

# Metabolic complications of childhood obesity

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## ABSTRACT

**Context:** Childhood obesity is a global health problem. A percentage of 2.3 of Indian boys and 2.5 of Indian girls are obese. Childhood obesity is associated with many morbidities like diabetes mellitus, coronary artery disease, musculoskeletal problems, and increased mortality. **Aims:** The aim of this study is to estimate burden of metabolic complications of obesity in child and parents of obese children and compare it with normal-weight children. **Settings and Design:** The study was done at a tertiary health center in northern India. It was a cross-sectional study. **Methods and Material:** We enrolled 60 obese children and age- and sex-matched 26 controls, based on Indian Academy of Pediatrics (IAP) 2015 body mass index (BMI) charts. Anthropometric parameters and metabolic complications in family were compared between cases and controls. Clinical markers of metabolic derangements and laboratory metabolic profile were assessed for obese children. **Statistical Analysis Used:** Descriptive statistics was used to describe frequencies. Chi-square test and Mann-Whitney test and Spearman correlation were used for comparison. **Results:** The prevalence of obesity and obesity-related complications was high in families of obese children. Ten percent of obese children had impaired fasting glucose and 30% had Haemoglobin A1c (HbA1c) in prediabetes category. Forty percent of obese children had dyslipidemia, 45% had transaminitis, and 46.7 were vitamin D deficient. A percentage of 41.7 of obese children had fatty liver on ultrasound. **Conclusions:** The family health and child weight are linked through home environment and genetics. The metabolic complications of obesity prediabetes, dyslipidemia, fatty liver, and lower vitamin D level are common in childhood obesity. Regular screening and interventions of metabolic complications are essential for saving child's future health.

**Keywords:** Child, lifestyle, metabolic complications, obesity

## Introduction

Childhood obesity is an emerging and challenging problem of the 21<sup>st</sup> century. The overweight and obesity prevalence in children and adolescents has increased by 47.1% between 1980 and 2013.<sup>[1]</sup> Khadilkar *et al.* did a multisite study on 20,243 children of 2–17 years and found obesity on 18.4% of boys and 12.8% of girls.<sup>[2]</sup> In another Indian study by Chhatwal *et al.* in 2008 children of 9–15 years, prevalence of obesity in males (12.4%) was higher than that of females (9.9%).<sup>[3]</sup> The tracking of childhood obesity in adolescence will lead to greater exposure to obesity throughout their lives, and this increase will contribute to the early

development of type 2 diabetes, fatty liver, and cardiovascular complications.<sup>[4,5]</sup>

The early clinical manifestations of abnormalities, related to childhood obesity like impaired glucose metabolism and nonalcoholic fatty liver disease (NAFLD), are secondary to increased insulin resistance in obesity. Abnormalities of impaired glucose metabolism and NAFLD are clinically silent and clinical suspicion and lab testing are required for the diagnosis. The clinician should gather information from history and a focused physical examination to stratify patients by their risk. Lifestyle-focused interventions have shown promising results in improving the metabolic profile of obese children.<sup>[6]</sup> Early diagnosis and treatment will help in conservation of financial resources which can be better utilized for care of obese children with comorbidities. This study was conducted to evaluate the metabolic complications seen in Indian obese children.

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## Subjects and Methods

Ours was a cross-sectional analytical study. Children of 5–12 years attending pediatric OPD and specialty clinic were screened for inclusion into the study. Children satisfying the inclusion criteria were enrolled after taking informed consent from parents/primary caregivers and assent from the child wherever applicable. Study subjects were categorized based on the IAP 2015 BMI charts for 5–18 years. Children with BMI greater than 27 adult equivalents were classified as obese and children with BMI less than 23 adult equivalents in IAP growth chart 2015 were classified as normal-weight children.

### Inclusion criteria

#### Cases

Children of 5–12 years with exogenous obesity were categorized as obese, according to IAP 2015 growth charts.

