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ORIGINALARBEIT

Influence of the Type and Degree of Left Ventricular Hypertrophy on the Prevalence of Ventricular Arrhythmias in Patients with Hypertensive Heart Disease

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ABSTRACT

Purpose: To investigate the correlation between the prevalence of ventricular arrhythmias (VA) and the type and degree of left ventricular hypertrophy (LVH) in hypertensive patients using exercise testing and Holter monitoring.

Patients and Methods: A total of 192 patients (87 men and 105 women) without coronary disease were divided into three groups according to type of LVH (concentric, eccentric, and asymmetric) and three subgroups in relation to the degree of hypertrophy (mild, moderate, and severe). In all subjects blood pressure was measured, electrocardiographic and echocardiographic data obtained and the prevalence of VA determined by Holter monitoring and bicycle ergometry.

Results: The most frequent LVH type was the concentric (63%), followed by eccentric (28%) and asymmetric (9%). Severe LVH was found in 10% of patients. Complex VA during Holter monitoring were identified in > 40% of patients. During the stress test this percentage increased by additional 7.4%. There was no statistically significant difference between groups in frequency of simple ($p = 0.757$) and complex ($p = 0.657$, $p = 0.819$, $p = 0.617$, for polytopic, pairs and ventricular tachycardia, respectively) VA. Increased prevalence of VA was found for the moderate and severe degree in all types. In the concentric type the difference was statistically significant for simple VA ($p = 0.042$).

Conclusion: There was no correlation between type of LVH and prevalence of VA. The severity of hypertrophy contributes more to a greater prevalence of VA than the LVH pattern. The combination of severe degree and concentric type carries the greatest cardiovascular risk.

Key Words: Hypertension · Left ventricular hypertrophy · Asymmetric · Concentric · Eccentric · Ventricular arrhythmias

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A large number of clinical and epidemiologic 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 & Terranova [5] indicated that nonsustained ventricular arrhythmias (VA) 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 sarcomeres in a parallel arrangement. This type of hypertrophy is characterized by increased mass and increased relative wall thickness. In a smaller number of hypertensives LVH is initially eccentric, with a multiplication of sarcomeres 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 (ischemia for instance). According to some authors [9] the correlation between left ventricular mass (LVM) and VA 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 (VT) [10].

The aim of this investigation was to examine the correlation between the prevalence of VA and the type and degree of LVH at daily activities and during exercise testing in hypertensive patients with LVH.

Patients and Methods

In a period of 5.5 years, 7,647 hypertensive patients were examined at the outpatient cardiologic department. Suspect LVH on electrocardiography (ECG) was observed in 1,606 patients of whom 1,414 were immediately excluded from the study for not satisfying the inclusion criteria. The diagnosis of LVH in the remaining 192 patients (87 men and 105 women, aged from 43 to 80 years) was confirmed by ECG and they were included in the study. Inclusion criteria were: patients who had only essential hypertension and LVH confirmed by ECG. Hypertensive patients were considered those with blood pressure ≥ 140/90 mmHg, measured three or more times by mercury sphygmomanometer, according to the guidelines of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC) [11]. Blood pressure and cardiac frequency were measured in office and the mean arterial pressure and pulse pressure were calculated. Upon previous patient consent and the approval of the School of Medicine Ethical Committee, medications were discontinued in all subjects for 48 h prior to ergometric examination and Holter monitoring. Alcohol intake was prohibited through the same period. Patients were advised to contact the principal investigator by phone or in person in the event any symptoms should appear. Exclusion criteria were: 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 [HOCM] in the absence of systemic hypertension), diabetes mellitus, alcoholics (exclusion was based on the medical history, clinical status and laboratory findings), mental disorders, overuse of non-antihypertensive drugs (psychiatric drugs: sedatives, psychopharmaceuticals etc.; antiparkinsonians, antirheumatics, analgesics, and hormones), malignant or accelerated hypertension, stroke in the previous 6 months. Patients with cancer, abnormal electrolytes, anemia, cardiopulmonary diseases, serum creatinine > 140 µmol/l and abnormal thyroid function were also excluded.

