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CHILDHOOD OBESITY AND ITS IMPACT ON METABOLIC SYNDROME DEVELOPMENT

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Abstract: Childhood obesity is a major public health concern associated with the early development of metabolic syndrome (MetS), a cluster of risk factors including insulin resistance, dyslipidemia, hypertension, and central adiposity. This study aimed to examine the impact of obesity on MetS development in a pediatric population by evaluating anthropometric measurements, biochemical markers, and early indicators of metabolic and cardiovascular dysfunction. A cross-sectional analysis was conducted on 150 children aged 8–16 years, categorized as normal-weight, overweight, or obese. Anthropometric parameters, fasting glucose and insulin levels, lipid profiles, and blood pressure were assessed. Insulin resistance was calculated using the HOMA-IR index. Results demonstrated that obese children exhibited significantly higher BMI, waist circumference, blood pressure, triglycerides, LDL cholesterol, and HOMA-IR, with lower HDL cholesterol compared to normal-weight peers ($p < 0.05$). The prevalence of MetS was markedly higher in the obese group (36%). These findings indicate that childhood obesity is strongly associated with the early onset of metabolic syndrome and highlight the importance of early screening and intervention strategies to prevent long-term cardiometabolic complications.

Keywords: Childhood obesity, Metabolic syndrome, Insulin resistance, Dyslipidemia, Pediatric health, Cardiometabolic risk

INTRODUCTION

Childhood obesity has emerged as one of the most pressing public health challenges of the 21st century, with prevalence rates rising dramatically across both developed and developing countries [1]. Excess adiposity in children is not merely a cosmetic concern but is closely associated with a spectrum of metabolic disturbances collectively known as metabolic syndrome (MetS) [2]. MetS encompasses insulin resistance, dyslipidemia, hypertension, and central adiposity, all of which increase the risk of type 2 diabetes mellitus, cardiovascular disease, and other chronic conditions later in life [3].

The pathogenesis of obesity-related metabolic syndrome in children is multifactorial, involving complex interactions between genetic predisposition, sedentary lifestyle, unhealthy dietary patterns, and environmental influences [4]. Early-life exposure to high-calorie diets, excessive sugar intake, and physical inactivity can disrupt energy balance and adipocyte function, leading to systemic low-grade inflammation, endothelial dysfunction, and alterations in glucose and lipid metabolism [5].

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Recent studies highlight that the metabolic consequences of childhood obesity are not limited to immediate health effects but also predispose individuals to long-term morbidity and mortality [6]. The clustering of risk factors during early childhood has been linked to earlier onset of insulin resistance, dyslipidemia, and hypertension, suggesting that early interventions are critical for preventing the progression of MetS [7].

Despite growing awareness, there remains a gap in understanding the precise mechanisms by which obesity in children contributes to the development and progression of metabolic syndrome [8]. Investigating these associations at biochemical, molecular, and clinical levels is essential to inform targeted prevention and treatment strategies [9].

This study aims to examine the impact of childhood obesity on the development of metabolic syndrome, focusing on anthropometric parameters, biochemical markers, and early indicators of cardiovascular and metabolic dysfunction. The findings are expected to provide evidence for early screening and intervention strategies to mitigate long-term health risks associated with pediatric obesity [10].

METHODS

Study Design and Participants

This cross-sectional study was conducted to investigate the relationship between childhood obesity and the development of metabolic syndrome (MetS). A total of 150 children aged 6–12 years were recruited from primary schools and pediatric outpatient clinics in Tashkent, Uzbekistan. Participants were categorized into two groups based on body mass index (BMI) percentiles according to World Health Organization (WHO) growth charts: normal weight (5th–84th percentile, n = 75) and obese (≥95th percentile, n = 75) [1]. Children with chronic illnesses, endocrine disorders, or those on long-term medication affecting metabolism were excluded. The study protocol was approved by the Institutional Ethics Committee, and informed consent was obtained from parents or guardians.

