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ORIGINAL ARTICLE

Relative Fat Mass, Body Mass Index, and Insulin Resistance in Hemodialysis Children at Zagazig University Children's Hospital

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ABSTRACT:

Background: Children with chronic kidney disease (CKD) are at increased risk of metabolic disturbances, including insulin resistance (IR). Traditional anthropometric measures such as body mass index (BMI) may not accurately reflect true adiposity or metabolic risk in this population. Relative fat mass (RFM) has emerged as a promising alternative for evaluating body composition and metabolic status. This study aimed to investigate the association between insulin resistance and relative fat mass in hemodialysis children and to evaluate the diagnostic value of RFM compared to BMI.

Methods: This cross-sectional study included 80 pediatric patients on regular haemodialysis for at least 6 months before the study, aged 6–18 years, treated at the pediatric nephrology unit of Zagazig University Hospitals. RFM was calculated using height and waist circumference, and participants were stratified into three RFM categories. Insulin resistance was assessed using the homeostasis model assessment of insulin resistance (HOMA-IR).

Results: Higher RFM categories were significantly associated with increased prevalence of insulin resistance ($p = 0.01$), while most demographic and clinical features did not differ by RFM. The area under the ROC curve (AUC) for RFM was 0.803 (95% CI: 0.70–0.89, $p < 0.001$), indicating strong diagnostic accuracy. The optimal cutoff value of RFM (≥33.15%) achieved a sensitivity of 75.6% and specificity of 80%, with a positive predictive value of 82.9% and a negative predictive value of 71.8%, yielding an overall accuracy of 77.5%. In contrast, BMI performed less effectively, with an AUC of 0.62 (95% CI: 0.50–0.73), lower sensitivity (26.7% when using obesity as a cutoff), but comparable specificity (85.7%).

Conclusions: RFM provides a more accurate, non-invasive tool than BMI for identifying insulin resistance in pediatric CKD patients. Incorporating RFM into routine clinical practice may improve risk stratification and support earlier intervention to prevent adverse metabolic and cardiovascular outcomes.

Keywords: Insulin Resistance; Relative Fat Mass; Haemodialysis; Children.

INTRODUCTION

Chronic kidney disease (CKD) in children is frequently associated with metabolic disturbances, including impaired glucose regulation, dyslipidemia, muscle wasting, and increased visceral fat. Insulin resistance (IR) is linked to declining kidney function and heightened cardiovascular risk in this population [1].

Insulin resistance occurs when organs respond poorly to insulin, reducing its effectiveness in regulating blood glucose.

Insulin controls glucose levels by facilitating muscle uptake, inhibiting hepatic glucose production, and decreasing lipolysis in adipose tissue [2]. Early identification and management of IR are critical for improving outcomes in pediatric CKD. Obesity is the primary risk factor for IR in the general population [3]. The homeostasis model assessment (HOMA) is widely used to estimate IR using fasting insulin and glucose measurements [4].

In children with kidney failure, the breakdowns of insulin by non-kidney organs like the liver and muscles are slowed down, making insulin last longer in the blood. Some believe that toxins building up in the body can block the liver from clearing insulin as it should, even though the liver normally removes about half of the insulin produced [5].

Body mass index (BMI) has traditionally been used to assess body fat, but its reliability is increasingly questioned. "Normal weight obesity" (NWO) defined as a high body fat percentage despite a normal BMI has been associated with increased cardiovascular risk [6]. Children with CKD may be especially vulnerable to NWO due to poor nutrition, inactivity, and muscle wasting. However, data on the prevalence and impact of NWO on insulin and glucose metabolism in pediatric CKD are limited [7].

Malnutrition is also a frequent problem for children with CKD, particularly in advanced stages of the disease. In recent years, though, doctors have started to see more cases of overnutrition and unhealthy lifestyles in this group. Because of poor nutrition; whether undernutrition or overnutrition can impact on the overall health and quality of life for these patients, early recognition is critical. Factors like hormone disturbances and acidosis, in addition to poor intake, can make malnutrition worse, leading to weight loss, low albumin, and increased energy needs [8].