#### Controls

Age- and sex-matched children of 5–12 years were categorized as normal weight, according to IAP 2015 growth chart.

### Exclusion criteria

1. <5 years and >12 years
2. Children with syndromic obesity, chronic diseases like rheumatological and endocrinal disorders, renal failure, musculoskeletal disease, and use of medicines affecting bone such as steroids and anticonvulsant drugs.
3. Calcium and vitamin D supplements in the last 6 months.
4. Refusal to give consent.

Normal weight children attending immunization clinic and pediatric OPD for other ailments like upper respiratory tract infection (URI) were enrolled as controls.

Anthropometric evaluation of the study participants was carried out in the growth laboratory of the institute using standardized instruments and techniques.<sup>[7]</sup>

The record of blood investigations was done in obese children as a part of clinical care and recorded in proforma and no special blood investigation was carried out in normal-weight children. Standard pediatric reference intervals for the laboratory investigations were taken to define abnormal lab results.<sup>[8]</sup>

The study was conducted after obtaining ethical clearance from the ethical committee of the institute. Informed consent was obtained from the parents of the participants and assent was obtained from the participants.

All the data were coded and entered in the excel sheet for analysis. This excel sheet was exported to the Statistical Package for Social Sciences (SPSS) software and proper labeling and attributes were added. All analyses were performed using SPSS for

Windows (Version 23.0. Armonk, NY: IBM Corp.). Comparisons of means were done with *t*-tests and Mann–Whitney tests for variables with normal and skewed distribution, respectively. Correlations were done with the Pearson and Spearman correlation coefficient for variables with normal and skewed distribution, respectively.

## Results

### Demographic distribution

We enrolled 60 obese (42 males) and 26 controls (18 males) who were age and sex matched. Mean age of cases was  $9.5 \pm 2.1$  years. Among cases, mean age of males was  $9.9 \pm 2.0$  years and mean age of females was  $8.4 \pm 2.0$  years. Mean age of all controls was  $9.2 \pm 2.2$  years, while that of males in the control group was  $9.5 \pm 2.2$  years and of females was  $8.7 \pm 2.3$  years.

Majority of cases were from urban background (70%,  $n = 42$ ) and belonged to lower middle and upper lower strata according to Modified Kuppaswamy Scale 2019. Fifty percent of normal-weighted children resided in rural areas and most of them belonged to upper lower socioeconomic class.

### Family history of obesity and related diseases

Median BMI of father was 26.3 (range: 19.9–40.0)  $\text{kg}/\text{m}^2$  in cases and 24.2 (range: 19.1–28.0)  $\text{kg}/\text{m}^2$  in controls. Median BMI of mother was 26.4 (range: 20.6–39.1)  $\text{kg}/\text{m}^2$  in cases and 23 (range: 19.4–37.10)  $\text{kg}/\text{m}^2$  in controls. The prevalence of overweight and obesity in father of obese children and normal controls was 65% ( $n = 65\%$ ), 26.7% ( $n = 16$ ) and 61.5% ( $n = 16$ ), and 7.7% ( $n = 2$ ). Mothers of 50% ( $n = 22$ ) of obese children and 46.2% ( $n = 12$ ) of normal weight were overweight. Obesity was prevalent in 36.7% ( $n = 22$ ) and 11.5% ( $n = 3$ ) of mothers of obese and normal weight children, respectively. The BMI of both mother and father was significantly correlated with BMI of their child ( $P$ -value  $< 0.01$ ) [Figure 1]. The prevalence of obesity, diabetes, and hypertension were greater in families of obese child as compared to normal weight child [Table 1].

