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Effects of Obesity and Sports on the Left Ventricle Mass in Preadolescent and Adolescent Children

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Abstract

Objectives: An increase in left ventricular mass (LVM), a determinant of left ventricular hypertrophy (LVH) in adults and children, is a major risk factor for cardiovascular morbidity and mortality. This study evaluated LVM in athletes, healthy and obese children.

Materials and Methods: The study included 260 children aged 9-17.8 years (170 males, 90 females). No participants had diseases such as aortic pathologies, hypertension, or hypertrophic cardiomyopathy. The participants were divided into three groups: Group 1, athletes (n=89); Group 2, control (n=87); Group 3, obesity (n=84). Obese athletes were excluded from the study. The participants were further divided into the preadolescent group (n=72, aged 9-11.91 years) and the adolescent group (n=188, aged 12-17.8 years). LVM, LVM index (LVMI) were calculated using the M-mode echocardiography.

Results: The mean LVMI for all participants was 32.72±8.48 g/m$^{2.7}$. The cut-off value of 42.76 g/m$^{2.7}$, which was the 95th percentile for the control group LVMI value, was taken as the LVH criterion. There was a significant difference between Group 1-3 and Group 2-3 in terms of LVMI.

Conclusion: Because of obesity and accompanying comorbid diseases in children have increased recently, being informed about the presence of LVH is critical for the diagnosis of cardiovascular diseases that may cause morbidity in childhood and young adulthood, and for early treatment planning.

Keywords: Left ventricular mass, children, obesity, sports

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Introduction

The hemodynamic load is assessed with left ventricular mass (LVM)[1] in congenital and acquired heart diseases such as coarctation the aorta, aortic valve stenosis and systemic hypertension when the left ventricle is subjected to pressure overload or when it is volume overload in aortic and mitral valve regurgitation.

Left ventricular hypertrophy (LVH), defined as an increase in the LVM, is associated with age, gender, and body size and composition. The LVM index (LVMI) is the ratio of LVM (g) to body surface area (BSA, m^2). The formula of dividing LVM by body height (m) to the power of 2.7 is generally used to calculate this index [LVMI: LVM (g)/(height) $m^{2.7}$]. In adults and children, LVH is defined as an LVMI equivalent to or greater than the 95th percentile[2]. Overweight and obesity are among the most important factors contributing to diseases and adverse health outcomes, such as metabolic syndrome, diabetes mellitus, and cardiovascular disease in the medium- or long-term course. The causes of obesity in adulthood can be traced back to early childhood, as a high body mass index (BMI) in childhood is a predictor of overweight and obesity later in life. In recent decades, a substantial increase in the prevalence of childhood obesity has been observed worldwide[3].

In the study by Twig et al.[4], obese adolescents were monitored for an average of 12 years, after that period the total cardiovascular death (coronary artery diseases+stroke+sudden death) ratio was reported as 0.2% and the mean death age was reported as 45.2±9.7 years. Total cardiovascular deaths were reported as 0.18% for a total 281.033 overweight and obese adolescents[4].

It has been demonstrated that LVM is strongly determined by lean body mass (LBM). It was considered "a reference standard" scaling variable. LBM explains more of the variability in LVM than either weight or height alone[5,6].

Regular sports activities in children cause physiological changes in the heart, similar to the athlete's heart in adults[7,8]. These changes primarily include increased left ventricular myocardial thickness and LVM[9,10]. In the studies conducted so far, the relationships between hypertension and LVH, obesity and LVM, sports and LVMI have been investigated[11-16]. However, in a prospective study, LVMI was examined in normotensive obese, normal and athletic children.

This study evaluated LVMI, identify in which group the LVMI increase is significant and to determine the percentile values of LVMI in all groups.

Materials and Methods

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Health Sciences Ethics Committee of (the approval number 20.478.486) Manisa Celal Bayar University, Faculty of Medicine, Manisa, Turkey. Written informed consent was obtained from the participants before the inclusion in the study. The voluntary participation form was signed by the parents.

Patients

The study included 260, patients aged 9-17.8 (mean 13.26) years, including 170 males and 90 females, who presented to the pediatric cardiology outpatient clinic of Manisa Merkezefendi State Hospital, with the complaints of cardiac murmurs identified from previous auscultation, chest pain, palpitation and between June and October 2018. The patients were divided into three groups: athlete, control, and obesity.