The examined variables were age, gender, systolic and diastolic pressure, mean pressure, pulse pressure, body mass index (BMI), body surface area (BSA), LVM, left ventricular mass index (LVMI), LV geometry, LVH degree, and the prevalence of VA. VPB (single, polytopic, pairs were counted) and VT (three or more consecutive ventricular premature beats) were examined. The type and duration of antihypertensive and antiarrhythmic

ZUSAMMENFASSUNG

Einfluss von Typ und Grad der linksventrikulären Hypertrophie auf die Prävalenz von ventrikulären Arrhythmien bei Patienten mit hypertensiven Herzerkrankungen

☐ **Ziel:** Die Wechselbeziehung zwischen der Prävalenz von ventrikulären Arrhythmien (VA), dem Typ und dem Grad der linksventrikulären Hypertrophie (LVH) bei Patienten mit Bluthochdruck sollte mittels Ergometrie und Langzeit-EKG untersucht werden.

☐ **Patienten und Methodik:** Insgesamt 192 Patienten (87 Männer und 105 Frauen) ohne Herzerkrankung wurden in drei Gruppen in Bezug auf den LVH-Typ (konzentrisch, exzentrisch und asymmetrisch) und in drei weitere Untergruppen gemäß dem Grad der Hypertrophie (leicht, mäßig und schwer) eingeteilt. Bei allen Patienten wurde der Blutdruck gemessen, elektrokardiographische und echokardiographische Daten wurden ermittelt, und die Prävalenz von VA wurde mittels Langzeit-EKG und Ergometrie festgestellt.

☐ **Ergebnisse:** Der am weitesten verbreitete LVH-Typ war der konzentrische (63%), gefolgt vom exzentrischen (28%) und asymmetrischen (9%). Eine schwere LVH wurde bei 10% der Patienten gefunden. Komplexe VA wurden mittels Langzeit-EKG bei > 40% der Patienten identifiziert. Mittels Ergometrie wurde dieser Prozentsatz um weitere 7,4% erhöht. Es gab keinen statistisch signifikanten Unterschied zwischen den Gruppen in Bezug auf einfache ($p = 0{,}757$) und komplexe VA ($p = 0{,}657$, $p = 0{,}819$, $p = 0{,}617$, für polytope, für Paare oder für ventrikuläre Tachykardie). Eine erhöhte Prävalenz von VA wurde für den mäßigen und schweren Grad bei allen Typen gefunden. Beim konzentrischen Typ war der Unterschied für die einfache VA statistisch signifikant ($p = 0{,}042$).

☐ **Schlussfolgerung:** Es gab keine Wechselbeziehung zwischen dem LVH-Typ und der Prävalenz von VA. Die Schwere der Hypertrophie ist bedeutender für die Prävalenz von VA als das LVH-Muster. Die Kombination aus schwerem Grad und konzentrischem Typ bringt das größte Risiko für Herz und Kreislauf mit sich.

Schlüsselwörter: Bluthochdruck · Linksventrikuläre Hypertrophie · Asymmetrisch · Konzentrisch · Exzentrisch · Ventrikuläre Arrhythmien

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therapy received by the patients before entering the study were recorded for the purpose of analyzing the possible effects on the outcome.

Blood tests were performed for serum lipids, glucose, urea, creatinine, potassium and natrium, before consumption of food. Red blood count was determined (erythrocytes, hemoglobin, hematocrits, mean red cell volume [MCV], mean corpuscular hemoglobin [MCH], mean corpuscular hemoglobin concentration [MCHC], and iron), in order to exclude anemia. Pulse pressure was calculated as the difference between systolic and diastolic blood pressure. Mean blood pressure was calculated as diastolic pressure + pulse pressure/3. For each patient BSA was calculated according to Du Bois' formula [12] and BMI (body weight/height × height = kg/m^2) was obtained from body weight and height of the patient.

Patients were divided into three main groups with regard to LVH type: concentric (relative wall thickness [RWT] ≥ 0.45 and interventricular septum/left ventricular posterior wall [IVS/LVPW] < 1.3), eccentric (diameter LV in systoles > 32 and RWT < 0.45), and asymmetric (IVS/LVPW > 1.3), as in one previous study [13] and our earlier study [14]. RWT was measured at end-diastole as the ratio of twice the thickness of LVPW/LVIDd (left ventricular internal diastolic diameter) [15]. Each group was divided into subgroups according to the degree of LVH: mild (IVS or LVPW 11–12 mm), moderate (IVS or LVPW 13–14 mm) and severe (IVS or LVPW ≥ 15 mm).

