
ECHOCARDIOGRAPHY CHANGES IN SYSTOLIC AND DIASTOLIC FUNCTION IN OBESE AND OVERWEIGHT EGYPTIAN CHILDREN

By

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ABSTRACT

Background: Obesity is becoming a public and personal health crisis. The future of these populations' cardiovascular health is projected to be significantly affected by the rise in the occurrence of childhood & adolescent obesity.

Aim of the study: To assess using both conventional & tissue Doppler imaging to evaluate the anatomy & diastolic function of the left ventricle (LV) in children and adolescents from Egypt who are overweight or obese.

Methods: This prospective case-control study that was conducted on 90 children and adolescents (30 obese, 30 overweight, and 30 age- and gender-matched non-obesity controls) at Al-Hussein and Saied Galal Al-Azhar University Hospitals between March 2022 and March 2023.

Results: There's little variation among the studied groups in terms of LV morphology, but there is a significant difference in terms of MV E wave & A wave and E/A ratio.

Left ventricular structural and functional changes are predominantly expressed as a shift in diastolic function, even while global systolic function is conserved. It appears that the severity of obesity amplifies these changes.

Keywords: Doppler, echocardiography, pediatric obesity, ventricular dysfunction.

INTRODUCTION

One of the greatest global health concerns in recent decades has been the alarming increase in the prevalence of pediatric obesity, which is described as abnormal or excessive fat storage

that offers a health risk. There were an estimated 330 million overweight or obese children and adolescents worldwide in 2016. (Finkelstein EA et al., 2014).

The incidence of obesity was showed to be shockingly high by

(**Ng M et al., 2014**). Obesity in adolescents has been proven to predict obesity in later life (**Afshin A et al., 2017**). in addition to a greater possibility of developing cardiovascular illness (**Haass M et al., 2011**).

Even in young persons, obesity can reduce LV size and diastolic function. Hypertrophy of the LV, or LV enlargement, is a hazard factor for cardiovascular disease and mortality. (**Nagueh SF et al., 1997; Sohn D-W., 1997**).

Nagueh SF, Radgoudarzi M., and Leite-Moreira AF (2006) note that it is prevalent among overweight children and adolescents.

Obesity and LVMI have been linked since at least 1991, when it was shown in the Framingham Heart Study that BMI was a significant prognostic of LVM and LVH in adults, even afterward controlling for age & blood pressure (**Jing et al., 2017**).

Some studies find no difference in diastolic function among obese children & lean controls (**Ghanem S et al., 2010; Zoair AM., 2013**), While some researchers detect no difference between obese and thin controls in LV early relaxation, others do. (**Alkholly UM et al., 2016, El Saiedi, 2018, and Borade A, 2011**).

The discrepancies may have resulted from several factors, including differences in ultrasonography techniques, sample sizes, and definitions of overweight and obesity.

Obesity is linked to alterations in LV structure and function, but the pathophysiological mechanisms involved remain unclear. Volume overload caused by obesity-related increases in metabolic rate alters the left ventricular mass (LVM) & the geometric pattern of the LV in reaction to alterations in hemodynamics (**Okpara et al., 2005**).

Ethical consideration:

1. An informed consent was obtained from parents or legal guardians before getting involved in the study.
2. The study was done after approval of ethical committees of Pediatrics department and faculty of medicine for Al-Azhar University.
3. The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.
4. All the data of patients and results of the study are confidential and the patients have the right to keep it.

5. The parents have the right to withdraw from the study at any time without giving any reasons.

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Sample size calculation:

The sample size was calculated using Power and Sample size software version 3 (epi info). The sample size was calculated using the following formula:

(Hoban et al., 2020).

By calculation, the sample size will be equal to 90 in total.

PATIENTS AND METHODS

This is prospective case control study that was done during the period between March 2022 and March 2023, at Al-Hussein and Saied Galal Al-Azhar University Hospitals including 90 children and adolescents (30 obese, 30 overweight, and 30 age- and gender-matched non-obesity controls) our patients were selected according to the following criteria.

Inclusion criteria:

- Age > 6 years and <18 years.

- BMI > 95 percentile for age and sex for obese
- BMI > 85 and < 95 percentile for age and sex for over weight
- BMI < 85 percentile for age and sex for control

Exclusion criteria:

Any child with one or more of the following:

- Diabetic or hypertensive or heart disease.
- Hypothyroidism.
- Significant concomitant illness.
- Obstructive sleep apnea.
- Pulmonary hypertension or lung disease.
- Medication known to modify cardiac function.