None of the patients had disease-causing cardiac muscle hypertrophy, such as aortic coarctation/stenosis, systemic hypertension, and hypertrophic cardiomyopathy, or a family history of this disease. Patients with endogenous obesity pathologies and different syndromes such as Type 1/Type 2 diabetes mellitus, metabolic syndrome, insulin resistance, dyslipidemia and hypertension, were excluded from the study. In this regard, measured mean blood pressure values were 106/66 mmHg, 114/70 mmHg and 113/67 mmHg for groups, respectively.

Eighty-nine participants in Group 1 (athlete group) made up 34.2% of the total patient population. Athlete children were involved in at least one type of sports: soccer, basketball, handball and swimming (high dynamic, moderate static component) in order of frequency, and practiced sports in a sports club and/or school team. About half of the them engaged in soccer, 43.8% basketball, 6.7% handball and swimming. Sports engagement was in the form of regular and disciplined training under the supervision of an instructor and participation in competitions/matches. The participants were training for at least one and at most eleven (mean 4.46±2.37) years. They participated in training for at least two hours and at most twenty-four (mean 8.08±5.07) hours a week. Obese and hypertensive athletes and those engaged in more than one sport were excluded from the study.

Eighty-seven children in Group 2 (control group) made up 33.5% of all patients. This group consisted of children who presented with various complaints (chest pain, fatigue, syncope, etc.) had a functional murmur, and normal cardiovascular findings on examination. They attended the gym and sports classes for only two hours a week at school.

Eighty-four children in Group 3 (obesity group) made up 32.3% of all patients. 29.7% were mild, 32.1% were moderate, 23.8% were severe and 14.4% were very severe obese. Most of the children in this group were only participating in gentle warm-up exercises in sports classes.

Echocardiography and Other Examinations

The routine transthoracic echocardiographic was examined with the segmental analysis method, using a Philips Medical System Nederland BV 2005 and S4-2 MHz Broadband Sector Array transducer. Heart rate was recorded during this examination. All these echocardiographic were examined by the same pediatric cardiologist (Dr. SP). LVM and LVMI were calculated using 2D, spectral Doppler, and M-mode echocardiograms. LVM was estimated using the formula (Method I) by Devereux et al.[17], (LVM=0.8 [1.04 x (interventricular septal thickness + left ventricular posterior wall thickness + end-diastolic diameter)^3 - (end-diastolic diameter)^3] +0.6). As mentioned above, LVMI=LVM (g)/(height)m$^{2.7}$.

I used the internet site https://www.calculator.net to calculate exponential numbers. Additionally, I calculated M-mode echocardiographic LVM [Method II: American Society of Echocardiography (ASE) convention], Z-score expected LVM (expLVM), estimated LBM (eLBM) and ASE guidelines by using the internet site http://lvmassparameterz.com[5,6,18]. I used the internet site https://www.cdc.gov/healthyweight/bmi/calculator.html to calculate BMI and degree of obesity. Patients with missing data were excluded from the study.

Paraclinical parameters, such as lipid panel (total cholesterol, triglyceride, low-density lipoprotein, and high-density lipoprotein cholesterol), complete blood count, ferritin, iron, C-reactive protein, thyroid function tests, fasting blood glucose, and 24-hour ambulatory blood pressure measurements were performed for all obese and patients deemed necessary. The Health Sciences Ethics Committee of Manisa Celal Bayar University approval and a waiver of consent were obtained before data collection.

Statistical Analysis

All data were evaluated using the SPSS version 15.0 software. Minimum, maximum, mean, and standard deviation (SD) values were obtained by descriptive statistical methods. Continuous variables were expressed as mean ± SD. Pearson correlation test was used to evaluate correlations between variables. Weak, moderate and strong correlations are defined as having a correlation coefficient less than 0.3 (<0.3), between 0.3-0.7 (0.3-0.7), and greater than 0.7 (>0.7), respectively. The independent sample t-test was used to compare the parametric values between the groups (female-male/preadolescent-adolescent/Group 1-2/Group 1-3/Group 2-3). Variance analysis was used to compare the values of more than two independent groups. In the independent sample one-way analysis of variance, the homogeneity of the groups was analyzed using the One-Way ANOVA test, and in the

presence of a difference, the Tukey test was carried out to determine among which groups there was a difference. Logistic regression analysis, Fisher's exact test, and the chi-square test were used to determine the risk ratio (odds ratio) and confidence interval (CI) for LVH. Bland-Altman analysis was used to display harmony between means and differences for the values of LVM Method I and II, weight, and eLBM, LVM Method I, and expLVM. The level of statistical significance was set at $p<0.05$.