In all patients a twelve-lead ECG was performed (to select the studied patient sample with LVH), a stress test on a bicycle ergometer (Bruce protocol; patients were carefully loaded and blood pressure was frequently measured until systolic reached 220 mmHg or diastolic 120 mmHg), a 24-h ECG recording (Holter monitoring) and "M-mode", two-dimensional and Doppler echocardiography. ECG criteria to determine LVH according to Sokolow-Lyon and LV strain criterion were applied [16]. ECG interpretation was performed manually and by a qualified operator.

Echocardiographic measurements were performed and interpreted by three cardiologists working independently, with no knowledge of the study hypothesis. Throughout the study, two-dimensional "M-mode" imaging was performed on a 15 cm wide photosensitive paper, with a velocity of 50 mm/s, on Toshiba Corevision Pro QA apparatus, with a 2,5-MHz 16-mm probe (dimensions were measured from parasternal long axis view), compliant with the recommendations of the American Society of Echocardiography [17]. LVM was divided by the body surface in order to calculate LVMI. LVMI was calculated with the Devereux & Reichek formulae [18]: LVMI = (1.04 × [(IVS + LVPW + $LVIDd)^3$–$LVIDd^3$]–13.6)/BSA). LVH was defined as LVMI > 134 g/m^2 for men and > 110 g/m^2 for women [19].

Subgroups with arrhythmias consisted of patients with simple arrhythmias (grade I–II according to Lown) and complex (grades III–V according to Lown) VA registered during stress test and 24-h Holter recording. Possible correlation was investigated between the prevalence of VA and type, or degree of LVH.

Statistical Analysis

The distribution of numerical variables is described by mean value and standard deviation and median in distributions which are not normal. Ordinals are written as categorical variables, where gradation exists between some values of categories (e.g., grade LVH, grades according to Lown), and nominal (no gradation, e.g., type LVH). Parametric test of analysis of variance (ANOVA), was used to compare the values of the numerical variables according to the determined categories. In cases where the variables were not normally distributed, Kruskal-Wallis median test was used. For comparison of one numerical variable with two categorical variables, factorial analysis of variance (factorial ANOVA) was used. Linear regression was used for determination of possible correlation between numerical variables and coefficient correlation (r) was determined. The frequencies were analyzed by Pearson's χ^2-test in two-dimensional or by log-linear analysis in three-dimensional tables. Proportions (percentages) were compared using test for the difference of proportions. The

Table 1. Clinical patient characteristics, pressures. BMI: body mass index; SD: standard deviation.

Number, total 192	**Male 87 (45.3%)** **Female 105 (54.7%)**
Parameter	**Mean value ± SD**
Age (years)	69 ± 8
BMI (kg/m^2)	28.2 ± 4.0
Elevated cholesterol (%)	83.3
Elevated triglycerides (%)	74.5
Elevated urea (%)	70.8
Elevated creatinine [< 140 µmol/l (%)]	5.2
Smokers (%)	18.8
Physically inactive (%)	84.4
Duration of hypertension (years)	17 ± 7
Systolic blood pressure (mmHg)	182 ± 22
Diastolic blood pressure (mmHg)	105 ±11
Mean arterial pressure (mmHg)	133 ± 12
Pulse pressure (mmHg)	82 ±18
Frequency (beats/min)	79 ±11

level of statistical significance was set at 0.05 in all analyses.

Results

Subjects were older patients (mean age > 65 years), with excessive body weight and long-term hypertension (mean duration > 15 years). On average they had severe hypertension and high pulse pressure (Table 1). Analysis of variance showed no difference in age according to the type (ANOVA, F = 1.120, p = 0.328) and degree (ANOVA, F = 0.625, p = 0.567) of LVH. There was no difference for CV risk factors between groups either (for elevated cholesterol p = 0.360, for elevated triglycerides p = 0.990, for smoking p = 0.089, for physical activity p = 0.610, and for duration of hypertension p = 0.858).

No differences in BMI were determined either between the groups with regard to type (p = 0.289) or the degree of LVH (p = 0.416). Results were similar for BSA. Elevated cholesterol values and lack of physical activity were identified in a statistically significantly greater percentage of the remaining CV risk factors (p = 0.03).