While the metabolic complications of CKD have been studied in both adults and children, the relationship between insulin resistance and relative fat mass in children on hemodialysis remains poorly understood. Most research relies on BMI, which may not accurately reflect body fat distribution or its metabolic effects. There is a clear need for studies using precise body composition measures to assess these associations in pediatric CKD patients. So, this study aimed to detect the association between insulin resistance and relative fat mass in children receiving hemodialysis.

METHODS

This cross-sectional study was carried out at the pediatric nephrology unit in Zagazig University Hospitals. The research spanned from March 2024 to December 2024, covering a total period of about ten months after obtaining approval from the Institutional Review Board (IRB#11395/8-1-2024) and written informed consent from participants or their guardians. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research. The collected data was then analysed and reviewed.

All patients who fulfilled the inclusion and exclusion criteria during the study period were enrolled, resulting in a comprehensive sample of 80 children. Females represented 56.2% of the group. The sample included 80 patients, of whom 45 were females and 35 were males. The age of participants ranged between 6 and 18 years, with a mean age of 12.06 years.

We included children aged 6 to 18 years, On regular hemodialysis for at least 6 months before the start of the study. who had been on regular hemodialysis for at least six months before enrolment. Children who had diabetes mellitus, had missing data, declined

participation (either the child or their guardians), or had previously undergone kidney transplantation were excluded from the study.

All enrolled patients went through a full clinical assessment, starting with a thorough medical history and complete physical examination. Body mass index (BMI) was calculated for each participant using the standard formula (weight in kilograms divided by height in meters squared) [9]. Relative fat mass (RFM) was also determined for every child, using the equation:

RFM = 74 - (22 × height/waist circumference) + (5 × sex), where sex equals zero for boys and one for girls. The RFM values were divided into three groups (tertiles): Q1 was less than or equal to 30.14, Q2 ranged from 30.14 to 38.93, and Q3 was above 38.93 [10]. Waist circumference was measured using a tape placed over the clothing, and height was measured with a vertical gauge, while each child stood barefoot and upright with their head in a neutral position. All measurements were recorded in centimeters or meters as appropriate [11].

Venous blood samples (3 ml) were taken from each child and separated into two portions. One milliliter was used for a complete blood count (CBC), while the remaining two milliliters were allowed to clot and then centrifuged to separate serum for further tests. Laboratory investigations included fasting blood glucose, insulin levels, liver function, kidney function, and electrolyte analysis.

Insulin resistance was estimated using the HOMA-IR equation: **Serum insulin (µU/mL) × fasting glucose (mg/dL) / 405**. Fasting blood samples were collected after at least eight hours without food. Insulin was measured using a solid-phase, enzyme-labeled chemiluminescent immunometric assay (IMMULITE 2000 XPi, Siemens Healthineers Global). The cutoff for high plasma glucose was 100 mg/dL, and HOMA-IR was used as the main index for insulin resistance [12]. Other lab tests included kidney and liver function tests, CBC (measured by automated cell counter, model XN 2000, Sysmex, Japan), serum albumin, C-reactive protein (CRP) was measured with a COBAS c501 auto-analyzer.

Statistical Methods

Data were analyzed using IBM SPSS version 20.0 (IBM Corp., Armonk, NY). Qualitative data were expressed as numbers and percentages, while quantitative data were described using range, mean, standard deviation, and median after assessing normality with the Kolmogorov-Smirnov test. Statistical comparisons included the chi-square test for categorical variables, the Mann-Whitney test for non-normally distributed quantitative variables, and the two-sample t-test for comparing means between independent groups. Results were considered statistically significant at $p < 0.05$.