Results

The demographic and echocardiographic data of all patients are shown in Table 1 and Table 2. The mean age of all children (n=260) n included in the study was 13.26±2.06 years. All participants included in the study had a mean LVM of 119.53±37.68 g and a mean LVMI of 32.72±8.48 g/m$^{2.7}$. The demographic characteristics and echocardiographic findings of the male and female participants are shown in Table 3. The pre-adolescent group (n=72) included participants aged 9-11.91 years, while the adolescent group (n=188) included participants aged 12-17.8 years. In all participants, female/male and adolescent/preadolescent groups, the percentile values for LVMI were determined and are presented in Table 4.

Table 1. The demographic and echocardiographic data of all patients

	Total	Female	Male
n	260	90	170
	Minimum	**Maximum**	**Mean ± SD**
Age (year)	9	17.8	13.26±2.06
Body weight (kg)	25.3	144	59.59±16.22
Height (cm)	130	192	161.7±13.49
BSA (m^2)	0.93	2.49	1.6±0.25
BMI	14.21	48.16	22.64±4.97
w/h	68	217	108.89±25.39
IVS (cm)	0.61	1.25	0.87±0.13
LVDd (cm)	3.02	5.71	4.43±0.44
LVSd (cm)	1	3.67	2.71±0.34
LVPWd (cm)	0.6	1.25	0.84±0.11
EF (%)	59.7	81.7	68.84±4.64
FS (%)	31.7	50.7	38.42±3.77
LVM (g)	42.6	243	119.53±37.68
LVMI (g/m$^{2.7}$)	32.74	122.22	73.79±17.79
Expected LVM	76.8	311.6	183.07±44.52
Estimated LBM	19.7	92.4	41.33±10.54
LBM Z-score	-4.56	1.93	-0.56±1.03

BSA: Body surface area, BMI: Body mass index, SD: Standard deviation, LVMI: Left ventricular mass index, LVM: Left ventricular mass, LBM: Lean body mass, LVDd: Left ventricular diastolic diameter, LVSd: Left ventricular systolic diameter, LVPWd: Left ventricular posterior wall diastolic

Correlations between the entire patient group and individual groups were analyzed. There were statistically significant results for all the correlations between LVMI and height, body weight, BMI, w/h of the entire group. The correlation between LVMI and w/h was strongest and moderately statistically significant (r=0.531, p=0.001).

The analysis of variance by groups showed a significant difference between these three groups in terms of different parameters. There was a significant difference in terms of LVMI between Group 1 and Group 3 (p=0.001). The same significance in terms of LVMI between Group 2 and Group 3 was found (p=0.001).

M-mode echocardiographic LV mass, Z scores, expLVM, eLBM were examined in all groups. A statistically significant and important correlation was found between LVMI calculated based on the Devereux formula and LVMI calculated on the basis of the guideline reported by Lopez et al.[18] (there was a significant correlation between LVM calculated with calculator and LVM calculated with Devereux formula). There was a significant correlation in terms of LVM between Methods I and II (r=0.84, P=0.001).

There was a statistically significant and strong correlation between eLBM and weight (r=0.906, p=0.001). Also, there was a statistically significant and moderate correlation between LVM (Method I) and expLVM (Method II) (r=0.644, P=0.001).

Totally, there were 39 patients overweight (14.99%). There were 11 patients overweight (4.23%) in Group 1 (athlete) and 28 patients overweight (10.76%) in Group 2 (control). Since LVMI (m)$^{2.7}$ is a restrictive factor in patients whose height is less than 140 cm[2,5]. "I would like to state that there are eight patients whose height is less than 140 cm". The cut-off values for LVMI and the

incidence of LVH in the groups (in accordance with the values we accepted in our study and from various studies) are displayed in Table 5.

