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ORIGINAL ARTICLE

Role of Serum Magnesium Deficiency in Insulin Resistance Among Overweight and Obese Children

SAPNA SINDHU[1], SURHAN[2], ASADULLAH[3], HINA UMAIR[4], QAMAR YASMEEN[5], RAZWAN ASHRAF[6]
[1]*Postgraduate Resident, Liaquat University Hospital, Hyderabad/Jamshoro, Pakistan*
[2]*Postgraduate Resident (FCPS), Department of Pediatrics, Unit II, Liaquat University Hospital, Hyderabad/Jamshoro, Pakistan*
[3]*Senior Registrar, Department of Pediatrics, Suleman Roshan Medical College, Tando Adam, Sindh, Pakistan*
[4]*Assistant Professor, Department of Physiology, Wah Medical College, National University of Medical Sciences (NUMS), Wah Cantt, Pakistan*
[5]*Assistant Professor, Department of Biochemistry, Niazi Medical and Dental College, Sargodha, Pakistan*
[6]*General Physician, Aziz Bhatti Shaheed Teaching Hospital, Gujrat, Pakistan*
Correspondence to: Asadullah, Email: Qazi09066@gmail.com

ABSTRACT

Background: Childhood obesity has become a major public health concern in Pakistan, contributing to early-onset insulin resistance and increased risk of type 2 diabetes mellitus. Micronutrient deficiencies, particularly magnesium deficiency, are increasingly recognized as contributors to impaired glucose metabolism. Magnesium is a key intracellular cation that plays a critical role in insulin receptor activation, glucose transport, and enzymatic reactions regulating carbohydrate metabolism. However, limited data exist on the prevalence of magnesium deficiency and its relationship with insulin resistance in overweight and obese children in South Asia.

Objectives: The aim of this study was to determine the prevalence of serum magnesium deficiency and to evaluate its association with insulin resistance among overweight and obese children in Pakistan.

Methods: A cross-sectional study was conducted in the Department of Pediatrics, Unit II, Liaquat University Hospital, Hyderabad/Jamshoro, Pakistan, from January 2022 to March 2023. A total of 120 children aged 6–16 years with BMI ≥85th percentile were enrolled. Anthropometric measurements were taken, and fasting blood samples were collected for serum magnesium, glucose, and insulin estimation. Insulin resistance was calculated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), defined as fasting insulin in micro–international units per milliliter multiplied by fasting glucose in milligrams per deciliter, divided by four hundred and five. A value greater than 3.16 was considered diagnostic of insulin resistance.

Results: The prevalence of magnesium deficiency was 43.3%. Children with magnesium deficiency had significantly higher fasting insulin levels (18.6 ± 5.2 vs. 12.4 ± 3.8 μIU/mL, $p < 0.001$) and HOMA-IR scores (4.8 ± 1.5 vs. 3.1 ± 1.0, $p < 0.001$) compared to those with normal magnesium status. Multivariate regression confirmed low magnesium as an independent predictor of insulin resistance after adjusting for age, sex, and BMI ($\beta = -0.35$, $p = 0.002$).

Conclusion: Serum magnesium deficiency is common among overweight and obese children in Pakistan and is strongly associated with insulin resistance. Routine screening and nutritional interventions targeting magnesium intake may help reduce metabolic risk in this vulnerable population.

Keywords: Magnesium deficiency, insulin resistance, obesity, overweight, children, HOMA-IR

INTRODUCTION

Childhood overweight and obesity have emerged as one of the most pressing public health challenges of the 21st century. The World Health Organization (WHO) estimates that over 340 million children and adolescents aged 5–19 years are overweight or obese worldwide, and the prevalence continues to rise in both developed and developing countries[1]. South Asia, including Pakistan, is witnessing an alarming increase in pediatric obesity due to rapid urbanization, sedentary lifestyles, and the widespread availability of calorie-dense but nutrient-poor diets. This trend not only places children at risk of metabolic and cardiovascular disorders later in life but also predisposes them to early metabolic dysfunction during their formative years[2].