### Anthropometric parameters of cases and controls

Table 2 shows anthropometric parameters of males and females of obese children with that of normal-weight children. Anthropometric parameters like weight, BMI, waist circumference, waist–hip ratio, triceps skinfold thickness, biceps skinfold thickness, subscapular skinfold thickness, and suprailiac skinfold thickness were found to be higher in obese boys and obese girls when compared to normal weight boys and girls, respectively. Height was found to be higher in obese boys than normal weight boys; but there was no statistically significant difference in height between obese girls and normal weight girls. Most of the participants were prepubertal (25% cases, 23% control) or in early puberty (73% cases, 77% control)

### Intervention done to reduce weight in cases

Out of 60 cases, 40% ( $n = 24$ ) did intervention to reduce weight. However, controls didn't do any intervention to reduce

their weight. Out of 24 cases who did intervention to reduce weight, 21.7% ( $n = 13$ ) used exercise, 1.7% ( $n = 1$ ) used dietary modification, and 16.7% ( $n = 10$ ) used both exercise and dietary modification as modalities for reducing weight. Out of 23 cases doing exercise, 43.5% ( $n = 10$ ) were doing cycling, 17.4% ( $n = 4$ )

were doing jogging, 13% ( $n = 3$ ) were doing dancing, 8.7% ( $n = 2$ ) were doing exercise and swimming, and 4.3% ( $n = 1$ ) were playing football and also doing both dancing and cycling. Out of 23 cases doing exercise, majority (69.6%,  $n = 16$ ) were doing it daily for 30 min, 17.4% ( $n = 4$ ) were doing it daily for 45 min, 8.7% ( $n = 2$ ) were doing it daily for 15 min, and 4.3% ( $n = 1$ ) were doing it daily for 60 min.

**Table 1: Prevalence of obesity related complications in family**

Variable	Family of obese child	Family of normal weight child	P
Obesity	27	4	<0.01
Diabetes	28	3	<0.01
Hypertension	25	3	<0.01
Heart disease	1	1	0.52

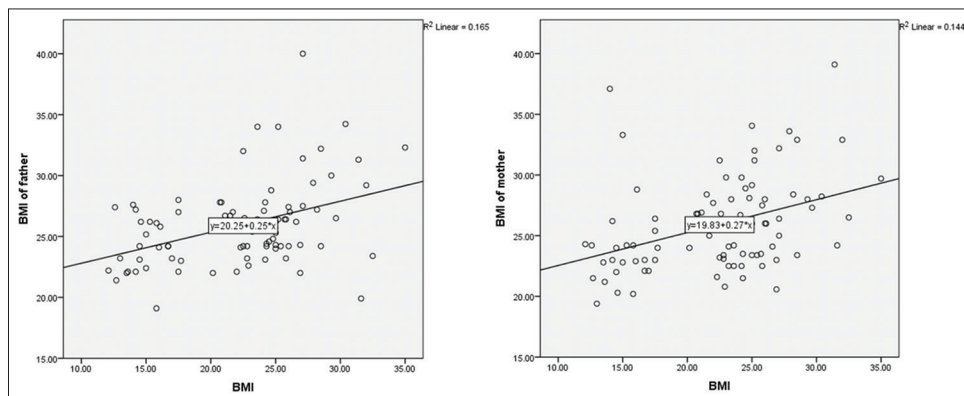
**Clinical markers of metabolic syndrome**

On examination, majority (76.7%,  $n = 46$ ) of cases had acanthosis nigricans (AN). However, acanthosis was absent in controls. Out of 46 cases having AN, majority (54.3%,  $n = 25$ ) were having grade II acanthosis, 28.3% ( $n = 13$ ) and 17.4% ( $n = 8$ ) were having grade I and grade III acanthosis, respectively. Thirty-five