The mean LVM for LBM z- score values, the mean eLBM values and p values of both, girls and boys are presented in Table 1-3. The mean LVM difference between Method I and II: -1.21±4.54 (CI 95%: -1.76/-0.65, p=0.001) is displayed in Figure 1. The mean LVM difference between Method I and expLVM: -64.53±34.51 (CI 95%: -68.79/-60.27, p=0.001) is displayed in Figure 2. The mean difference between mean weight and eLBM: 18.26±8.03 (CI 95%: 17.28-19.24, p=0.001) is displayed in Figure 3. In our study, the cut-off value of 42.76 g/m$^{2.7}$, which was the 95th percentile for the control group LVMI value, was taken as the LVH criterion.

Those with obesity and with overweight were found to have a 6.1-fold increased risk of having LVH compared with those without obesity (OR, CI 95%: 2,689-14,226) (Figure 4). Fisher's exact test p-value was 0.001. It was shown that those who did sports had a 0.348-fold increased

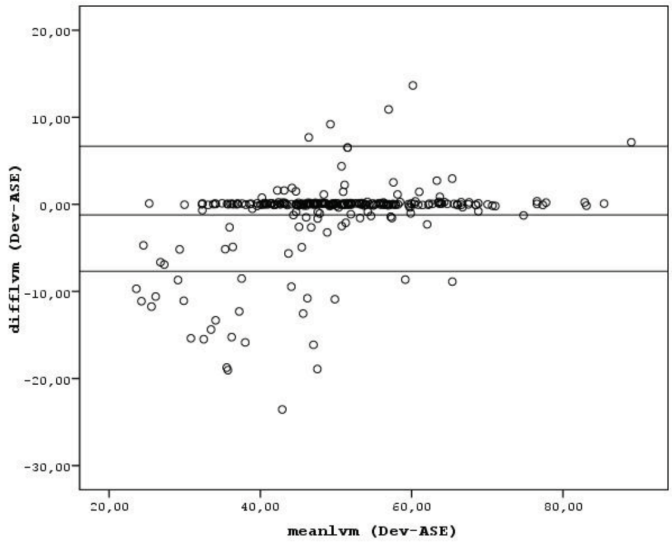

Figure 1. The mean LVM difference between Method I and expLVM

LVM: Left ventricular mass

Table 2. The demographic and echocardiographic data of all groups

	Group 1	Female	Male	Group 2	Female	Male	Group 3	Female	Male
n	89	21	68	87	33	54	84	36	48
	Minimum	Maximum	Mean ± SD	Minimum	Maximum	Mean ± SD	Minimum	Maximum	Mean ± SD
Age (year)	9.33	17.83	13.59±2.01	9	17.5	13.73±2.01	9	16.83	12.43±1.94
Body weight (kg)	25.3	92	52.9±14.27	27.5	90	55.89±12.52	40	144	70.49±16.14
Height (cm)	131	192	164.47±15.67	136	187	163.29±12.19	130	180	157.11±110.02
BSA (m^2)	0.93	2.06	1.49±0.25	0.99	2.03	1.55±0.21	1.28	2.49	1.77±0.2
BMI	14.3	27.54	19.19±2.59	14.21	27.77	20.76±2.99	21.97	48.17	28.25±3.53
w/h	68	114	90.43±11.51	71	118	98.22±13.31	121	217	139.48±15.98
IVS (cm)	0.63	1.25	0.89±0.14	0.61	1.17	0.83±0.12	0.67	1.25	0.9±0.13
LVDd (cm)	3.02	5.39	4.42±0.42	3.03	5.48	4.41±0.47	3.22	5.71	4.45±0.43
LVSd (cm)	1.8	3.56	2.72±0.31	1.94	3.6	2.71±0.34	1	3.67	2.7±0.37
LVPWd (cm)	0.6	1.13	0.84±0.11	0.61	1.09	0.81±0.1	0.67	1.05	0.87±0.11
EF (%)	59.7	80.7	68.48±4.58	60.5	81.3	68.71±4.64	62.2	81.7	69.36±4.69
FS (%)	31.7	49	38.08±3.57	32.5	49.3	38.39±3.89	32.8	50.7	38.81±3.84
LVM (g)	42.6	203	114±37.7	50.6	220	114.28±35.8	60	243	130.81±37.41
LVMI (g/m$^{2.7}$)	12.48	51.54	30.0±8.12	14.8	51.05	30.22±7.21	24.64	61.73	38.19±7.48
Expected LVM	97.3	264.3	178.24±46.92	97.3	296.7	182.08±43.11	76.8	311.6	189.35±43.11
Estimated LBM	19.7	67.5	40.05±11.11	20	63.1	40.47±9.41	24.9	92.4	43.56±10.78
LBM Z-score	-4.56	1.87	-0.27±1.05	-3.43	1.78	-0.83±1.05	-2.78	1.93	-0.57±0.9