RESULTS

The sample included 80 patients with 45 of them were females and 35 males with a mean age of 12.09 ± 3.52 years (ranging from 6 to 18 years). BMI classification showed that 21.3% of patients were underweight, 40% had a normal BMI, 22.5% were overweight, and 16.3% were obese, with a mean BMI of 18.66 ± 4.95 kg/m². The median dry weight was 24.4 kg (IQR: 16.63–39), spanning from 8 to 60 kg. Regarding relative fat mass, 35% of patients had ≤30.14%, 42.5% had between >30.14% and 38.93%, and 22.5% had >38.93%, with an overall mean of 33.84 ± 7.14% (Table 1). Mean fasting blood sugar was 88.34 mg/dl. Median insulin was 11.9. median HOMA-IR was 2.61 with 56.3% of patients had HOMA-IR≥2.5. Mean hemoglobin was 10 g/dl. Mean WBCs was 7.32 (10^3/mm^3).

Median platelet count was 227.5 (10^3/mm^3). Mean serum albumin, creatinine, BUN and uric acid were 4.06 g/dl, 6.74 mg/dl, 50.57 mg/dl and 5.92 mg/dl. Mean serum sodium, potassium, magnesium and calcium were 134.86 mEq/L, 4.94 mg/dl, 3.06 mg/dl and 9.06 mg/d

CRP 1.9 mg/dl. (Table 2). There is statistically non-significant relation between relative fat mass and age, gender, etiology, serum albumin, creatinine and hemoglobin. (Table 3).

Significant associations were revealed between RFM groups and BMI categories ($p < 0.001$), with obesity being most prevalent in the high RFM group (72.2%) and underweight status more common in the low RFM group (39.3%), insulin resistance (HOMA-IR ≥2.5) was significantly more common in intermediate (64.7%) and high RFM groups (72.2%) compared to the low RFM group. (Table 4).

A statistically significant relationship was revealed between insulin resistance and body mass index categories ($p = 0.013$), with insulin resistance being more commonly associated with overweight and obesity (Table5).

Being overweight and obese was able to predict insulin resistance with sensitivity 55.6%, specificity 82.9%, positive predictive value 80.7%, negative predictive value 59.2%, and overall accuracy 67.5% ($p<0.001$). Being obese was able to predict insulin resistance with sensitivity 26.7%, specifically 85.7%, positive predictive value 70.6%, negative predictive value 47.6%, and overall accuracy 52.5% ($p<0.001$) (Table 6).

The best cutoff of relative fat mass in prediction of insulin resistance is ≥33.15% with area under curve 0.803 with sensitivity 75.6%, specificity 80%, positive predictive value 82.9%, negative predictive value 71.8%, and overall accuracy 77.5% ($p<0.001$) (Figure 1).

Table 1: Demographic, Anthropometric, and Relative Fat Mass Characteristics of the Studied Patients (N = 80)

Parameter	Value/Category	n	%	Mean ± SD	Median (IQR)	Range
Gender	**Male**	**35**	**43.8%**			
	Female	**45**	**56.2%**			
Age (years)				**12.09 ± 3.52**		**6 – 18**
Height (cm)				**124.81 ± 20.66**		**64 – 170**
Body Mass Index (BMI, kg/m²)	**Low**	**17**	**21.3%**	**18.66 ± 4.95**		**11.7 – 40.2**
	Normal	**32**	**40%**			
	Overweight	**18**	**22.5%**			
	Obese	**13**	**16.3%**			
Waist Circumference (cm)				**67.15 ± 11.81**		**30 – 103**
Dry Weight (kg)					**24.4 (16.63–39)**	**8 – 60**
Relative Fat Mass (%)	**≤30.14%**	**28**	**35%**	**33.84 ± 7.14**		**19.56–46.2**
	>30.14–38.93%	**34**	**42.5%**			
	>38.93%	**18**	**22.5%**			

SD: Standard deviation; IQR: Interquartile range; BMI: Body mass index.