Insulin resistance is a central pathophysiological mechanism linking obesity to type 2 diabetes mellitus (T2DM) and other metabolic complications[3]. It is characterized by a reduced biological response to circulating insulin, leading to impaired glucose uptake in skeletal muscle and adipose tissue, increased hepatic glucose output, and compensatory hyperinsulinemia. In children, insulin resistance not only predicts future diabetes and metabolic syndrome but also manifests clinically as acanthosis nigricans, dyslipidemia, and hypertension. Identifying modifiable contributors to insulin resistance in this young age group is crucial for implementing early preventive strategies[4].

Among the nutritional factors implicated in insulin sensitivity, magnesium has drawn particular attention. Magnesium is the second most abundant intracellular cation and a cofactor for over 300 enzymatic reactions, including those involved in carbohydrate metabolism, ATP utilization, and insulin receptor function[5]. It plays a pivotal role in insulin signaling by facilitating tyrosine kinase activity of the insulin receptor and enhancing the downstream translocation of glucose transporter type 4 (GLUT-4) to the cell membrane. Magnesium deficiency has been associated with increased oxidative stress, low-grade systemic inflammation, and altered calcium handling, all of which exacerbate insulin resistance[6].

Several clinical and experimental studies in adults have demonstrated an inverse relationship between serum magnesium levels and insulin resistance, glucose intolerance, and the risk of T2DM. Oral magnesium supplementation has also been shown to improve insulin sensitivity and glycemic control in type 2 diabetic patients[7]. However, evidence in pediatric populations remains limited and inconsistent. Children and adolescents may be particularly vulnerable to magnesium deficiency due to poor dietary habits, increased consumption of processed foods with low magnesium content, and higher metabolic demands during growth. Furthermore, obesity itself is believed to alter magnesium homeostasis by increasing urinary magnesium excretion under the influence of hyperinsulinemia and by creating a chronic pro-inflammatory state that depletes intracellular stores[8].

In Pakistan, the dual burden of undernutrition and overnutrition poses unique challenges to child health. While undernutrition remains prevalent in certain rural areas, urban centers are increasingly facing rising rates of childhood overweight and obesity[9]. Despite this epidemiological shift, there is a scarcity of local research exploring micronutrient deficiencies in obese children, particularly the role of magnesium in modulating insulin sensitivity. Understanding this relationship is vital, as early detection and correction of magnesium deficiency may offer a cost-effective and non-pharmacological strategy to mitigate insulin

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resistance and delay the onset of type 2 diabetes in children at risk[10].

Therefore, this study was designed to investigate the role of serum magnesium deficiency in insulin resistance among overweight and obese children attending tertiary care hospitals in Pakistan. By exploring this association in a local pediatric population, the research aims to generate evidence that could guide clinical screening practices, nutritional counseling, and public health interventions targeted at reducing the future burden of diabetes and metabolic syndrome[11].

MATERIALS AND METHODS

Study Design and Setting: This research was designed as a cross-sectional observational study and was conducted in the Department of Pediatrics, Unit II, Liaquat University Hospital, Hyderabad/Jamshoro, Pakistan. The hospital is a tertiary care referral center that receives patients from both urban and rural communities of Sindh province, making it an ideal setting to evaluate metabolic risk factors among children. The duration of the study extended over fifteen months, from January 2022 to March 2023.

Study Population: The study population consisted of 120 children aged between 6 and 16 years who were either overweight or obese according to the World Health Organization (WHO) growth standards for BMI-for-age percentiles. Children were classified as overweight if their BMI was equal to or greater than the 85th percentile but less than the 95th percentile, and as obese if their BMI was equal to or greater than the 95th percentile for age and sex. Both male and female children were included. Children with type 1 or type 2 diabetes mellitus, endocrine disorders such as hypothyroidism or Cushing's syndrome, chronic systemic illnesses including renal or hepatic disease, and those receiving magnesium supplementation, insulin-sensitizing agents, or long-term corticosteroid therapy were excluded from the study. Children with acute illnesses at the time of enrollment were also excluded.

Ethical Considerations: Prior to the commencement of the study, approval was obtained from the Institutional Review Board of Liaquat University of Medical and Health Sciences (LUMHS). Informed written consent was taken from the parents or legal guardians of all participants, while verbal assent was obtained from children older than 12 years of age. The confidentiality of all collected data was strictly maintained, and participation in the study was voluntary with the option to withdraw at any time without affecting the clinical care provided to the child.