BSA: Body surface area, BMI: Body mass index, SD: Standard deviation, LVMI: Left ventricular mass index, LVM: Left ventricular mass, LBM: Lean body mass, LVDd: Left ventricular diastolic diameter, LVSd: Left ventricular systolic diameter, LVPWd: Left ventricular posterior wall diastolic

risk of having LVH compared to those who did not (OR, CI 95%: 0.12-0.942). Fisher's exact test p-value was 0.04.

When we accepted the cut-off value for LVH as 51.0 g/m$^{2.7}$, it was calculated that those with obesity and with overweight had a 5.07-fold increased risk of having LVH compared to those normal.

Discussion

Recently, an increasing number of children have been exposed to cardiovascular diseases such as coronary artery disease, obesity, hypertension, hypercholesterolemia, diabetes those accompanying the obesity due to general, one-sided, fast-food style nutrition, immobile living type, socioeconomic status, increasing stress factors. The number of studies on cardiovascular morbidity and mortality is limited due to the low incidence of cardiovascular events in preadolescents and adolescents.

the study by Twig et al.[4], 86.061 adolescents with a BMI of ≥95th percentile were monitored for an average of 12 years period, after that period, the total cardiovascular death (coronary artery diseases+stroke+sudden death) ratio was reported as 0.2% and the mean death age was reported as 45.2±9.7 years. When a total of 2.298.130

Table 3. The demographic and echocardiographic characteristics of female and male participants

Sex	Female	Male	Total
n	90	170	260
	Mean ± SD	**Mean ± SD**	**p-value**
Age (year)	13.28±1.92	13.25±2.14	0.923
Body weight (kg)	58.92±14.73	59.95±16.99	0.627
Height (cm)	157.74±9.18	163.8±14.9	0.001
BSA (m^2)	1.60±0.23	1.61±0.26	0.796
BMI	23.59±5.02	22.14±4.88	0.026
Percentile	73.16±31.3	65.93±30.15	0.071
W/H	113.21±24.66	106.6±25.54	0.046
IVS (cm)	0.83±0.12	0.904±0.13	0.081
LVDd (cm)	4.29±0.46	4.5±0.41	0.262
LVSd (cm)	2.65±0.3	2.75±0.36	0.15
LVPWd (cm)	0.8±0.098	0.86±0.12	0.017
EF (%)	68.65±3.86	68.94±5.01	0.64
FS (%)	38.11±3.1	38.59±4.07	0.334
LVM (g)	108.38±34.29	125.42±38.16	0.001
LVMI (m)$^{2.7}$	31.58±8.44	33.33±8.47	0.114
Expected LVM	169.08±31.2	190.69±48.74	0.001
Estimated LBM	37.79±7.32	43.2±11.48	0.001
LBM Z-score	-0.84±1.1	-0.41±0.96	0.001

BSA: Body surface area, BMI: Body mass index, W/H: Weight/Height, SD: Standard deviation, IVSD: Interventricular septum diastolic, LVDd: Left ventricular diastolic diameter, LVSd: Left ventricular systolic diameter, LVPWd: Left ventricular posterior wall diastolic, EF: Ejection fraction, FS: Fractional shortening, LVM: Left ventricular mass, LVMI: Left ventricular mass index

