

# Left Ventricular Hypertrophy in Obese Children

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## Abstract

Childhood obesity is one of the most troubling health conditions worldwide, and it is associated with many diseases that eventually lead to serious morbidities and mortality in adulthood, such as insulin resistance, type 2 diabetes mellitus, high blood pressure, dyslipidemia, obstructive sleep apnea and subsequent renal, and liver and cardiovascular diseases.

Cardiovascular disease carries the most risk of all of the long-term outcomes of obesity because it is associated with serious complications in adult life, such as heart failure, acute coronary syndrome, and premature sudden death.

This review aims to address childhood obesity as a worldwide health concern and focuses on the cardiovascular risk and adverse outcomes associated with this condition. This review presents details of obesity-related cardiac structural and functional changes, such as left ventricular hypertrophy (LVH) and dysfunction, respectively. A search of multiple medical databases was performed, and the results yielded many studies related to pediatric left ventricular hypertrophy; seventeen studies were found on left ventricular hypertrophy in obese children from 1980 to 2015.

Most of these studies demonstrated that obesity and its comorbidities are important predictors for left ventricular hypertrophy in the pediatric age group. Furthermore, weight reduction is an important measure to reverse these structural changes and reduce the associated risks.

**Keywords:** Obesity; Left ventricular hypertrophy; Obesity; Children

## Introduction

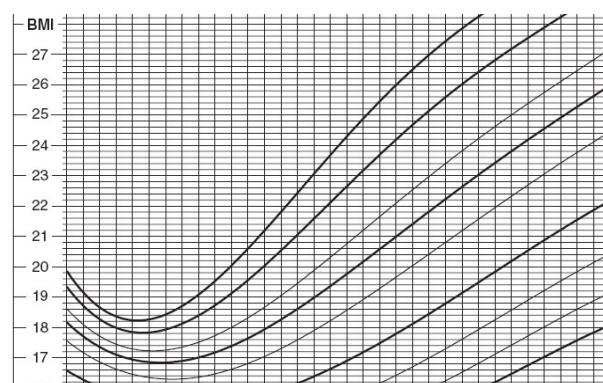
Childhood obesity is one of the most troubling health conditions worldwide [1]. The association of this condition with many diseases that eventually lead to serious morbidities and mortality in adulthood makes it a challenging public health concern. Morbidities include insulin resistance, type 2 diabetes mellitus, high blood pressure, dyslipidemia, obstructive sleep apnea and subsequent renal, liver and cardiovascular diseases. Obesity is also a risk factor for multiple cancers in adults [2-4].

The prevalence of childhood obesity has increased significantly in recent years, and it has become an epidemic in certain areas. The recent WHO Global Database on Child Growth and Malnutrition report states “in the year 2013, an estimate of 42 million children under the age of five around the world are either overweight or obese” [5]. In addition to that, the prevalence of obesity is increasing in all childhood ages, including both males and females of different ethnicities, [6]. Obesity is most often caused by a disturbance in energy balance, which occurs when a child embraces a sedentary life style with decreased physical activity, increased caloric intake, or both. Other factors, such as hormonal, psychosocial, medical and genetic factors, also contribute to the development of obesity but to a much lesser extent.

Classification of childhood obesity as “overweight” or “obese” is determined using the body mass index (BMI) of the child compared to the average BMI for children of the same sex and age rather than the fixed parameters used in the adult index. These measurements are plotted on the growth charts displayed in Figures 1 and 2.

The Center of Disease Control and Prevention defines ‘overweight’ as “a BMI at or above the 85<sup>th</sup> percentile and below the 95<sup>th</sup> percentile for children and teens of the same age and sex”. ‘Obesity’ is defined as “a BMI at or above the 95<sup>th</sup> percentile for children and teens of the same age and sex”.

Body mass index is calculated as the child’s weight in kilograms divided by the square of his\her height in meters. Many studies reported that obese children are more likely to turn into obese adults [7]. This



**Figure 1:** 2 to 20 years of age: Male body mass index-for-age percentiles Clinical growth chart for the 5th, 10th, 25th, 50th, 75th, 85th, 90th, 95th percentiles.

observation emphasizes the importance of addressing this problem at an early stage.