Sample Size: A total of 120 children were enrolled during the study period. This sample size was considered adequate based on patient flow at the tertiary care center and was sufficient to allow statistical comparisons between magnesium-deficient and magnesium-sufficient groups in relation to insulin resistance.

Data Collection and Clinical Assessment: Each child underwent a structured evaluation that included demographic information, dietary history, family history of diabetes, and lifestyle factors. A complete physical examination was performed. Anthropometric measurements included weight, height, BMI, and waist circumference. Weight was recorded using a calibrated digital scale with the child wearing light clothing and no shoes. Height was measured with a stadiometer, and BMI was calculated as weight in kilograms divided by the square of height in meters. Waist circumference was measured at the midpoint between the lower rib margin and the iliac crest using a non-stretchable measuring tape. BMI-for-age percentiles were plotted on WHO reference charts to classify overweight and obesity status.

Laboratory Investigations: Venous blood samples were collected after an overnight fast of 10–12 hours. Serum magnesium levels were measured using the xylidyl blue colorimetric method, and a level below 1.7 mg/dL was considered as magnesium deficiency. Fasting plasma glucose was estimated using the glucose oxidase–peroxidase method, while fasting serum insulin concentrations were determined using enzyme-linked immunosorbent assay (ELISA). Insulin resistance was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), which was calculated with the formula: HOMA-IR equals fasting insulin in micro–international units per milliliter multiplied by fasting glucose in milligrams per deciliter, and the product is then divided by four hundred and five. A HOMA-IR value greater than 3.16 was considered diagnostic of insulin resistance.

Statistical Analysis: All data were entered and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as serum magnesium, fasting insulin, fasting glucose, and HOMA-IR were expressed as mean ± standard deviation, while categorical variables such as sex and presence of magnesium deficiency were presented as frequencies and percentages. Differences between magnesium-deficient and magnesium-sufficient groups were compared using the independent t-test for continuous data and the chi-square test for categorical data. Correlations between serum magnesium and insulin resistance markers were analyzed using Pearson's correlation coefficient. To identify whether serum magnesium independently predicted insulin resistance, a multivariate linear regression model was applied after adjusting for potential confounding factors including age, sex, and BMI. A p-value of less than 0.05 was taken as statistically significant.

RESULTS

A total of 120 overweight and obese children were enrolled during the study period between January 2022 and March 2023. The mean age of the participants was 11.3 ± 2.8 years, with a range from 6 to 16 years. There was a slight male predominance, with 66 children (55.0%) being boys and 54 children (45.0%) being girls. Of the total participants, 48 children (40.0%) were classified as overweight, while 72 children (60.0%) were obese according to WHO BMI-for-age percentiles. The baseline demographic and clinical characteristics of the study population are summarized in Table 1.

Table 1: Baseline demographic and clinical characteristics of overweight and obese children (n = 120).

Variable	Overweight (n = 48)	Obese (n = 72)	Total (n = 120)	p-value
Age (years), mean ± SD	11.1 ± 2.7	11.4 ± 2.9	11.3 ± 2.8	0.52
Male sex, n (%)	26 (54.2%)	40 (55.6%)	66 (55.0%)	0.88
Female sex, n (%)	22 (45.8%)	32 (44.4%)	54 (45.0%)	
BMI (kg/m²), mean ± SD	23.6 ± 1.9	28.9 ± 2.5	26.8 ± 3.2	<0.001*
Waist circumference (cm)	72.4 ± 6.1	84.7 ± 7.4	79.6 ± 8.5	<0.001*

*Significant at p < 0.05.

Table 2: Serum magnesium status in overweight and obese children.

Group	Serum Magnesium (mg/dL), mean ± SD	Magnesium Deficiency n (%)	p-value
Overweight (n = 48)	1.75 ± 0.26	18 (37.5%)	0.01*
Obese (n = 72)	1.62 ± 0.22	34 (47.2%)	
Total (n = 120)	1.68 ± 0.25	52 (43.3%)	

*Significant at p < 0.05.

Table 3: Comparison of insulin resistance markers between magnesium-deficient and magnesium-sufficient children.