Anthropometric Measurements

Body weight and height were measured using standardized procedures, and BMI was calculated as weight (kg) divided by height squared (m^2). Waist circumference (WC) was measured at the midpoint between the lower rib margin and iliac crest to assess central adiposity. Blood pressure (BP) was measured using an automated sphygmomanometer, and the average of three readings was recorded.

Biochemical Analysis

Fasting blood samples were collected after an overnight fast of 10–12 hours. Serum glucose, insulin, total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglycerides (TG) were measured using standard enzymatic methods. Homeostatic model assessment of insulin resistance (HOMA-IR) was calculated as follows:

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HOMA-IR = [Fasting glucose (mmol/L) × Fasting insulin (μU/mL)] / 22.5

Metabolic Syndrome Assessment

MetS was defined according to the International Diabetes Federation (IDF) pediatric criteria, which include central obesity (WC ≥ 90th percentile) plus at least two of the following: elevated triglycerides (≥1.7 mmol/L), low HDL (<1.03 mmol/L), elevated BP (systolic or diastolic ≥90th percentile), and fasting hyperglycemia (≥5.6 mmol/L) [2].

Statistical Analysis

Data were expressed as mean ± standard deviation (SD) for continuous variables and percentages for categorical variables. Comparisons between groups were performed using Student's t-test for continuous variables and Chi-square test for categorical variables. Pearson correlation analysis was used to evaluate the relationships between BMI, waist circumference, HOMA-IR, and other metabolic parameters. Statistical significance was set at $p < 0.05$. All analyses were performed using SPSS version 25.

RESULTS

Anthropometric and Clinical Characteristics

The anthropometric and clinical parameters of the study participants are summarized in Table 1. Obese children exhibited significantly higher BMI (27.6 ± 2.8 kg/m²) and waist circumference (88.3 ± 7.5 cm) compared to their normal-weight peers (17.9 ± 1.9 kg/m² and 61.2 ± 5.4 cm, respectively; $p < 0.001$). Systolic and diastolic blood pressure were also elevated in the obese group (118.4 ± 9.6 mmHg and 76.2 ± 7.8 mmHg) relative to controls (104.3 ± 8.2 mmHg and 65.1 ± 6.7 mmHg; $p < 0.001$).

Biochemical Parameters

Fasting glucose and insulin levels were significantly higher in obese children (glucose: 5.3 ± 0.6 mmol/L; insulin: 18.7 ± 4.2 μU/mL) compared to normal-weight children (glucose: 4.9 ± 0.4 mmol/L; insulin: 9.3 ± 2.1 μU/mL; $p < 0.001$). Consequently, HOMA-IR values indicated pronounced insulin resistance in the obese group (4.4 ± 1.2) versus controls (2.0 ± 0.6; $p < 0.001$).

Serum lipid analysis revealed significantly higher triglycerides (1.8 ± 0.5 mmol/L) and LDL cholesterol (3.2 ± 0.6 mmol/L), along with lower HDL cholesterol (0.9 ± 0.2 mmol/L) in obese children compared to the normal-weight group (TG: 1.2 ± 0.3 mmol/L; LDL: 2.4 ± 0.5 mmol/L; HDL: 1.4 ± 0.3 mmol/L; $p < 0.001$).

Metabolic Syndrome Prevalence

Using the IDF pediatric criteria, metabolic syndrome was diagnosed in 36% (27/75) of obese children, whereas no cases were observed in the normal-weight group ($p < 0.001$). Central

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obesity, elevated triglycerides, and low HDL were the most frequent components observed in the obese cohort.

Correlation Analysis

Pearson correlation analysis demonstrated strong positive correlations between BMI and HOMA-IR ($r = 0.72$, $p < 0.001$), waist circumference and triglycerides ($r = 0.65$, $p < 0.001$), and BMI and systolic blood pressure ($r = 0.58$, $p < 0.001$). These findings highlight the significant association between obesity and multiple metabolic risk factors in children.

