity in patients with LVH. Increased stimulation of hypertrophic myocytes, fibrosis in hypertrophic myocardium that

leads to electrophysiological inhomogeneity, distention of certain myocytes, increased oxygen requirement of the myocardium, damaged membrane porosity for various ions, and increased sympatic activity are possible pathophysiological factors for the increased incidence of ventricular arrhythmias [47,48].

In subjects without heart disease, during a 15-20 years follow-up period, prospective studies found significant correlation between prolonged QTc interval and increased risk for coronary events [49], cardiovascular (CV) mortality and all-cause mortality (mean follow-up of 4.9 years) [50], as well as for sudden cardiac death or CV death [51]. The Zutphen study [51] concluded that men with QTc interval >420 ms were at greater risk for CV death than men with shorter QTc interval. Increased QTd in patients with LVH and higher incidence of ventricular premature beats (VPB) or complex VA was also described by other authors [22,24,52]. Ichkhan et al. [22] found a significant correlation between QTd and LVM in hypertensive patients. They concluded that LVH and not hypertension *per se* leads to increased QTd, because hypertensive patients without LVH did not have an increased QTd. Ozdemir et al. [53] studing 80 patients with concentric LVH have found strong correlation between increased QTd and the incidence of VA. Other authors [33] obtained the highest influence of asymmetric LVH on QTd, but without correlation between VPB and QTd, neither between VPB and LVMI.

According to some investigations patients with increased QTd should be treated with angiotensin receptor blockers and nebivolol [54,55].

It is very important to *exclude the possible effect of* the extent of *antihypertensive therapy* and the types of antihypertensive drugs on the result. Discontinuation of all drugs seven days prior to ergometric examination and Holter monitoring is optimal. It is sometimes not possible to keep patients 7 days without antihypertensive and anti-arrhythmic drugs because a longer suspension of treatment could have threatened the patient or lead to reduced cooperation.. Shorter discontinuation can bee applied but the duration and the type of drugs should not differ between the examined groups.

2. Conclusion

In conclusion, The degree of LVH contributes more to the greater prevalence of VA then the LVH pattern. Given that this correlation (between the degree and VA) is most expressed in the concentric type (which is at the same time the most frequent pattern of LVH in hypertensive patients), the combination of severe degree and concentric type carries the greatest risk. Asymmetric LVH does not necessarily represent an increased risk. Concentric and eccentric types have a greater impact on the frequency of atrial arrhythmias. The prevalence of supraventricular premature beats correlates with the degree of left ventricular hypertrophy in the concentric type. QTc interval and QT dispersion tend to increase proportionally to the left ventricular mass probably only in the concentric and eccentric LVH type.

In clinical practice this means that patients with moderate and severe LVH (concentric in particular) should be tested by Holter monitoring and bicycle ergometry and treated with maximally tolerable doses of antihypertensives, particularly with angiotensin converting enzyme inhibitors/ angiotensin receptor blockers and also with outpatient training program.

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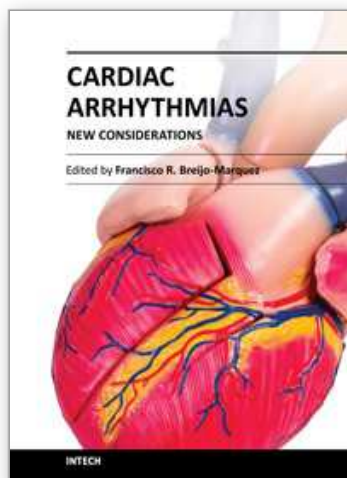
Cardiac Arrhythmias - New Considerations

Edited by Prof. Francisco R. Breijo-Marquez

The most intimate mechanisms of cardiac arrhythmias are still quite unknown to scientists. Genetic studies on ionic alterations, the electrocardiographic features of cardiac rhythm and an arsenal of diagnostic tests have done more in the last five years than in all the history of cardiology. Similarly, therapy to prevent or cure such diseases is growing rapidly day by day. In this book the reader will be able to see with brighter light some of these intimate mechanisms of production, as well as cutting-edge therapies to date. Genetic studies, electrophysiological and electrocardiographyc features, ion channel alterations, heart diseases still unknown , and even the relationship between the psychic sphere and the heart have been exposed in this book. It deserves to be read!

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