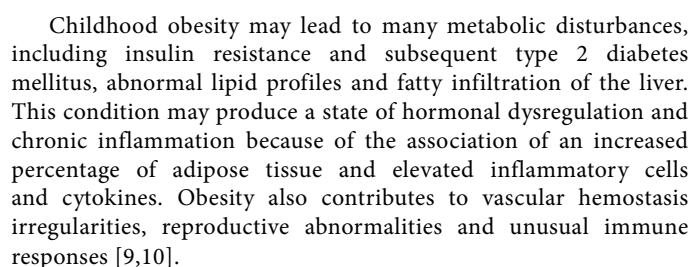
Cardiovascular complications carry the greatest risk of all of the long-term complications of obesity because they are associated with serious consequences in adult life, such as heart failure, acute coronary syndrome and premature sudden death [8]. This review addresses childhood obesity and short-term changes related to obesity, including hormonal, metabolic, structural and functional changes, and it focuses on cardiovascular changes and risk. Details address cardiac structural

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**Received** February 16 2016; **Accepted** April 21, 2016; **Published** April 24, 2016

**Citation:** Attar RZ, Safdar OY, Ghoneim A, Alnajjar AA, Azhar AS, et al. (2016) Left Ventricular Hypertrophy in Obese Children. J Obes Weight Loss Ther 6: 309. doi:10.4172/2165-7904.1000309

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Weight is strongly associated with blood pressure, which supports the importance of weight loss on reducing elevated blood pressure. Weight loss is one of the most effective measures in the management and prevention of hypertension and its complications. Clarke et al.

Ref.	Sample	Date	Outcome
Dušan et al. [36]	103 obese subjects aged 9 to 19 years	2015	LVH was found in 10 % of subjects
Pieruzzi et al. [44]	526 obese children aged 6 to 15 years	2015	Obesity was significantly associated with diastolic dysfunction and correlated with concentric hypertrophy of the left ventricles ( $P < 0.001$ )
Bonito et al. [58]	281 obese children aged 6 to 16 years	2014	47 % had abnormal LV geometry, 18.5 % had eccentric LVH, 15.5 % had concentric LVH, and 13 % had concentric LV remodeling.
Alp et al. [37]	430 obese children aged 6 to 17 years	2014	BMI and total fat mass were predictors for abnormal left ventricular geometry.
Falkner et al. [51]	301 African -American adolescents aged 13 to 18 years	2013	High left ventricular mass index was found in 24 % obese children compared to 12 % non-obese children.
Dhuper et al. [45]	343 children, 213 obese, 130 non- obese	2011	The obese subjects had significantly higher LV mass index (LVMI; $49.6 \pm 0.9$ vs. $46.0 \pm 1.0$ g/m <sup>2.7</sup> , ( $P = 0.01$ ). Concentric remodeling (CR) was the most prevalent pattern noted in the obese group.
Khositseth et al. [32]	49 obese children aged 3.4 to 15.4 years	2010	47% had LVH, 6% had concentric remodeling, 12% had concentric hypertrophy, and 30% had eccentric hypertrophy.
Movahed et al. [33]	1,500 adolescences between the ages of 12 and 20 years who are actively involved in school sport programs	2009	10.32 % of obese athletes had left ventricular hypertrophy (LVM >215 g) in contrast to 0.2 % of controls. 41.4 % of obese students had relative wall thickness >0.43 vs. 15.7 % of controls.
Chinali et al. [34]	460 adolescent participants aged 14 to 20 years Categorized as overweight, obese, or normal BMI	2006	Left ventricular hypertrophy was more prevalent in obese (33.5 %) and overweight (12.4 %), compared with normal weight participants (3.5 %); $P < (0.001)$ .
Kinik et al. [48]	30 obese children who were matched to a control group	2006	Left ventricular hypertrophy was associated with children who had elevated BMI, insulin resistance, and dyslipidemia.
Friberg et al. [59]	19 obese and 20 lean adolescents were recruited.	2004	Left ventricular mass index was 16 % greater in obese compared to lean adolescents.
Urbina et al. [60]	160 healthy children and young adults, 9 to 22 years old	1995	Weight excess is an important predictor for elevated left ventricular mass.
Stephen et al. [41]	201 children and adolescents from 6 to 17 years old	1995	Lean body mass is a significant predictor for left ventricular mass.