**Table 2: Comparison of anthropometric parameters between cases and controls**

Anthropometric parameters	Gender	Cases Mean±SD	Controls Mean±SD	P
Height (cm)	Male	140.47±10.70	132.52±14.15	0.022 <sup>#</sup>
	Female	132.29±13.12	128.33±14.93	0.486
Height Z score	Male	0.58±1.39	-0.27±0.95	0.024 <sup>#</sup>
	Female	0.74±0.82	-0.10±0.80	0.019 <sup>#</sup>
Weight (kg)	Male	51.84±11.13	27.86±6.75	0.000 <sup>*</sup>
	Female	42.34±10.51	23.96±7.94	0.000 <sup>*</sup>
Weight Z score	Male	2.00±0.76	-0.38±0.60	0.000 <sup>*</sup>
	Female	2.08±0.60	-0.39±0.92	0.000 <sup>*</sup>
BMI (kg/m <sup>2</sup> )	Male	25.55±4.00 <sup>*</sup>	15.80±2.35 <sup>*</sup>	0.000 <sup>*</sup>
	Female	23.78±2.75	14.12±1.76	0.000 <sup>*</sup>
BMI Z score	Male	2.24±0.51	-0.32±0.59	0.000 <sup>*</sup>
	Female	2.14±0.50 <sup>*</sup>	-1.43±1.44 <sup>*</sup>	0.000 <sup>*</sup>
Waist circumference (cm)	Male	84.81±9.63	57.88±5.86	0.000 <sup>*</sup>
	Female	79.17±8.84	53.78±8.09	0.000 <sup>*</sup>
Waist: hip	Male	0.99±0.06	0.90±0.05	0.000 <sup>*</sup>
	Female	0.95±0.05	0.87±0.05	0.001 <sup>*</sup>
Triceps skinfold thickness (mm)	Male	28.07±7.06	9.89±3.27	0.000 <sup>*</sup>
	Female	25.17±5.60	8.80±2.10	0.000 <sup>*</sup>
Biceps' skinfold thickness (mm)	Male	18.20±10.5 <sup>*</sup>	6.00±2.70 <sup>*</sup>	0.000 <sup>*</sup>
	Female	18.48±5.89	6.18±1.12	0.000 <sup>*</sup>
Subscapular skinfold thickness (mm)	Male	34.40±12.5 <sup>*</sup>	7.40±3.50 <sup>*</sup>	0.000 <sup>*</sup>
	Female	28.10±17.2 <sup>*</sup>	7.60±3.90 <sup>*</sup>	0.000 <sup>*</sup>
Supra iliac skinfold thickness (mm)	Male	34.60±7.36	11.56±5.95	0.000 <sup>*</sup>
	Female	32.87±7.22	10.24±3.98	0.000 <sup>*</sup>

\*Median±IQR. <sup>#</sup>P<0.05, <sup>\*</sup>P<0.01



**Figure 1: Scatter diagram showing linear relationship between BMI of parents and child**

percent ( $n = 21$ ) of cases had hepatomegaly, one case (1.7%) had striae, and one case (1.7%) had skin tags, whereas these were absent in controls.

### Laboratory investigations in cases

Impaired fasting glucose (IFG) was present in 10% ( $n = 6$ ) of cases and 30% ( $n = 18$ ) of cases had Haemoglobin A1c falling into prediabetes category. Fifteen percent ( $n = 9$ ) of cases had high cholesterol, 26.7% ( $n = 16$ ) had borderline cholesterol, whereas 58.3% ( $n = 35$ ) of cases had normal cholesterol. Increased triglycerides (TGs) level was seen in 40% ( $n = 24$ ) of cases. Fifteen percent ( $n = 9$ ) of cases had decreased High density lipoprotein (HDL), whereas majority of cases (85%,  $n = 51$ ) had desirable HDL level. Thirty-five percent ( $n = 21$ ) of cases had increased Aspartate Aminotransferase (AST) and majority (65%,  $n = 39$ ) had normal AST level. Forty-five percent ( $n = 27$ ) of cases had increased alanine aminotransferase (ALT) and 55% ( $n = 33$ ) had normal ALT level. Vitamin D was deficient in majority of cases (46.7%,  $n = 28$ ) and was insufficient in 38.3% ( $n = 23$ ) of cases. A percentage of 41.7 ( $n = 25$ ) of cases had fatty liver on ultrasound abdomen. Out of 25 cases having fatty liver, majority (68%,  $n = 17$ ) had grade I and 32% ( $n = 8$ ) had grade II fatty liver based on ultrasound grading of fatty liver Table 3.