Table 2: Glycemic Profile and Laboratory Data of the Studied Patients (N = 80)

Parameter	Value/Category	n	%	Mean ± SD	Median (IQR)	Range
Fasting Blood Sugar (FBS, mg/dl)	Elevated blood glucose	**11**	**13.8%**	**88.34 ± 13.47**		**63 – 131**
Insulin (µIU/mL)					**11.9 (7.31–16)**	**2.79 – 34.2**
HOMA-IR	<2.5	**35**	**43.8%**		**2.61(1.789–3.543)**	**0.578–20.071**
	≥2.5	**45**	**56.3%**			
Hemoglobin (g/dl)				**10.0 ± 1.42**		**7.7 – 14.2**
WBCs (10^3/mm^3)				**7.32 ± 2.42**		**2.6 – 15.4**
Platelet count (10^3/mm^3)					**227.5(102.75–301.25)**	**130 – 469**
Albumin (g/dl)				**4.06 ± 0.43**		**2.91 – 5.4**
Creatinine (mg/dl)				**6.74 ± 2.32**		**1.8 – 11.8**
BUN (mg/dl)				**50.57 ± 15.27**		**25 – 106**
CRP (mg/L)					**1.9 (0.86–10.96)**	**0.3 – 91**

SD: Standard deviation; IQR: Interquartile range; FBS: Fasting blood sugar; HOMA-IR: Homeostatic model assessment of insulin resistance; WBCs: White blood cells; BUN: Blood urea nitrogen; CRP: C-reactive protein.

Table 3: Relation between relative fat mass, base line and laboratory data

	Low N=28 (%)	**Intermediate N=34 (%)**	**High N=18 (%)**	**F**	**p**
	Mean ± SD	**Mean ± SD**	**Mean ± SD**		
Age (years)	**12.32 ± 3.64**	**12.06 ±3.66**	**11.81 ± 3.2**	**0.118**	**0.889**
Gender				χ^2	
Female	**12 (42.9%)**	**17 (50%)**	**6 (33.3%)**	**0.237**	**0.627**
Male	**16 (57.1%)**	**17 (50%)**	**12 (66.7%)**		
Etiology					
Renal	**19 (67.9%)**	**17 (50%)**	**8 (44.4%)**		
Post-renal	**7 (25%)**	**9 (26.5%)**	**5 (27.8%)**	**4.603**	**0.551**
Unexplained	**2 (7.1%)**	**8 (23.5%)**	**5 (27.8%)**		
Albumin (g/dl)	**4.07 ± 0.43**	**4.07 ± 0.39**	**4.03 ± 0.52**	**0.057**	**0.945**

Table 4: Relation Between Relative Fat Mass Groups, Body Mass Index, Anthropometric Measures, Glycemic Profile, and Insulin Resistance in Studied Patients (N = 80)

	Low RFM N=28 (%)	Intermediate RFM N=34 (%)	High RFM N=18 (%)	χ^2 / F / KW	p	Pairwise (p)
BMI Category				**51.805**	**<0.001****	
- Low	**11 (39.3%)**	**4 (11.8%)**	**2 (11.1%)**			**P1 <0.0001****
- Average	**16 (57.1%)**	**14 (41.2%)**	**2 (11.1%)**			**P2 0.002***
- Overweight	**0 (0%)**	**12 (35.3%)**	**1 (5.6%)**			**P3 <0.001****
- Obese	**1 (3.6%)**	**4 (11.8%)**	**13 (72.2%)**			
Dry weight (kg)	**29.33 ± 11.37**	**27.65 ± 11.39**	**32.22 ± 14.27**	**0.844 (F)**	**0.434**	
Waist circumference (cm)	**65.29 ± 10.88**	**65.71 ± 10.27**	**72.78 ± 14.55**	**2.766 (F)**	**0.069**	
Hyperglycemia				**0.491**	**0.483**	
- Absent	**24 (85.7%)**	**28 (82.4%)**	**17 (94.4%)**			
- Present	**4 (14.3%)**	**6 (17.6%)**	**1 (5.6%)**			
HOMA-IR				**6.671**	**0.01***	
- Normal	**17 (64.3%)**	**12 (35.3%)**	**5 (27.8%)**			
- Resistance	**10 (35.7%)**	**22 (64.7%)**	**13 (72.2%)**			
FBG (mg/dl)	**86.14 ± 14.01**	**88.74 ± 15.62**	**91.0 ± 6.44**	**0.734 (F)**	**0.4832**	
Insulin (µIU/mL)	**9.78 (7.02–16)**	**12.7(7.71–15.93)**	**13.2 (7.83–16.2)**	**1.799 (KW)**	**0.407**	
HOMA-IR (Median, IQR)	**1.91 (1.35–2.62)**	**2.67 (2.19–3.74)**	**3.09 (2.36–5.43)**	**10.887 (KW)**	**0.004***	**P1 0.018, *P2 0.239, P3 0.002***
Creatinine (mg/dl)	**7.06 ± 2.52**	**6.64 ± 2.3**	**6.42 ± 2.1**	**0.467**	**0.628**	
Hemoglobin (g/dl)	**10.07 ± 1.5**	**9.86 ± 1.3**	**10.17 ± 1.54**	**0.332**	**0.719**	