**Table 1:** Summary of published studies of LVH in obese children.

Ref.	Sample	Date	Outcome
<b>Hypertension</b>			
Pieruzzi et al. [44]	526 obese children aged 6 to 15 years	2015	Blood pressure values and hypertension are independently associated with an increase in cardiac mass and the presence of cardiac hypertrophy ( $P < 0.001$ and $P < 0.05$ , respectively).
Falkner et al. [51]	301 African- American adolescents aged 13 to 18 years	2013	LVMI was high in hypertensive children (19 %) compared to children with normal blood pressure (12 %), and it was the highest in obese hypertensive children (57 %).
Pruette et al. [61]	141 hypertensive children	2013	The prevalence of LVH was 35 % overall, and it is more prevalent in obese African Americans than non-obese non- African-Americans
Karen et al [50]	163 adolescents, 44 with normal blood pressure, 116 hypertensive	2004	The prevalence of LVH in hypertensive children was higher (44.5 %) compared to children with normal blood pressure (9.1 %).
Stephen et al. [39]	201 children and adolescents from 6 to 17 years old	1995	The variance of left ventricular mass explained by the model was 77 %. Systolic blood pressure explained only 0.5 % of the variance.
<b>Fat mass and distribution</b>			
Pieruzzi et al. [44]	526 obese children aged 6 to 15 years	2015	Waist circumference z-scores were significantly associated diastolic dysfunction.
Daniels et al. [49]	123 children and adolescent aged 9 to 17 years	1999	There was a significant correlation between fat percentage and fat distribution with left ventricular mass ( $r = 0.37$ ).
<b>Lipid profile</b>			

Kinik et al. [48]	30 obese children who were matched to a control group	2006	There was a slight correlation between insulin and lipid profile with relative wall thickness of the heart, but when these measures were indexed to height, the study failed to show any correlation.
<b>Obstructive sleep apnea or nocturnal hypoxemia</b>			
Avelar et al. [57]	455 severely obese subjects with body mass index 35 to 92 kg/m <sup>2</sup> and 59 non-obese reference subjects.	2007	'Low nocturnal oxygen saturation' compared to other factors, such as systolic BP and BMI, showed the strongest probability for predicting LVH (P<0.001).

**Table 2:** Summary of published studies of LVH associated with other comorbidities.

The IDF consensus definition of metabolic syndrome in children and adolescents					
Age group (years)	Obesity* (WC)	Triglycerides	HDL-C	Blood pressure	Glucose (mmol/L) or Known T2DM
6-<10	>90° Percentile	Metabolic syndrome cannot be diagnosed, but further measurement should be made if there is a family history of metabolic syndrome, T2DM, dyslipidemia, cardiovascular disease, hypertension and/or obesity			
10-<16 Metabolic syndrome	>90° percentile or adult cut-off if lower	>1.7 mmol/L > 150 mg/dL	<1.03 mmol/L (<40 mg/dL)	systolic > 130/ diastolic >85mm Hg	>5.6 mmol/L (100 mg/dL) (if >5.6 mmol/L [or known T2DM] recommend an OGTT)
16+ Metabolic syndrome	Use existing IDF criteria for adults, i.e: Central obesity (defined as waist circumference >94 cm for Europid men and > 80 cm for Europid women, with ethnicity specific values for other groups*) Plus any two of the following four factors: <ul style="list-style-type: none"><li>• Raised triglycerides &gt; 1.7 mmol/L</li><li>• Reduced HDL-cholesterol: &lt;1.03 mmol/L (&lt;40 mg/dL) in males and &lt;1.29 mmol/L (&lt;50 mg/dL) in females, or specific treatment for these lipids abnormalities</li><li>• Raised blood pressure: systolic Bð + 130 or diastolic Bð = 85 mm Hg, or treatment of previously diagnosed hypertension</li><li>• Impaired fasting glycemia (IFG): fasting plasma glucose (FPG)=5.6 „,ol/L (&gt; 100 mg/dL), or previously diagnosed type 2 diabetes</li></ul>				
WC: waist Circumference; HDL-C: high-density lipoprotein cholestrol; T2DM: type 2 diabetes mellitus; OGTT: oral glucose tolerance test. The IDF Consensus group recognise that these are ethnic, gender and age difference but research is still needed an outcomes to establish risk.					

**Table 3:** International Diabetes Federation's definition of metabolic syndrome in children and adolescents

[21,22] used a 10-year retrospective observational study and reported that any weight change directly affected blood pressure. Weight gain led to elevations in blood pressure, and weight loss helped in reduction of blood pressure.