Concentric LVH was most frequent, followed by eccentric and asymmetric. Severe LVH was found in 10% of patients (Table 2). Eccentric LVH was more frequent in men, while the concentric and asymmetric types were distributed equally between genders. The LVMI differed significantly with regard to LVH degree (it increased) presenting a statistically significant difference in all correlation comparisons (p = 0.001). LVMI also differed significantly with regard to LVH type. Patients with eccentric LVH had a statistically significantly higher LVMI (183.97 ± 33.32 g/m^2) than those with concentric LVH (169.94 ± 32.51 g/m^2; p = 0.011), while patients with asymmetric LVH (LVMI 179.43 ± 35.02 g/m^2) presented no significant difference in relation to the concentric and eccentric type.

There was no difference in duration of treatment between groups (p = 0.858) or in the type of applied medication. Antihypertensives were evenly distributed within groups with particular types of LVH (Pearson's χ^2-test). Prior to the study the patients were using two or more drugs (Table 3). There was no difference in the frequencies of applied antiarrhythmics between groups or subgroups (Fisher's test, p = 0.423 for propafenone [26 patients]; p = 0.150 for amiodarone [eight patients], p = 0.750 for verapamil [nine patients], and p = 0.571 for medigoxin [14 patients], only two patients were treated with mexiletine). There was no difference between type and degree of LVH in drug concentrations either (ANOVA, p = 0.536 and 0.146 for propafenone; p = 0.563 and 0.465 for amiodarone; p = 0.219 and

Table 2. Relation between the degree and type of left ventricular hypertrophy (LVH).

LVH degree	LVH type [patients (n)]				p-value
	Concentric	Eccentric	Asymmetric	Total	
Mild	37	30	0	67 (35%)	
Moderate	73	22	10	105 (55%)	**< 0.001**
Severe	11	2	7	20 (10%)	
Total	121 (63%)	54 (28%)	17 (9%)	192	

Table 3. Antihypertensive therapy (and antiarrhythmics) applied in the examined population. ACE: angiotensin-converting enzyme; ARBs: angiotensin receptor blockers; LVH: left ventricular hypertrophy.

Antihypertensives	LVH type [patients (n)]				p-value
	Concentric (n = 121)	Eccentric (n = 54)	Asymmetric (n = 17)	Total (n = 192)	
ACE inhibitors or ARBs	87 (72%)	35 (65%)	14 (82%)	136 (71%)	0.298
Calcium antagonists	97 (80%)	40 (74%)	11 (65%)	148 (77%)	0.409
β-blockers	61 (50%)	27 (50%)	9 (53%)	97 (51%)	0.952
Diuretics	57 (47%)	25 (46%)	7 (41%)	89 (46%)	0.794
Antiarrhythmics	27 (22%)	16 (30%)	2 (12%)	45 (23%)	0.287

Table 4. Number and prevalence of ventricular arrhythmias according to the type of left ventricular hypertrophy (LVH; obtained by Holter monitoring and stress test). SD: standard deviation; VPB: ventricular premature beats; VT: ventricular tachycardia.

	Ventricular arrhythmias	Total (n = 192)	LVH type [patients (n)]			p-value
			Concentric (n = 121)	Eccentric (n = 54)	Asymmetric (n = 17)	
Lown I–II	Single VPB [median (quartile range)]	45 (4–456)	45 (3–510)	47.5 (6–219)	40 (2–112)	0.577
	Log (VPB single ± SD)	3.93 ± 2.61	3.94 ± 2.70	4.04 ± 2.46	3.50 ± 2.61	0.757
Lown	Polytopic [n (%)]	42 (22)	24 (20)	14 (26)	4 (24)	0.657
III–V	Pairs [n (%)]	51 (27)	34 (28)	13 (24)	4 (24)	0.819
	VT [n (%)]	19 (10)	11 (9)	7 (13)	1 (6)	0.617

0.298 for verapamil; all patients were treated with the same dose of medigoxin [0.1 mg once a day]). Results were similar for β-blockers. There was no difference between the type and degree of LVH in the frequencies of applied β-blockers (Fisher's test for atenolol p = 0.083, for carvedilol p = 0.572, and for bisoprolol p = 0.400) and in drug concentrations (ANOVA according to the type of LVH for atenolol p = 0.355, for carvedilol p = 0.524, and for bisoprolol p = 0.423; ANOVA according to the degree of LVH for atenolol p = 0.060, for carvedilol 0.067, and for bisoprolol p = 0.342). Only one patient was treated with sotalol.