### Discussion

Multiple factors such as genetic predisposition, imbalance between caloric intake and expenditures, and basal metabolic rate variation are implicated in childhood obesity. Parental feeding style, obesogenic home environment, and sedentary lifestyle are major risk factors for childhood obesity. The sharing of genetic pool and home environment predisposes all members of family at risk of development of obesity. Family-based screening will be helpful in identification of the at-risk members of family and moreover, family-based early interventions may prevent or reverse the metabolic derangements in both children and adolescent.<sup>[9]</sup>

We found higher incidence of obesity, diabetes, hypertension, and ischemic heart diseases in families of obese children as

compared to normal weight controls. The parental weight correlated significantly with child's weight.

Obesity is considered as disease of affluent society in India. In our study, most of the obese children belonged to lower socioeconomic strata. This shows shifting of economic dynamics similar to western environment where economic adversity is a risk factor for poor nutrition and obesity in children.<sup>[10-12]</sup>

The association between parental weight and child weight has been linked in many recent studies. The obesogenic home environment and epigenetics may explain this strong association. Zarychta *et al.* showed parent and child dyads with obesity perceived fewer healthier eating options at home and community.<sup>[13]</sup> Obesity is linked with poorer metabolic health and increased incidence of metabolic diseases has been found in family of obese children. Family-based interventions are emerging as new models for prevention and management of childhood obesity.<sup>[14-17]</sup>

Obesity is associated with increased insulin resistance and impaired carbohydrate metabolism. We found AN in 76.7% ( $n = 46$ ) of obese children. IFG was present in 10% ( $n = 6$ ) of cases and 30% ( $n = 18$ ) of cases had HbA1c falling into prediabetes category. Similarly, in the study by Asma Deeb *et al.*, 63.9% of obese and overweight children had AN.<sup>[18]</sup> In a study done by Elham Al Amiri *et al.* in 1034 Emirati obese and overweight children and adolescents, 5.4% and 0.87% had prediabetes and diabetes, respectively, based on Oral glucose tolerance test (OGTT). Similar to our study, HbA1c showed discrepancy with 22.9% of prediabetes and no diabetes.<sup>[19]</sup>

Prasad *et al.* reported AN in 55.55% of obese children and 77.63% of obese adolescents in 224 obese children and adolescents of 6–17 years from Andhra Pradesh. Among the 72 obese children, 8.83% had IFG, 9.7% had impaired glucose tolerance (IGT), and 1.38% had type 2 diabetes mellitus (DM), and among the 152 obese adolescents, 9.86% had IFG, 15.78% had IGT, and 1.97% had type 2 DM.<sup>[20]</sup>

We found increased TGs level in 40% ( $n = 24$ ), high cholesterol in 15% ( $n = 9$ ), borderline cholesterol in 26.7% ( $n = 16$ ), and

Table 3: Mean/Median value of laboratory investigations in cases

Investigations	Mean/Median*	SD/IQR**	Minimum	Maximum
Fasting blood sugar (mg/dL)	86.65	9.55	69	110
HbA1c (%)	5.45*	0.6**	4.7	6.3
Cholesterol (mg/dL)	162*	50.4**	109	296
Triglycerides (mg/dL)	129.7*	58.3**	59.8	366
HDL (mg/dL)	39.65*	7.8**	29.5	140.8
AST (U/L)	38.60*	14.92**	15	212
ALT (U/L)	42.24*	31.32**	13.2	268.08
Calcium (mg/dL)	9.81	0.62	8.4	11.2
Phosphorus (mg/dL)	4.6*	0.9**	2.5	8.3
ALP (U/L)	284.05	75.32	112.4	484
Vitamin D (ng/mL)	13.23*	6.7**	4.2	42.16
PTH (pg/mL)	35.88*	18.77*	11.29	83.80