### Childhood obesity and cardiovascular risk

The presence of obesity in children increases the risk of developing cardiovascular disease during adulthood because of the association between obesity and the development of atherosclerosis (which is the main element of the pathogenesis of this disease). Excess adipose tissue in children often leads to physiological, inflammatory and metabolic disturbances. These dysregulations increase the probability of children exhibiting signs of premature cardiac dysfunction and structural abnormalities in subsequent events. Therefore, obese children may exhibit cardiometabolic patterns that are similar to obese adults [23,24].

Wong et al. [25] defined obesity-related cardiomyopathy as "myocardial disease in obese individual that cannot be explained by diabetes mellitus, hypertension, coronary artery disease or other etiologies".

Recent studies identified obesity as an independent predictor for heart [26,27]. Furthermore, the duration of Obesity is another important predictor for the development of cardiac dysfunction and structural abnormalities, which predisposes children to heart failure in early adulthood [28,29]. Since obese children tend to grow into obese adults, early identification of these cardiovascular changes and weight reduction are important to ensure the reversibility of these changes.

### Obesity-related left ventricular hypertrophy (LVH) and cardiac dysfunction

Left ventricular geometry is assessed using echocardiographic

measurements. It is classified into the following four groups based on left ventricular mass (LVM) and relative wall thickness (RWT): concentric hypertrophy "increased mass and increased relative wall thickness", eccentric hypertrophy "increased mass and normal relative wall thickness", concentric remodeling "normal mass and increased relative wall thickness", and normal geometry "normal mass and normal relative wall thickness" [30] (Figure 3). Left ventricular mass index (LVMI) is calculated by indexing the echocardiographic measurement to height to a 2.7 power. One study of more than 2000 children revealed that "for children above 9 years LVM/height 2.7 values > 40 g/m 2.7 in girls and >45 g/m 2.7 in boys can be considered abnormal because it falls beyond the 95<sup>th</sup> percentile of the normal LVMI distribution for age and sex" [31]. Cardiac diastolic function is evaluated using a Doppler echocardiography by measuring the mitral E/A ratio.

Obese children, especially children with severe obesity, exhibit a tendency to develop abnormal left ventricular geometry (left ventricular hypertrophy or relative wall thickness) and cardiac dysfunction [32-34]. The underlying pathogenesis is that obese children have increased metabolic activity due to the presence of excess adipose tissue, which leads to increased heart output and total blood volume to meet the metabolic demands. This compensatory process causes the structural changes and cardiac dysfunction that are occasionally observed in obese children [35].

One study of the relative effect of elevated blood pressure and obesity on left ventricular mass demonstrated statistically significant results, which supports the roles of obesity and elevated blood pressure as independent factors that increase the probability of the occurrence of LVH. Data analysis revealed that "obesity OR = 3.26, p<0.001. In addition, high blood pressure showed an OR = 2.92, p<0.001". High blood pressure and obesity together exert an additive effect on the



development of LVH [36-39].

Relative wall thickness correlates directly to BMI. Other metabolic conditions that are generally associated with obesity, such as waist circumference, insulin resistance, and hyperlipidemia, are also associated with LVH [40].

This is a topic of interest because LVH (among other factors) has been established as an independent risk factor for cardiovascular disease in adults. Multiple data analyses demonstrated that LVMI exhibited the highest independent relative risk for the development of these events in the future [41,42].

Among different left ventricular geometry patterns, concentric hypertrophy is the most prevalent type of LV remodeling observed in obese children, and this pattern in particular is shown to have the worst prognosis. One recent study reported [43] that 40% of obese children had concentric hypertrophy, 40% had normal geometry, and 20% had eccentric hypertrophy or concentric remodeling. These signs may be present at an early age in obese children, even before 9 years of age. Other studies reported similar results [44,45]. Obesity prevention and early management using weight reduction (conventional or surgical) are important nonpharmacological measures to ameliorate LVM and reverse most cardiac abnormalities that are associated with obesity in children. One study [46] has evaluated morbidly obese adolescents before and after bariatric surgery and demonstrated that the prevalence of concentric LVH improved from 28% pre-operatively to only 3% at follow up. Furthermore, normal LV geometry was more frequently observed from 36% to 79% at follow up, and diastolic function also improved.