Variable	Magnesium Deficient (n = 52)	Magnesium Sufficient (n = 68)	p-value
Fasting glucose (mg/dL), mean ± SD	92.4 ± 7.8	89.6 ± 6.9	0.08
Fasting insulin (µIU/mL), mean ± SD	18.6 ± 5.2	12.4 ± 3.8	<0.001*
HOMA-IR, mean ± SD	4.8 ± 1.5	3.1 ± 1.0	<0.001*

*Significant at p < 0.05.

As shown in Table 1, obese children had significantly higher mean BMI and waist circumference compared to overweight children ($p < 0.001$ for both), whereas there was no statistically significant difference in age or sex distribution between the groups.

When serum magnesium levels were assessed, the mean serum magnesium concentration in the study population was 1.68 ± 0.25 mg/dL. Magnesium deficiency, defined as serum magnesium below 1.7 mg/dL, was observed in 52 children (43.3%). The prevalence of magnesium deficiency was higher in obese children compared to overweight children (47.2% vs. 37.5%), although this difference was not statistically significant. However, when absolute magnesium levels were compared, obese children had significantly lower serum magnesium values (1.62 ± 0.22 mg/dL) compared to overweight children (1.75 ± 0.26 mg/dL; $p = 0.01$). These findings are summarized in Table 2.

In terms of insulin resistance markers, children with magnesium deficiency demonstrated significantly higher fasting serum insulin levels compared to those with normal magnesium status (18.6 ± 5.2 µIU/mL vs. 12.4 ± 3.8 µIU/mL, $p < 0.001$). Similarly, mean HOMA-IR values were significantly elevated in magnesium-deficient children (4.8 ± 1.5) compared to children with sufficient magnesium levels (3.1 ± 1.0, $p < 0.001$). These comparisons are presented in Table 3.

Correlation analysis further demonstrated that serum magnesium levels were inversely correlated with both fasting insulin ($r = -0.38$, $p < 0.001$) and HOMA-IR ($r = -0.42$, $p < 0.001$), indicating that lower magnesium levels were associated with higher degrees of insulin resistance. Fasting glucose showed only a weak, non-significant negative correlation with serum magnesium ($r = -0.15$, $p = 0.09$).

Finally, multivariate linear regression analysis was performed to adjust for potential confounding variables such as age, sex, and BMI. After adjustment, low serum magnesium remained a significant independent predictor of HOMA-IR ($\beta = -0.35$, $p = 0.002$). The regression model explained approximately 24% of the variance in HOMA-IR, suggesting that serum magnesium plays a clinically relevant role in insulin sensitivity among overweight and obese children. These regression findings are displayed in Table 4.

Table 4: Multivariate linear regression analysis of predictors of insulin resistance (HOMA-IR).

Predictor Variable	β coefficient	Standard Error	p-value
Age (years)	0.08	0.05	0.11
Sex (male vs. female)	-0.06	0.07	0.28
BMI (kg/m²)	0.21	0.06	0.004*
Serum magnesium (mg/dL)	-0.35	0.09	0.002*

*Significant at $p < 0.05$.

Taken together, these results indicate that magnesium deficiency was highly prevalent in the study population and was strongly associated with increased fasting insulin levels and elevated HOMA-IR scores. Even after adjusting for BMI and demographic variables, serum magnesium deficiency remained an independent predictor of insulin resistance, highlighting its potential importance as a modifiable risk factor in obese children.

DISCUSSION

The present study investigated the association between serum magnesium deficiency and insulin resistance in overweight and obese children attending a tertiary care hospital in Pakistan[12]. Our findings demonstrate that nearly half of the participants had magnesium deficiency, and those with lower serum magnesium levels exhibited significantly higher fasting insulin concentrations and HOMA-IR scores compared to children with adequate magnesium status. Even after adjustment for age, sex, and BMI, serum magnesium remained an independent predictor of insulin resistance, underscoring its potential importance as a modifiable biochemical factor in the development of metabolic dysfunction[13].