\*Median, \*\*denotes IQR

decreased HDL in 15% ( $n = 9$ ) of obese children. In another study by Deeb *et al.* in 201 obese and 15 overweight children, 55.3% had dyslipidemia with high cholesterol in 11.7%, high TG in 28.6%, high Low Density Lipoprotein (LDL) in 32.7%, and low HDL in 18% cases.<sup>[18]</sup> National Health and Nutrition Examination Survey for 1999–2006 found dyslipidemia in 42.9% of obese and 22.3% of overweight US adolescents.<sup>[21]</sup>

Hypertriglyceridemia is the hallmark of dyslipidemia in obesity. In obesity, there is increased influx of free fatty acid into the liver, which leads to accumulation of TGs in liver. This in turn leads to an increased hepatic synthesis of very low-density lipoproteins (VLDL). VLDL by competing with the increased TGs remnants transported to liver, for lipoprotein lipase, can affect the lipolysis of chylomicrons by lipoprotein lipase.

We found increased AST in 35% ( $n = 21$ ) and increased ALT in 45% ( $n = 27$ ) of obese children. USG abdomen revealed fatty liver in 41.7% ( $n = 25$ ) of obese children. In the study by Deeb *et al.* in 201 obese and 15 overweight children, 51 patients (24.3%) had either elevated AST or ALT or both (high AST in 14.6% and high ALT in 19.8%). Of those 51 with elevated transaminases, 43 (84%) had fatty liver.<sup>[18]</sup> Elevated transaminases, particularly elevated ALT, are considered as an indicator for NAFLD in obese children and elevation of ALT is considered as an indication for biopsy (59). In the study done by Schwimmer *et al.* in biopsy-proven 100 NAFLD children and adolescents in the United States, obesity was found in 92% of cases.<sup>[22]</sup>

We found vitamin D deficiency in 46.7% ( $n = 28$ ) and insufficiency in 38.3% ( $n = 23$ ) of obese cases. This is similar to the Indian study by Reddy *et al.*, which was done in 16 obese and 14 overweight adolescents, in which 62.5% of obese and 71.4% of overweight adolescents had vitamin D deficiency, 6.25% of obese and 21.4% of overweight adolescents had vitamin D insufficiency.<sup>[23]</sup> In another study done by Christy B. Turer, the prevalence of vitamin D deficiency was determined in 12,292 US children of 6–18 years, and the prevalence of vitamin D deficiency among normal weight, overweight, obese, and severely obese children was 21%, 29%, 34%, and 49%, respectively.<sup>[24]</sup>

The prevalence of lifestyle-related diseases like obesity, diabetes, hypertension, and heart disease is increased in parents of obese child. Our as well as previous studies affirm this finding. Tojjar *et al.* found 31% prevalence of overweight and obesity in diabetic parents in comparison to 21% in healthy control. Similarly, Todd *et al.* found strong association of cardiovascular risks in parents as well as children.<sup>[25,26]</sup> Thus, metabolic health of family members is glued together with genetics and home environment and interventions at family are needed to improve the metabolic health of child as well as parents.

## In Summary

1. Childhood obesity is no longer a disease of affluent society and associated with metabolic complications at presentation.

2. Parental weight and metabolic health are linked to child's weight.
3. The risk of metabolic complications is more in obese children as well as their parents in comparison to healthy control.
4. All family members of an obese child should be screened for obesity and its complications to prevent morbidity and mortality in future.

**Key Messages:** Parental weight and metabolic health are linked to child's weight and family-based interventions will be beneficial for child and family. The metabolic complications of obesity start early in childhood obesity; timely screening and interventions for metabolic complications are essential to prevent morbidity and mortality in future.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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## Conflicts of interest

There are no conflicts of interest.

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