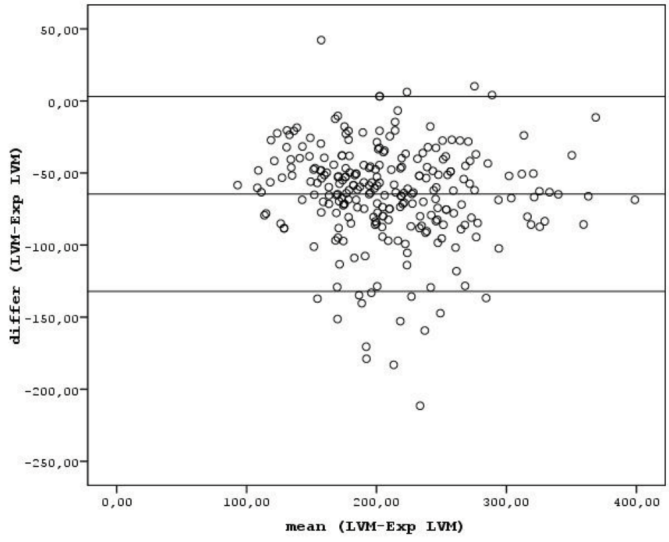

Figure 2. The mean LVM difference between Method I and expLVM

LVM: Left ventricular mass

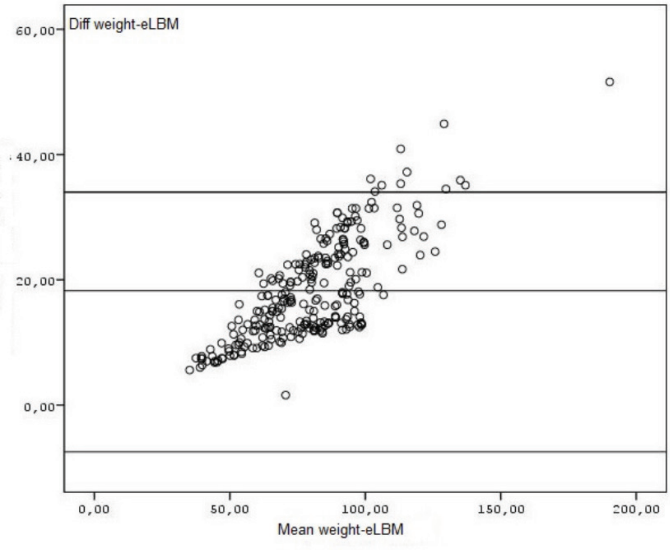

Figure 3. The mean difference between mean weight and eLBM

adolescents were monitored for an average of 18.4-year period, the mean death age was reported as 45.3±9.8 years, whereas the total cardiovascular death ratio was reported as 0.1%. Total cardiovascular deaths were reported as 0.18% for much overweight and obese adolescents.

Although LVMI (LVM/$m^{2.7}$) is also a popular method for normalizing LVM in children, it has an important limitation[5,19]. LVMI increases with decreasing height below about 130-140 cm[2,5]. In several studies, the cut-off value of >38.6 g/$m^{2.7}$ for LVMI, which is the conventionally reported value, is used for the definition of LVH[2,20,21]. In the few studies, the cut-off value of >51.0 g/$m^{2.7}$ for LVMI, which is the conventionally reported value in adults, is used for the definition of LVH[22,23]. In our study, the cut-off value of 42.76 g/$m^{2.7}$, which was the 95th percentile for the control group LVMI value, was taken as the LVH criterion. The 95th percentile cut-off value for LVMI was 46.9 g/$m^{2.7}$ in boys and 45.1 g/$m^{2.7}$ in girls, with the mean values of 33.33±8.44 g/$m^{2.7}$ in boys and 31.58±8.44 g/$m^{2.7}$ in girls. In the studies by Khoury et al.[2], LVH was defined by values above 40 g/$m^{2.7}$ and 45 g/$m^{2.7}$, for girls and boys, respectively[2].