No statistically significant correlation was found in VA occurrence according to Lown's classification and type of LVH (Table 4).

Lown's patient classification and the degree of LVH revealed no statistically significant difference in the frequency of VA (Table 5).

There was no statistically significant difference between the number of single VPB and different degrees of LVH. Logarithm values were abnormally distributed (K-S test, p < 0.20). However, the central value of the recorded number of arrhythmias was greatest for severe LVH. Marginally statistically significant difference was observed in the prevalence of pairs of VPB, where the frequency was greater for moderate and severe LVH. VT was more frequent in moderate and severe LVH (not significant) and also more common in men than in women (18% and 7%, respectively; p = 0.024). Ventricular fibrillation (VF) was not registered. Increased prevalence (although statistically insignificant) of polytopic VPB with a degree of LVH was also identified.

The number and prevalence of VA for concentric LVH showed statistically significant differences with regard to the degree of LVH (Table 6). The number of single VPB for moderate and severe concentric LVH was significantly greater, and the prevalence of polytopic VES for severe LVH was marginally significantly greater than for mild and moderate concentric LVH.

Considering the degree of LVH, no significant difference was observed in the number and prevalence of VA in eccentric LVH. Slightly greater prevalence of pairs of VPB and VT were recorded for moderate and severe LVH (p = 0.109 and p = 0.107, respectively) in this type. No statistically significant difference was found in asymmetric LVH, although there was a greater prevalence of single VPB and VT for severe LVH compared to moderate LVH (p = 0.166 and p = 0.412, respectively).

The correlation between LVM and LVMI values and BMI was also analyzed. No significant correlation was obtained for LVM and BMI (r = 0.185; p = 0.016) or for LVMI and BMI (r = 0.035; p = 0.647). The correlation between BSA values and LVM presented significant correlation coefficient (r = 0.45; p < 0.0001), and no correlation between BSA and LVMI (r = 0.02; p = 0.814).

Analysis of the contingency tables shows that there was no correlation between LVMI and the incidence of VA. There was only a mild incidence increase up to a value of 180 g/m^2.

Table 5. Number and prevalence of ventricular arrhythmias according to the degree of left ventricular hypertrophy (LVH; obtained by Holter monitoring and stress test). SD: standard deviation; VPB: ventricular premature beats; VT: ventricular tachycardia.

	Ventricular arrhythmias	LVH degree [patients (n)]				p-value
		Total (n = 192)	Mild (n = 67)	Moderate (n = 105)	Severe (n = 20)	
Lown I–II	Single VPB [median (quartile range)]	45 (4–456)	45 (4–144)	45 (3–595)	102 (9–601)	0.448
	Log (VPB single ± SD)	3.93 ± 2.61	3.67 ± 2.52	4.05 ± 2.72	4.18 ± 2.32	0.561
Lown III–V	Polytopic [n (%)]	42 (22)	11 (**16**)	24 (**23**)	7 (**35**)	0.197
	Pairs [n (%)]	51 (27)	12 (18)	34 (**32**)	5 (**25**)	0.083
	VT [n (%)]	19 (10)	5 (7)	12 (11)	2 (10)	0.648

Table 6. Number and prevalence of ventricular arrhythmias according to the degree of concentric left ventricular hypertrophy (LVH; obtained by Holter monitoring and stress test). SD: standard deviation; VPB: ventricular premature beats; VT: ventricular tachycardia.

	Ventricular arrhythmias	Concentric LVH degree [patients (n)]			p-value
		Mild (n = 37)	Moderate (n = 73)	Severe (n = 11)	
Lown I–II	Single VPB [median (quartile range)]	9 (2–96)	58 (5–677)	102 (9–679)	**0.042**
	Log (VPB single ± SD)	3.08 ± 2.63	4.31 ± 2.72	4.44 ± 2.31	**0.042**
Lown III–V	Polytopic [n (%)]	5 (14)	14 (19)	5 (**45**)	0.064
	Pairs [n (%)]	7 (19)	24 (33)	3 (27)	0.305
	VT [n (%)]	3 (8)	7 (10)	1 (9)	0.968

DISCUSSION

The examined sample consisted of subjects with long-term hypertension, with an average duration of 17 years and excessive body weight. The majority of our subjects were physically inactive with elevated values of lipids and urea in serum [14].