Our studied patients were classified into 3 groups:

- **Group 1 obese:** BMI > 95 percentile for age and sex (30 Patient).
- **Group 2 over weight:** BMI > 85 and < 95 percentiles for age and sex.
(30 Patient).
- **Group 3 control:** BMI < 85 percentile for age and sex.
(30 Normal health control).

All studied children were subjected to the following:

I. Complete history with stress on dietetic history, life style habits, family history of diabetes, obesity and hypertension.

II. General & systematic examination including: anthropometric measurements. We utilized conventional equipment to measure height (in meters) to the nearest 0.1 cm on a stadiometer, our weight (in kilograms) to the nearest 0.1 kg on a scale, and several body circumferences to the nearest 0.1 cm on a measuring tape after removing shoes and other heavy things. (Schusterova I. et al., 2013 and Peterson LR, 2004).

BMI was determined using Quetelet's formula (weight in kilos proportional to height in meters squared).

The pediatric cardiologist focused on cardiac examination. A blood pressure measurement using two cuffs of varying sizes on each arm.

III. Laboratory evaluation including: subsequent biochemical markers were measured: hemoglobin A1c (percent), aspartate amino-transferase (AST), alanine amino-transferase (ALT), and thyroid stimulating hormone (TSH). Those children who

were found to have abnormally high levels of TSH were excluded from the trial and sent to an endocrinologist for additional testing. Also, lipid profile were done to all cases & controls.

IV. Echocardiographic study: After positioning the patient so that they were lying on their left side with their knees bent, an echocardiogram was carried out on them using a transducer that was scaled to their body. In the longitudinal direction, we recorded the left atrial diameter (LAD), intraventricular septal thickness (IVSd), left ventricular end diastolic dimension (LVIDd), left ventricular posterior wall dimension (LVPWd), left ventricular mass (LVM), intraventricular septal thickness (IVSs), left ventricular end systolic dimension (LVIDs), left ventricular posterior wall dimension (LVPWs), and ejection fraction (EF). According to Franssen WM et al.'s 2019 research, a more accurate estimate of the LVM can be obtained for younger patients by multiplying the patient's height in meters by 2.7. WM et al. (2019) determined that the value for

LVH in females should be greater than 40 g/m^{2.7}, whereas the value for LVH in men should be greater than 45 g/m^{2.7}. Using the formula $(IVSd+LVPWd)/LVIDd$, we were able to determine the relative wall thickness (RWT), and we determined that values more than 0.41 were regarded abnormal, indicating a departure from the 95th percentile [28]. Using LVMI and RWT,

Tissue Doppler imaging, abbreviated as TDI, will be utilized in order to measure diastolic performance. The sample volume was positioned at the septal & lateral mitral annuli such that the e' and a' velocities could be calculated. The speeds at both the starting point and the ending point were averaged out. As a surrogate for the volumetric pressure of (LV) in early diastole, proportional to early mitral flow velocity (E) to early mitral annulus diastolic velocity (e') was

utilized. An E/e' ratio that was high indicated that there was insufficient relaxation.

Statistical analysis: our data were statistically analyzed using:

In order to certain whether or not the data followed a normal distribution, the Shapiro-Walk test was carried out.

Both the Chi-squared test (2) and the Fisher exact test were utilized in order to determine whether or not there were significant differences across qualitative variables. Quantitative information was displayed as mean and standard deviation (standard deviation) for parametric data.

The one-way analysis of variance (ANOVA) test was utilized in order to make comparisons across several dependent groups that had variables that followed a normal distribution.

RESULTS

Our data will be demonstrated in the following:

Table (1): Demographic characteristics of the three studied groups & vital signs

		<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (N=30)	<i>F/χ²</i>	<i>P</i>
<i>Age (years)</i> <i>Mean ± SD</i>		16.35 ± 4.71	17.42 ± 4.56	16.83 ± 4.69	.398	.673
<i>Sex</i>	<i>Female</i>	18 (60%)	16 (53.3%)	14 (46.7%)	3.4	.183
	<i>Male</i>	12 (40%)	14 (46.7%)	16 (53.3%)		
<i>BMI (kg/m²)</i> <i>Mean ± SD</i>		30.73 ± 4.57	27.83 ± 4.93	20.69 ± 2.67	43	<0.001
<i>HR (beat/min)</i> <i>Mean ± SD</i>		85.19 ± 13.29	83.77 ± 16.23	77.3 ± 15.8	.463	.631
<i>MAP (mmHg)</i> <i>Mean ± SD</i>		78.38 ± 15.88	80.03 ± 15.39	80.07 ± 13.99	.178	.837
<i>RR (Cycle/min)</i> <i>Mean ± SD</i>		22.37 ± 5.92	23.54 ± 4.99	21.63 ± 5.01	.208	.813

There is no substantial variance in BMI among the three groups as well as vital signs.