### Lipid profile and LVH

An abnormal lipid profile is one of the metabolic disturbances caused by obesity and elevated fat mass. Moreover, it is associated with cardiovascular accidents, and plays a role in the pathogenesis of developing atherosclerosis. However, it is not well known whether a significant relationship between dyslipidemia and LVH exists or the relationship is not more than an indirect association because dyslipidemia is a “comorbidity of obesity”. A recent study in Indonesia [47] examined the relationship between lipid profile and LVM in obese adolescents, and the results demonstrated no significant association between lipid profile disturbances with LVM. Another study [48] demonstrated that relative left ventricular wall thickness (intra-ventricular septum and left ventricular posterior wall thickness) correlated with insulin, very low-density lipoprotein, and triglyceride

levels. Data in this area are inconsistent, and further research is needed.

### Fat mass and distribution and LVH

In addition to abnormal lipid profile, the percentage of fat and its distribution are also related to LVH. One study demonstrated significant ( $P<0.05$ ) univariate correlations between fat distribution (central deposition of fat-android pattern) and fat mass with LVM. The correlation coefficient between the android pattern and LVH was equal ( $r=0.37$ ) [49].

### High blood pressure and LVH

Hypertension is an independent predictor for the development of LVH, and LVH is the most prevalent end organ damage in children with hypertension. LVH correlates with the severity of hypertension, and it generally indicates a worse prognosis [50]. Falkner et al. [51] investigated 301 African-American adolescents and reported that “adolescents with higher blood pressure had higher left ventricular mass index (33.2 vs. 38.7 gm/m<sup>2.7</sup>,  $p<0.001$ ) and greater left ventricular hypertrophy”. The effect of blood pressure was independent of obesity.

Obesity and high blood pressure promote concentric LV remodeling, but this change is more highly associated with high blood pressure than obesity [52,53].

### Obstructive sleep apnea and LVH

Obstructive sleep apnea is defined as a complete cessation of airflow for >10s during sleep. The decrease in airflow leads to a fall in oxygen saturation (i.e., nocturnal hypoxemia). Obstructive sleep apnea is associated with obesity. A strong association between obstructive sleep apnea and LVH was reported previously [54]. LVH was observed in 78-88% of adults [55,56] as well as 40% of children with obstructive sleep apnea. “Low nocturnal oxygen saturation” was the strongest independent predictor for LVH compared to other factors, such as systolic BP and BMI ( $P<0.001$ ) [57,58].

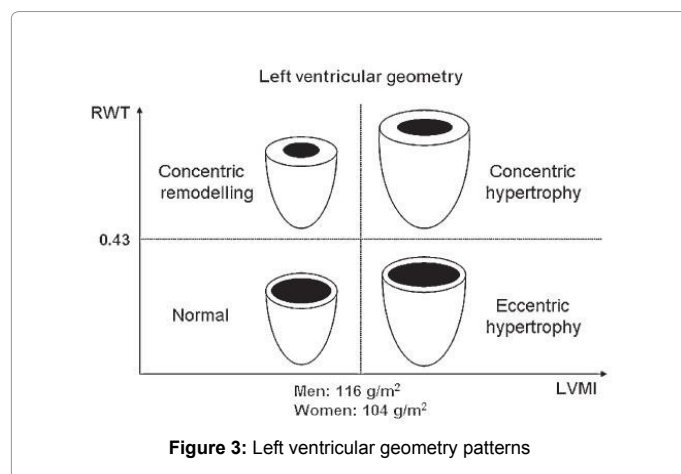
### Conclusion

Obesity is one of the most prevalent public health problems worldwide, and it affects nearly one in five children. Obesity is associated with many short- and long- term complications, and it predisposes children to adult onset obesity, cardiovascular- associated morbidities and premature death.

The process of developing this risk begins during childhood because obesity is an independent risk factor for the development of LVH and cardiac dysfunction. Other factors that are generally associated with obesity, such as dyslipidemia, insulin resistance, fat mass distribution, high blood pressure, and nocturnal hypoxemia, are also related to LVH. These factors, especially high blood pressure and nocturnal hypoxemia, exert a synergetic effect with obesity for the development of LVH, which is more common and prevalent when these two conditions coexist.

Obesity prevention and early management using weight reduction (conventional or surgical) are important nonpharmacological measures to ameliorate left ventricular mass and reverse most cardiac abnormalities that are associated with obesity in children.

Future efforts should focus on parents and the public to recognize the health consequences of obesity in young children and adolescents and spread greater awareness of this condition by encouraging everyone to play an active role in the prevention and early management of obesity in the young. Completion of this goal requires a collaborative effort between the medical profession, government and the public.



## Acknowledgment

Special thanks to Solafa Ghoneim for their valuable assistance in the reviewing and editing of this article.

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