χ^2Chi square test F One way ANOVA test.RFM: Relative fat mass; BMI: Body mass index; SD: Standard deviation; IQR: Interquartile range; FBG: Fasting blood glucose; HOMA-IR: Homeostatic model assessment of insulin resistance. χ^2: Chi-square test; F: One-way ANOVA test; KW: Kruskal-Wallis test; **p≤0.001 highly significant; *p<0.05 significant. P1: low vs intermediate RFM; P2: intermediate vs high RFM; P3: low vs high RFM.

Table 5: Relation between insulin resistance and body mass index

	Normal	Insulin resistance	χ^2	p
	n=35 (%)	N=45 (%)		
BMI				
Low	**9 (25.7%)**	**8 (17.8%)**		
Average	**20 (57.1%)**	**12 (26.7%)**	**6.167**	**0.013***
Overweight	**1 (2.9%)**	**13 (28.9%)**		
Obese	**5 (14.3%)**	**12 (26.7%)**		

χ2Chi square for trend test *p<0.05 is statistically significant.

Table 6: Performance of BMI in prediction of insulin resistance

Score	Sensitivity	Specificity	PPV	NPV	Accuracy	p
Obese +overweight	55.6%	82.9%	80.7%	59.2%	67.5%	<0.001**
Obese	26.7%	85.7%	70.6%	47.6%	52.5%	0.179

AUC: area under curve, PPV: positive predictive value, NPV: negative predictive value , **p≤0.001 is statistically highly significant.

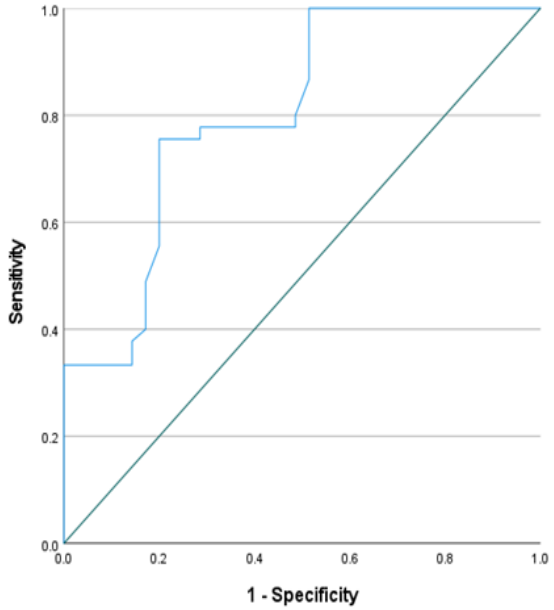

Figure 1: ROC curve showing performance of relative fat mass in prediction of insulin resistance

DISCUSSION

A growing body of evidence highlights the intricate interplay between insulin resistance and body composition in children with chronic kidney disease, underlining the considerable metabolic burden faced by this vulnerable group. The current study findings build on previous research and provide further clarity regarding how relative fat mass could be related to metabolic health and insulin sensitivity in pediatric CKD.

In current study Mean BMI was 17.997 (kg/m2). 40% had average BMI, 21.3% had low BMI, 22.5% were overweight and16.3% were obese. This meant that about 40% of our cases had elevated BMI and this was in accordance with Kogon et al. [13] who

confirmed the correlation between higher BMI with kidney function decline. Obesity could lead to CKD directly through proceeding as an independent risk factor and indirectly through its association and being risk factor for hypertension, diabetes, hypertension, as well as atherosclerosis, which is group of well-established independent risk factors for CKD [13].