Table (2): Laboratory parameters among the three studied groups

	Group I	Group II	Group III		
	<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (N=30)	<i>F</i>	<i>P</i>
<i>Hb (g/dL)</i> <i>Mean ± SD</i>	12.81 ± 2.15	12.4 ± 2.31	13.12 ± 0.757	1.11	.333
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>TLC (x 10³/L)</i> <i>Mean ± SD</i>	8.51 ± 3.65	8.75 ± 3.16	7.3 ± 1.49	2.13	.125
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>PLT (x 10³/L)</i> <i>Mean ± SD</i>	283.43 ± 47.9	275.3 ± 71.3	291.5 ± 36.64	.677	.511
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>Creatinine</i> (mg/dl) <i>Mean ± SD</i>	0.612 ± 0.183	0.647 ± 0.206	0.690 ± 0.147	1.37	.260
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>ALT (IU/L)</i> <i>Mean ± SD</i>	36.6 ± 8.45	35.2 ± 9.94	37.1 ± 8.92	.350	.706
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>AST (IU/L)</i> <i>Mean ± SD</i>	31.1 ± 8.4	32.68 ± 7.19	30.87 ± 6.1	.548	.580
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>Albumin (g/dL)</i> <i>Mean ± SD</i>	3.58 ± 0.710	3.61 ± 0.299	3.67 ± 0.182	.302	.741
<i>P-value</i>	P1 >0.05	P2 >0.05	P3 >0.05		
<i>RBS (mg/dL)</i> <i>Mean ± SD</i>	123.6 ± 11.54	117.33 ± 8.53	114.59 ± 10.74	6	.004
<i>P-value</i>	P1 .019	P2 .003	P3 .278		

This table shows statistical insignificant difference between the three studied groups

regarding all laboratory findings except RBS that increased in obese children.

P1: correlation between Group I & II.

P2: correlation between Group I & III.

P3: correlation between Group II & III.

Table (3): Lipid profile among the three groups

	<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (n=30)	<i>F</i>	<i>P</i>
Cholesterol (TC) (mg/dL) Mean ± SD	183.21 ± 35.05	178.17 ± 31.96	154.74 ± 29.67	6.64	.002
	P1 0.563	P2 .001	P3 .005		
Triglycerides (mg/dL) Mean ± SD	124.5 ± 39.35	119.17 ± 36.38	108.4 ± 29.74	1.61	.205
	P1 >0.05	P2 >0.05	P3 >0.05		
LDL (mg/dL) Mean ± SD	117.5 ± 26.71	111.54 ± 25.83	93.8 ± 20.16	7.65	.001
	P1 .383	P2 .001	P3 .004		
HDL (mg/dL) Mean ± SD	46.67 ± 7.55	45.92 ± 8.46	48.4 ± 7.71	.774	.464
	P1 >0.05	P2 >0.05	P3 >0.05		

This table show insignificant difference between the three studied groups regarding lipid profile except LDL & TC. significantly increased in obese group than other groups.

Table (4): Echo parameters among the three studied groups

	<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (n=30)	<i>F</i>	<i>P</i>
LVSD Mean ± SD	0.831 ± 0.155	0.79 ± 0.167	0.64 ± 0.095	15	<0.001
	P1 .328	P2 <0.001	P3 <0.001		
LVSs Mean ± SD	1.25 ± 0.35	1.19 ± 0.3	0.84 ± 0.17	18	<0.001
	P1 .479	P2 <0.001	P3 <0.001		
LVIDd Mean ± SD	4.5 ± 0.571	4.14 ± 0.483	3.89 ± 0.182	14	<0.001
	P1 .011	P2 <0.001	P3 .010		
LVIDs Mean ± SD	2.86 ± 0.557	2.62 ± 0.468	2.34 ± 0.107	11	<0.001
	P1 .076	P2 <0.001	P3 .002		
LV PWD Mean ± SD	0.82 ± 0.238	0.764 ± 0.216	0.457 ± 0.163	26	<0.001
	P1 .344	P2 <0.001	P3 <0.001		
LA size Mean ± SD	3.27 ± 0.529	2.73 ± 0.432	2.14 ± 0.261	54	<0.001
	P1 <0.001	P2 <0.001	P3 <0.001		

The dimensions of the left side of the heart vary significantly between the three studied groups, as seen in the table above.