The prevalence of magnesium deficiency observed in our study (43.3%) is consistent with findings from previous international research. Rodríguez-Morán and Guerrero-Romero reported a similar burden of hypomagnesemia among obese adolescents in Mexico, where magnesium deficiency was strongly associated with impaired glucose metabolism and elevated insulin resistance indices[14]. Likewise, Hruby and colleagues in the Framingham Offspring Study found that higher dietary magnesium intake was associated with a lower risk of developing insulin resistance and type 2 diabetes, reinforcing the notion that magnesium plays a protective metabolic role. Our results extend this evidence to the South Asian pediatric population, which has been underrepresented in previous studies despite its high burden of both obesity and early-onset diabetes[15].

The biological plausibility of the observed association is well supported by mechanistic insights. Magnesium serves as a critical cofactor in insulin receptor autophosphorylation and in the activation of downstream kinases involved in glucose transport. Deficiency of magnesium is known to impair glucose uptake in skeletal muscle and adipose tissue, thereby precipitating a state of peripheral insulin resistance[16]. Furthermore, hypomagnesemia has been linked to chronic low-grade inflammation and oxidative stress, both of which are recognized contributors to metabolic syndrome. Our observation of higher fasting insulin and HOMA-IR in magnesium-deficient children is in line with these pathophysiological mechanisms[17].

An important finding of our study was that serum magnesium remained a significant predictor of insulin resistance even after adjustment for BMI. This suggests that magnesium deficiency is not merely a consequence of obesity but may independently contribute to insulin resistance[18]. Similar conclusions have been drawn from clinical trials where oral magnesium supplementation improved insulin sensitivity in adults with diabetes and in non-diabetic subjects with low magnesium status. In pediatric populations, however, such interventional data are limited. Our study adds evidence that magnesium deficiency may be an early, correctable risk factor for metabolic dysfunction in children, and therefore deserves more attention in clinical practice and nutritional guidelines[19].

The local context of Pakistan makes these findings particularly relevant. Dietary patterns in urban Pakistani children often include high consumption of processed foods, refined carbohydrates, and sugar-sweetened beverages, which are typically poor in magnesium content[20]. Simultaneously, the intake of magnesium-rich foods such as leafy green vegetables, legumes, nuts, and whole grains is relatively low. Coupled with the rising rates of pediatric obesity and sedentary lifestyles, these dietary inadequacies may exacerbate the burden of hypomagnesemia and its metabolic consequences. Early identification of magnesium deficiency in overweight and obese children could provide a low-cost, nutrition-based strategy to reduce the risk of developing type 2 diabetes mellitus and other complications of metabolic syndrome in this vulnerable group[21].

Our study has several strengths. It is among the few to examine serum magnesium and insulin resistance in South Asian children, a high-risk population where data are scarce. The use of standardized methods for anthropometric and biochemical assessment strengthens the validity of our findings[22]. However, certain limitations must also be acknowledged. The cross-sectional design precludes causal inference, meaning we cannot definitively conclude whether magnesium deficiency directly causes insulin resistance or whether it is a consequence of metabolic disturbances. Dietary magnesium intake and urinary magnesium excretion were not assessed, which could have provided further insights into the mechanisms underlying low magnesium levels. Finally, the sample size, although adequate for detecting associations, was limited to a single tertiary care center and may not reflect the wider population of Pakistan[23,24].

Despite these limitations, our findings highlight the potential of serum magnesium as a simple biomarker to identify children at

higher risk of insulin resistance. They also raise the possibility that magnesium supplementation or dietary modification may improve insulin sensitivity in obese children, although longitudinal and interventional studies are required to confirm these hypotheses[25].

CONCLUSION

In conclusion, this study demonstrates that serum magnesium deficiency is highly prevalent among overweight and obese children in Pakistan and is strongly associated with increased fasting insulin levels and higher HOMA-IR scores. Importantly, magnesium deficiency remained an independent predictor of insulin resistance even after adjusting for age, sex, and BMI. These findings underscore the need to incorporate serum magnesium measurement into the routine evaluation of obese children and to consider dietary interventions aimed at improving magnesium intake. Addressing magnesium deficiency at an early stage may represent a cost-effective and practical approach to reducing the long-term burden of type 2 diabetes mellitus and metabolic syndrome in the pediatric population.

Availability of data and materials: The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

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All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

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