In current study, distribution of patients according to RFM was 35% of cases ≤30.14% (mild RFM), 42.5% of cases >30.14 – 38.93% (moderate RFM) and 22.5% of cases >38.93% (severe RFM). This was in accordance with Karava et al. [14] declared that elevated RFM level is prevalent in CKD patients.

The current study showed that fasting blood sugar was 88.34 mg/dl. Median insulin was 11.9. median HOMA-IR was 2.54 with 56.3% of patients had HOMA-IR≥2.5. This means that IR is highly encountered in patients with CKD. Similar results were reported by Thomas et al. [15] who declared that IR is prevalent among cases who had end-stage renal disease and the understanding of insulin resistance mechanisms may lead to better therapeutic strategies which could improve the metabolism among CKD patients.

The current study showed that that higher RFM was significantly associated with increased insulin resistance, as measured by HOMA-IR, regardless of age, sex, or disease etiology among our participants. These findings are in line with the results of those of Karava et al. [14], who reported that RFM is a more sensitive marker for early metabolic risk in pediatric CKD than traditional BMI. Their research further emphasized the limitation of relying solely on BMI to detect early metabolic alterations, as it may fail to identify children with excess fat despite having a normal weight.

Our findings are consistent with Foster et al. [16] observed that differences in RFM among children with CKD are mainly related to the disease process itself, rather than demographic characteristics, which aligns with our data showing comparable distributions of age and sex across all RFM categories.

Contrastingly, some earlier studies such as Han et al. [17] and Schmidt et al. [18] revealed that demographic factors like age and gender may affect body composition and kidney development in children, with potential impacts on outcomes. Han et al. [17] demonstrated age- and gender-related variations in body composition, while Schmidt et al. [18] identified gender and body mass as significant predictors of kidney size. Despite these observations, the current study did not detect any significant demographic effects on RFM categories, reinforcing the concept that CKD-related factors exert a more pronounced influence on fat mass distribution than age or gender in this population. This finding is important, as it suggests that risk stratification for metabolic complications in pediatric CKD should prioritize clinical and anthropometric assessment over demographic variables alone.

When we assessed the relationship between RFM and BMI, we found a strong and statistically significant association: children in the higher RFM categories were much more likely to fall into overweight or obese BMI groups. However, this overlap was not complete, reflecting the limitations of BMI in accurately capturing adiposity, especially in children with altered muscle mass or those presenting with normal obesity. In clinical terms, BMI alone may underestimate true fat burden in CKD, missing patients who are at risk for metabolic disturbances. This agreed with findings by Sgambat et al. [19], who demonstrated that RFM provides a more nuanced and sensitive measure of body

fatness than BMI or waist circumference, particularly in pediatric CKD.

Likewise, Patel et al. [20] showed that BMI and waist circumference, while correlated with obesity, do not adequately distinguish between lean and fat mass in children with kidney disease. The clinical implication is that RFM is a valuable adjunct to traditional anthropometric measures in routine assessment and should be considered in risk prediction models for pediatric CKD [20].

In the current study other anthropometric indicators such as dry weight and waist circumference did not differ significantly across RFM categories, though waist circumference was higher in those with greater RFM. This observation supports the belief that visceral fat may play a central role in metabolic dysregulation in CKD, as described by Patel et al. [20], but also emphasizes that direct body composition analysis remains superior to single measures like waist circumference or weight alone. The inability of these markers to reliably separate patients by metabolic risk highlights the need for comprehensive assessments in pediatric nephrology clinics.

The current study findings showed that most children maintained normal fasting glucose values regardless of RFM category, yet the prevalence of insulin resistance increased markedly with higher RFM. This suggests that abnormal fat accumulation can impair insulin sensitivity before overt disturbances in glycemic control emerge. These results reinforce the findings of Karava et al. [14], Trirogoff et al. [21], and Yang et al. [22], all of whom found that fat mass rather than BMI or glucose levels served as a primary driver of insulin resistance among children with CKD.