Table (5): Systolic function parameters among the three studied groups

	<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (n=30)	<i>F</i>	<i>P</i>
LVEF <i>Mean ± SD</i>	68.78 ± 6.57	69.35 ± 3.51	69.26 ± 4.23	.115	.891
	P1 >0.05	P2 >0.05	P3 >0.05		
LVFS <i>Mean ± SD</i>	38.62 ± 4.67	37.65 ± 4.18	38.32 ± 5.02	.344	.710
	P1 >0.05	P2 >0.05	P3 >0.05		
RWT <i>Mean ± SD</i>	0.372 ± 0.105	0.339 ± 0.107	0.264 ± 0.071	10	<0.001
	P1 .233	P2 <0.001	P3 .002		
LV mass <i>Mean ± SD</i>	124.65 ± 39.75	91.74 ± 34.48	58.39 ± 18.22	32	<0.001
	P1 .001	P2 <0.001	P3 <0.001		
LV mass index <i>Mean ± SD</i>	72.36 ± 23.55	61.54 ± 19.46	45.34 ± 9.71	16	<0.001
	P1 .057	P2 <0.001	P3 <0.001		

The data in this table demonstrates that the groups differ significantly with respect

to RWT, LV mass, and LV mass index.

Table (6): LV morphology among the three studied groups

	<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (n=30)	<i>F</i>	<i>P</i>
Normal	22 (73.3%)	26 (86.7%)	30 (100%)	9.48	.051
Concentric remodeling	3 (10%)	1 (3.3%)	0		
Concentric hypertrophy	5 (16.7%)	3 (10%)	0		

The data in the table reveal that there is not a statistically significant variance in LV

morphology among the two groups.

Table (7): Diastolic function parameters among the three studied groups

	<i>Obese</i> (N=30)	<i>Overweight</i> (N=30)	<i>Controls</i> (n=30)	<i>F</i>	<i>P</i>
<i>MV E</i> Mean ± SD	1.09 ± 0.185	1.02 ± 0.154	0.89 ± 0.119	13	<0.001
	P1 .651	P2 <0.001	P3 <0.001		
<i>MV A</i> Mean ± SD	0.641 ± 0.115	0.593 ± 0.107	0.534 ± 0.064	8.99	<0.001
	.099	P2 <0.001	P3 .012		
<i>E/A ratio</i> Mean ± SD	1.7 ± 0.351	1.58 ± 0.306	1.43 ± 0.171	6.69	.002
	.163	P2 <0.001	P3 .023		

This table demonstrates that there is a statistically significant variation in MV E & A waves,

and E/A ratio across the investigated groups.

DISCUSSION

Overweight and obesity are global epidemics that affect people of all ages and have serious effects for their health.

In our study we found that there is no significant statistical difference between the studied groups regarding vital signs, also **Jing et al. (2017)** observed that neither the normal weight nor the obese or overweight groups differed significantly in terms of age, sex, heart rate, or mean arterial pressure.

Furthermore, **Mohamed et al. (2021)** found that when comparing the normal weight group to the obese or overweight participants, there was no statistically significant change in heart rates.

Also, **Alkholy et al. (2016)** found that the heart rate & systolic blood pressure of obese & overweight children & adolescents are considerably higher than those of normal weight children and adolescents. This result could be explained by differences in the sample sizes of the two groups.

Radgoudarzi et al. (2020) found that the blood sugar levels of overweight and obese kids & adolescents tend to be higher than those of children and adolescents of normal weight and this agree with our result.

Similar to this study, **Borade et al. (2011)** found that patients with a high BMI had higher blood sugar levels than those with a normal BMI. The body mass index

was also discovered to correlate positively with random blood sugar measurements.

Also, **Alkholy et al., (2016)** revealed that fasting glucose, fasting insulin & the HOMA index were all considerably greater ($p < 0.05$) in obese children than in the control group.

Our study show that Triglycerides (TG) and high-density lipoprotein (HDL) were not considerably different between the three groups, whereas total cholesterol (TC) & low-density lipoprotein (LDL) were significantly increased in obese & overweight than control, and this agree with result of **Alkholy et al., (2016)**.

Ghanem et al., (2010) and **Zoair et al., (2013)** have demonstrated that TC and LDL are substantially elevated in obese/overweight children and adolescents & this agree with our result.