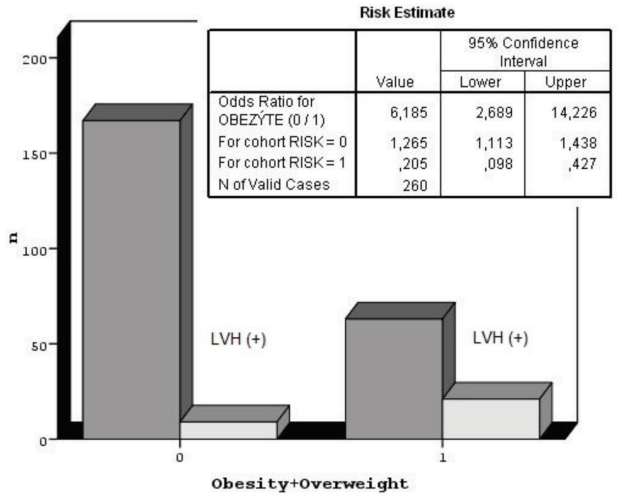

Figure 4. The risk estimation of LVH/Obesity+overweight
LVM: Left ventricular mass

In a study by Hietalampi et al.[24], the gender-specific 90th percentile for LVMI was 34.02 g/$m^{2.7}$ in girls and 37.08 g/$m^{2.7}$ in boys. This study also showed that there was a direct correlation between birth weight and LVM in adolescents, and the level of physical activity was associated with increased LVM and LWPWd. In our study, the gender-specific 90th percentile for LVMI is displayed

Table 4. LVMI percentile values for different groups

Percentiles	Girls (n=90)	Boys (n=170)	Adolescents (n=188)	Preadolescents (n=72)	Group 1 (athlete) (n=89)	Group 2 (control) (n=87)	Group 3 (obesity) (n=84)	Total
5 p	16.22	17.48	16.5	20.28	14.66	16.88	27.48	17.44
10 p	21.35	22.55	20.73	24.02	17.44	21.47	28.98	21.87
25 p	25.77	28.77	26.85	30.22	25.88	24.69	33.16	27.65
50 p	31.1	33.22	31.56	33.9	31.06	29.94	36.71	32.69
75 p	36.87	37.74	37.3	38.08	35.85	35.43	43.04	33.72
90 p	43.06	44.37	43.21	44.41	39.06	39.87	47.18	43.69
95 p	45.16	46.89	45.63	50.17	43.57	42.76	54.47	45.9

LVMI: Left ventricular mass index

Table 5. LVH and LVMI cut-off values

Groups	1 (athlete) (n=89)	2 (control) (n=87)	3 (obesity) (n=84)	Total (n=260)
LVMI cut-off values	LVH	LVH	LVH	LVH
42.6 g/$m^{2.7}$/ n-(%)	5 (5.6%)	3 (3.44%)	21 (25%)	29 (11.15%)
38.6 g/$m^{2.7}$/n-(%)	9 (10.1%)	10 (11.49%)	35 (41.66%)	54 (20.76%)
51.0 g/$m^{2.7}$/n-(%)	1 (1.12%)	1 (1.14%)	6 (7.14%)	8 (3.07%)

LVH: Left ventricular hypertrophy, LVMI: Left ventricular mass index

Table 4. Bartkevičienė[25] reported that LVH was present in 48% of all athletes, in our study incidence of LVH was 5,6%. In a study conducted by Krysztofiak et al.[15] LVM was 113.88 and 93.46 g, in our study 125.42 and 108.38 g, male, and female patients, respectively.

In our study, there was a significant difference between the two groups in terms of LVM and LVMI (p=0.001 and p=0.04, respectively). Furthermore, the 95th percentile cut-off value for LVMI was 50.1 g/m$^{2.7}$ in the preadolescent group and 45.63 g/m$^{2.7}$ in the adolescent group (Table 4).

In the study of Gupta-Malhotra et al.[20] in which they dealt with the pediatric hypertensive patients aged 9-18 years, the risk of LVH was found to be 8.9 times higher in obese compared with normal-weight children. Additionally, in a similar way, also in the study by Kharod et al.[23], it was reported that the risk of LVH in children without hypertension varies between 5.3 and 8.5 depending on whether they are overweight or obese. Cardiovascular adaptation mechanisms that develop in response to exercise are well defined[26]. In the athlete's heart, adaptation to increased hemodynamic load due to physical activity leads to physiological changes in cardiac morphology[27]. In their study, Hietalampi et al.[24], showed that excessive physical activity in healthy adolescents increased LVM, especially the LVPW thickness. This increase in LVM was shown to predict cardiovascular disease, morbidity, and mortality in adults, and subclinical changes caused by cardiovascular disease were shown to start in the childhood age group.