Trirogoff et al. [21] revealed that both BMI and fat mass are important correlates of insulin resistance in non-diabetic CKD, suggesting that metabolic dysregulation often predates hyperglycemia in these patients. Yang et al. [22] similarly showed that increased fat mass significantly contributes to the development of insulin resistance, again supporting the utility of fat mass measurement over traditional markers.

In our cohort, RFM outperformed BMI in predicting insulin resistance, with a higher AUC (0.803 vs. 0.62) and a better balance of sensitivity and specificity. Importantly, the ROC-derived cutoff of RFM (≥33.15%) captured most children with metabolic risk who would have been missed using BMI alone. The superior diagnostic accuracy of RFM suggests it could be integrated into clinical screening algorithms for pediatric CKD patients, providing earlier detection and intervention opportunities. Karava et al. [14] provided similar evidence that higher fat percentages are strongly correlated with insulin resistance, even among children with a normal BMI. Trirogoffet al. [22] confirmed that for incorporating fat mass measurements into routine nephrology guidelines, arguing that such measures capture metabolic risk more effectively than BMI. The high positive predictive value observed in our study supports the notion that RFM can serve as a valuable clinical tool for screening and risk stratification in pediatric CKD.

When we evaluated BMI as a predictor for insulin resistance, the current study revealed that BMI has only moderate sensitivity, especially when obesity alone is used as the cut-off, although specificity remained high. Combining overweight and obese categories improved sensitivity and overall accuracy. Rodig et al. [23] observed a similar trend, noting that the transition from overweight to obesity in CKD children is associated with worsening metabolic profiles. Patel et al. [20] and Karava et al. [14] also demonstrated that BMI alone cannot reliably identify all children at risk for metabolic disturbance, particularly those with normal weight obesity. The inability of BMI to

account for visceral or hidden fat mass limits its use as a sole screening method. The lower negative predictive value of BMI observed in our data suggests that many children with significant fat accumulation and metabolic risk would be missed if BMI were the only criterion used.

A particularly important aspect of our results is the strong association between increased BMI and insulin resistance. Our findings are in line with the established link between excess adiposity and impaired insulin sensitivity in pediatric CKD, as described by Karava et al. [14] and Lai et al. [24]. These studies previously reported that higher BMI and increased fat mass were associated with elevated insulin levels and greater cardiovascular risk in children with CKD. Savino et al. [25] also revealed the same findings by demonstrating that excess weight contributes to CKD progression through its cumulative metabolic effects, underscoring the importance of regular monitoring of adiposity and metabolic parameters in routine care.

A key strength of this study lies in its comprehensive assessment of body composition through relative fat mass, providing a more accurate evaluation of metabolic risk than traditional anthropometric measures in pediatric CKD. The standardized methodology and adequate sample size further support the strength of the findings. Nevertheless, the cross-sectional design limits causal interpretation, and the single-center setting may restrict generalizability. The absence of a healthy control group and longitudinal follow-up are additional limitations to consider.

CONCLUSION

The RFM represents a superior, non-invasive metric for early detection of insulin resistance and hyperuricemia in pediatric CKD patients, offering advantages over BMI alone. Incorporating RFM into routine clinical assessments may improve risk stratification and support earlier interventions aimed at mitigating long-term metabolic and cardiovascular complications.

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Availability of Data: The datasets used or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contribution: S.M.I.R. conceptualized the study, led the research design, and was primarily responsible for data acquisition and manuscript drafting. E.K.A. provided critical revisions, oversight, and expert guidance throughout all stages of the project. R.A.A.A. coordinated manuscript preparation and correspondence, contributed substantially to the anatomical and embryological framework, and ensured the integrity of the final submission. H.E.M.S. was chiefly responsible for all clinical pathological aspects, including data analysis and interpretation in this domain. A.G. supported patient recruitment, clinical data collection, and contributed to the final review of the manuscript with approval of all authors for this final version.

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