Echocardiographic measurements confirmed anthropological differences between genders (not shown in tables) and corroborated earlier investigations [20]. Men had larger cardiac cavities and LVM. By indexing LVM according

to the body surface (LVMI), this difference between genders is lost. Ejection fraction (EF) was very good in the entire group (60.5 ± 7.4) and in both genders. Only three patients in the male group and three patients in the female group had an EF < 50%.The values of mean LVMI in the entire group of patients (173.98 g/m^2), and in both genders (178.12 and 170.84 g/m^2 in men and women, respectively) were much higher than normal values, which affects the relevance of further analyses. LVMI was greater by 6.62 g/m^2 in men than in women (statistically not significant). In a similar number of patients some authors [21] found a difference of 10.4 g/m^2. There were no patients with significant obstructions of left ventricular outflow tract (HOCM) in the examined group.

Concentric LVH is the most frequent (63% in our subjects), which is not in agreement with some earlier investigations [22]. Eccentric LVH, usually of a mild degree, was more frequently observed in our male patients. This could be explained by the larger diameter of the cardiac cavity in men. In 35% of patients mild LVH was found, which, according to some authors [23], does not carry an increased risk either of complex or of simple VA.

Medications were discontinued 48 h prior to ergometric examination and Holter monitoring with the intention to avoid the effects of treatment duration and type of antihypertensive and antiarrhythmic drugs (applied in patients with complex VA [Lown III–V]) on the outcome. We investigated whether these parameters differed between the examined groups. The obtained result was negative. A longer suspension of treatment could have threatened the patient or lead to reduced cooperation. Withdrawal of β-blockers could have affected the increase of cardiac rate and rhythm control; however, all three study groups were under identical conditions.

Complex VA on ECG were found in 4% of patients. During Holter monitoring this percentage increased to > 40% of patients, and during the stress test it increased by an additional 7.4% (in proportion to heart rate and blood pressure). VT was found in 18% of men and only in 7% of women (data not shown in tables). In the examined sample there was no statistically significant difference between groups in the frequency of simple and complex VA. Considering the degree of LVH, the trend of increased VA incidence was found for the moderate and severe degree in all types. In the concentric type the difference was statistically significant for simple VA. Patients are periodically under control at our polyclinic. The inclusion period was 5.5 years and the follow-up period 7 years until now. During the inclusion period no sudden cardiac deaths occurred. About 6 months later, after the inclusion ended, one elderly patient was hospitalized because of syncope and died soon after discharge (without implantable cardioverter defibrillator therapy).

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 VA in hypertensive patients with concentric and asymmetric LVH. Some earlier investigations [8] reported an equal incidence of VA with regard to the morphological type of LVH and LVMI, similar to our results.

No significant positive correlation was recorded between LVMI and the incidence of VA in our patients, although a mild value increase of LVMI up to 180 g/m^2 was observed, followed by a lower incidence. However, a higher incidence of arrhythmia appeared in patients with LVMI ≥ 200 g/m^2 than in those with LVMI of 180–200 g/m^2. We also observed a greater percentage of complex VA in more severe degrees of LVH associated with the thickness of the myocardium. Lastly, the incidence of VA tends to increase with the increase of LVMI, although the reasons are still unclear. Devereux & Reichek [18] 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.

CONCLUSION

There was no correlation between the type of LVH and the incidence of simple and complex VA even when exercise testing was performed; however, the severity of hypertrophy slightly contributed to a greater incidence of the same (significantly in the concentric type) suggesting that the degree of LVH contributes more to the greater prevalence of VA than 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. 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.

Study Limitations

It was impossible to completely rule out coronary artery disease. Coronary angiography has no logic in asymptomatic patients and radionuclide scintigraphy is too expensive and time-consuming for a more comprehensive trial. Modified methods from other similar studies were therefore applied. A 48-h antihypertensive drug withdrawal period is relatively short. We studied the duration of treatment and the type of antihypertensive and antiarrhythmic drugs and found no differences between the examined groups.

Our small groups of patients with asymmetric type and severe degree of LVH were analyzed with appropriate statistical methods mentioned in the related paragraph. An inadvertent inclusion of a patient with hypertrophic cardiomyopathy (HCM) and hypertension had probably no effect on the results considering the very small prevalence of this disease.

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