By comparing echo parameters among the three studied groups, it was determined that the dimensions of the LV were substantially larger in the obese and overweight groups. LV septal wall in diastole (LVSD), LV posterior wall thickness in diastole (LVPWd), LV relative wall thickness (RWT), LV end diastolic and systolic diameter

(LVEDD & LVESD), and LV mass (P -value < 0.000) were considerably different among obese and non-obese groups.

The obese group had substantially larger left atrial (LA) dimensions than the control group ($P < 0.0001$).

Also, **Ghanem et al. (2010)** found that Obese children have been found to have LV abnormalities (such as larger left ventricular wall dimensions and mass and decreased diastolic performance.

According to **Okpara et al. (2005)**, Left atrial hypertrophy was correlated with a worse prognosis for cardiovascular events. Obesity was associated with an increase in left ventricular mass, left ventricular hypertrophy, LV internal diameter, and LV wall thickness.

In the 2018 study by **El Saiedi et al.**, showed that LV end diastolic & end systolic dimensions, interventricular septal dimensions, and posterior wall dimensions did not differ significantly among obese cases & controls, early diastolic transmitral filling velocity was lower in cases matched to controls ($p = 0.004$). Cases with left ventricular hypertrophy were not included, which may explain the disparity.

In terms of systolic function parameters, the obese and overweight groups show significant increases in resting weight, LV mass & LV mass index, indicating a statistically significant difference between groups.

Comparing the RWT, LV mass & LV mass index of obese and overweight individuals to those of controls, we found results comparable to those observed by **(Mohamed et al. 2021)**. Nonetheless, there were no discernible changes in the percentage of ejections.

Alkholy et al. (2016). showed no statistically significant difference in systolic function (as measured by ejection percentage and fractional shortening) between adolescents who were either overweight or obese and a control group.

Although **El Saiedi et al. (2018)** did not detect a substantial variance in LVEF among the obese and non-obese groups, they did identify a statistically substantial variance in LV mass index.

The LV ejection fraction (LVEF) and LV filling size (LVFS) of **Zoair et al. (2013)** did not vary considerably among the two groups statistically. Therefore,

all of the children had normal left ventricular (LV) systolic function.

Schusterova et al. (2013). found that overweight children have an enlarged left ventricle, diastolic dysfunction, and preserved systolic function.

The degree of concentric remodeling and hypertrophy in the left ventricles (LVs) of obese and overweight patients was marginally higher than that of controls.

It's possible that **Mohamed et al.'s (2021)** smaller sample size of obese teenagers compared to controls explains this discrepancy insignificance. Peterson et al. (2004). also discovered that the most prevalent cardiac anomaly in overweight patients was concentric LV hypertrophy. Obesity-related left ventricular hypertrophy (LVH) is a powerful independent predictor of cardiovascular morbidity and mortality, as determined by echocardiography (1990).

I agree, as this inquiry has proved. **Mohamed et al., (2021)**. showed that the E/A ratio was considerably greater in the obese group matched to the control group. Diastolic function appears to be impaired in the obese matched to a control group.

Wouter et al. (2019). Reevaluated previous findings that found significantly higher mitral A-wave velocities and E-wave velocities in obese adolescents matched to lean controls.

While we did detect significant differences in diastolic function parameters among the obese and non-obese groups, **Schusterova et al. (2013)** did not **Ghanem et al. (2010)** also found no relation between childhood obesity and diastolic function.

CONCLUSIONS

- The current study concluded that LV dimensions, systolic function parameters & diastolic function parameters were significantly altered in obese and overweight children and adolescents matched to non-obese patients.
- It appears that the severity of obesity amplifies these alterations.
- Alterations in lipid metabolism may have a role in the development of several heart conditions.
- Hyperglycemia and dyslipidemia are both more common in children and adolescents who are overweight or obese.
- Echocardiography, in conjunction with lipid profile

testing, is necessary for a comprehensive evaluation of heart function in obese children.

RECOMMENDATION

we recommend conducting additional research using a larger sample size and a broader geographic scope.

- Sample size, length of follow-up & the extent of weight loss are just three of the confounding factors that need further investigation.
- Our findings highlight the most sensitive approaches for the early diagnosis of LVDF in children with OW/Ob and should aid in the development of pediatric recommendations for the evaluation of LVDF.

LIMITATIONS

1. Limited numbers of studied children at our hospital.
2. Repeated echocardiography examination is difficult.
3. Associated heart disease, hypertension with some cases.

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