In a study by Castanheira et al.[28]. it was reported that the mean LVM and LVMI of 164 athletes who were involved in federated sports (basketball, swimming, judo, and hockey) for at least five years was 157.3 g and 87.9 g/m^2, respectively; the mean LVMI was 83.8±14.4 g/m^2 in the age group of 14-14.9 years and 91.5±13.2 g/m^2 in the age group of 16-16.9 years. In our study, the mean LVM and LVMI of the athletes were 114 g and 76 g/m^2, respectively.

In a study conducted by Sharma et al.[29], investigating the physiological limits of LVH in high-level athletes, it was reported that there were no female athletes with an LVPWd above 11 mm and only three of the male athletes (0.4% of the total) had an LVPWd above 12 mm. It was reported that LVPWd was increased in athletes compared with controls, it was rarely above 12 mm, and hypertrophic cardiomyopathy should be considered in the presence of LVPWd exceeding 12 mm in boys and 11 mm in girls, and non-dilated left ventricle. While all athletes participated in regional competitions for 4.3±1.5 (1-10) years, 50% of them participated in national competitions during the study period. The mean weekly intense training time was 9.8±3.6 (5-27) hours. In our study, the maximum LVPWd values of the participants in the athlete and control groups were 11.3 mm and 10.9 mm, respectively. In our study, there were no athletes participated in national competitions. The duration of sports engagement was 4.46±2.37 (1-11) years, and the mean weekly training time was 8.08±5.07 (2-24) hours.

Obesity-related LVH reflects the volume and pressure overload, causes different left ventricular geometry adaptations in both children and adults[30]. Childhood obesity is a precursor of increased LVM in adults and is an independent risk factor for subclinical left ventricular dysfunction[31]. In the study by Alkholy et al.[32] on the assessment of LVMI in obese children, the mean BMI was 32.8±4.6 kg/m^2 and the mean LVMI was 56.6±14.1 g/m$^{2.7}$ in the obesity group, while the mean BMI was 18.7±2.9 kg/m^2 and the mean LVMI was 42.7±12.6 g/m$^{2.7}$ in the control group. In our study, the mean BMI was 28.25±4.6 kg/m^2 and the mean LVMI was 38.19±7.48 g/m$^{2.7}$ in the obesity group, while the mean BMI was 20.76±2.99 kg/m^2 and the mean LVMI was 30.22±7.21 g/m$^{2.7}$ in the control group.

In our study, 260 echocardiographically calculated LVMI percentile values were determined for pre-adolescent and adolescent children, girls and boys, and those who were overweight and obese. The cut-off value of 42.76 g/m$^{2.7}$, which was the 95th percentile for the control group LVMI value, was taken as the LVH criterion. It was shown that there was a significant difference between

athlete/obesity and control/obesity groups in terms of LVMI. Those with obesity and with overweight were found to have a 6.1-fold increased risk of having LVH compared with those without obesity. Those who practice sports were found to have a 0.348-fold increased risk of having LVH compared with those who did not. Obesity was found to be a factor affecting LVMI and determining LVH.

Study Limitations

If our study could have had a greater patient population, we would have been able to adopt our findings to the other pediatric populations.

During the study period, most of the participants in the obesity group were followed up endocrinologically at different hospitals. Detailed obesity-related hormonal tests could be performed for all obese participants but not performed for others. The participants in the athlete group were also not the athletes from the same club or team. They were engaged in the same sportive categories (high dynamic, moderate static component) at least.

Conclusion

Recently, obesity and accompanying comorbid diseases in children have gradually increased. In most of these studies, attention is attracted to LVH and its risks. In this study, the LVH risks of children with obesity and overweight, without accompanying comorbid diseases (diabetes, hypertension, dyslipidemia) and who are interested in sports were compared with those of normal healthy children. LVH, the existence of which was investigated in our study, is a critical determinant in cardiovascular diseases, as in obesity, and in diseases that cause significant morbidities such as hypertension and cardiomyopathy. Therefore, it is critical to determine LVH using echocardiography, which can be easily applied in childhood.

Ethics

Ethics Committee Approval: The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Health Sciences Ethics Committee of (The approval number 20.478.486) Manisa Celal Bayar University, Faculty of Medicine, Manisa, Turkey.

Informed Consent: Written informed consent was obtained from participants before the inclusion in the study. The voluntary participation form sign by parents.

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