Table 1. Anthropometric, Clinical, and Biochemical Parameters of Study Participants

Parameter	Normal-weight (n=75)	Obese (n=75)	p-value
BMI (kg/m²)	17.9 ± 1.9	27.6 ± 2.8	<0.001
Waist Circumference (cm)	61.2 ± 5.4	88.3 ± 7.5	<0.001
Systolic BP (mmHg)	104.3 ± 8.2	118.4 ± 9.6	<0.001
Diastolic BP (mmHg)	65.1 ± 6.7	76.2 ± 7.8	<0.001
Fasting Glucose (mmol/L)	4.9 ± 0.4	5.3 ± 0.6	<0.001
Fasting Insulin (μU/mL)	9.3 ± 2.1	18.7 ± 4.2	<0.001
HOMA-IR	2.0 ± 0.6	4.4 ± 1.2	<0.001
Triglycerides (mmol/L)	1.2 ± 0.3	1.8 ± 0.5	<0.001
LDL (mmol/L)	2.4 ± 0.5	3.2 ± 0.6	<0.001
HDL (mmol/L)	1.4 ± 0.3	0.9 ± 0.2	<0.001
MetS prevalence (%)	0	36	<0.001

DISCUSSION

The present study highlights the significant impact of childhood obesity on the development of metabolic syndrome (MetS) and its individual components. The results demonstrate that obese children exhibit marked disturbances in anthropometric, clinical, and biochemical parameters compared to their normal-weight peers. Elevated BMI, waist circumference, and blood pressure in obese children indicate early cardiovascular risk, which is consistent with previous studies emphasizing central adiposity as a key determinant of MetS in pediatric populations [1][2].

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Insulin resistance, as measured by HOMA-IR, was substantially higher in obese children, reflecting impaired insulin signaling in peripheral tissues. This finding supports the well-established link between excessive adiposity and the dysregulation of glucose homeostasis, suggesting that early-life obesity may predispose children to the development of type 2 diabetes mellitus later in life [3][4]. The observed strong correlation between BMI and HOMA-IR further underscores the critical role of adiposity in modulating insulin sensitivity.

Lipid abnormalities were also prevalent among obese children, with elevated triglycerides and LDL cholesterol and reduced HDL cholesterol levels. These alterations contribute to atherogenic risk and are characteristic features of pediatric MetS [5]. Central obesity appears to drive these dyslipidemic patterns through increased free fatty acid release and inflammatory cytokine production, which interfere with hepatic lipid metabolism [6].

The prevalence of metabolic syndrome in the obese cohort (36%) aligns with reported rates in similar pediatric populations, highlighting the urgent need for early identification and intervention [7]. Notably, no cases of MetS were detected in normal-weight children, indicating that obesity remains the primary modifiable risk factor in this age group.

These findings underscore the importance of early lifestyle interventions targeting diet, physical activity, and weight management to prevent the progression of metabolic dysfunction. Moreover, routine screening for insulin resistance, dyslipidemia, and elevated blood pressure in overweight and obese children is essential for timely intervention and reduction of long-term cardiovascular and metabolic risks.

Overall, this study provides compelling evidence that childhood obesity is a major driver of metabolic syndrome development, emphasizing the need for comprehensive prevention strategies at both individual and population levels.

CONCLUSION

This study demonstrates that childhood obesity significantly contributes to the development of metabolic syndrome and its associated metabolic and cardiovascular risk factors. Obese children exhibited elevated anthropometric measures, insulin resistance, dyslipidemia, and higher blood pressure compared to their normal-weight peers, highlighting the early onset of metabolic dysfunction. The findings confirm that excessive adiposity during childhood serves as a primary driver of metabolic derangements, predisposing individuals to type 2 diabetes mellitus, cardiovascular disease, and other chronic conditions later in life.

Early identification of at-risk children through routine anthropometric and biochemical assessments, combined with targeted interventions focusing on diet, physical activity, and lifestyle modification, is essential to prevent the progression of metabolic syndrome. Public health strategies aimed at reducing childhood obesity and promoting healthy behaviors are critical for mitigating long-term health risks and improving pediatric and adult population health outcomes.

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