

Estimation of Iron Deficiency Anemia among Obese & Overweight Children in Benha University Hospital

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Abstract:

Background: Iron deficiency (ID) is a widespread nutritional issue that affects children and teenagers across the globe. Recent insights from the Global Burden of Disease report reveal that ID anemia is the only nonfatal condition listed among the top 10 causes of disability-adjusted life years for this age group. This study aimed to assess screen and determine the frequency and relation between overweight (OW), obesity and ID among young children in Benha University Hospital. **Methods:** This cross sectional and case control study included 100 obese and OW children. All studied cases were subjected to Lab measurements [Complete blood count, Iron profile, kidney and liver function test]. Anthropometric measurements: Weight, Height, The body mass index (BMI). **Results:** Receiver operating characteristic (ROC) analysis was done for BMI to predict ID anemia. It revealed that the Area Under Curve (AUC) was 0.766, with a 95% confidence interval (CI) of 0.660 - 0.871. The best cutoff point was > 28.2, at which sensitivity, specificity, Positive Predictive Value (PPV), and Negative Predictive Value (NPV) were 75.8%, 60%, 44%, and 85.7%, respectively. A multivariate logistic regression analysis was done to predict ID anemia. The model revealed that obesity was associated with 5 times increased risk of ID anemia (Odds Ratio (OR) =5.007, 95% CI=1.801 - 13.923, P=0.002), controlling for age and gender. **Conclusion:** IDA is prevalent among OW and obese children, with obesity and female gender identified as significant risk factors. These findings highlight the need for early screening and targeted interventions to address this dual burden of malnutrition in pediatric populations.

Keywords: Iron Deficiency Anemia; Obese; Overweight; Children.

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Introduction

Iron deficiency (ID) is a widespread nutritional issue that affects children and teenagers across the globe. Recent insights from the Global Burden of Disease report reveal that ID anemia is the only nonfatal condition listed among the top 10 causes of disability-adjusted life years for this age group.⁽¹⁾ With a global age-standardized prevalence rate of 25.1%, the disparity between developed and developing nations is minimal⁽²⁾. ID continues to be a prominent public health concern, impacting both economically advanced and underdeveloped regions alike⁽³⁾.

Remarkably, the link between ID and nutritional deficiency has been upended by emerging evidence suggesting that overweight (OW) individuals may also be vulnerable to ID, challenging the prevailing understanding. Traditionally, ID has been closely associated with poor nutrition, especially in developing nations where it correlates with diminished socioeconomic status⁽³⁾.

However, recent studies point to an unexpected convergence of these two health issues, highlighting the complexity of malnutrition in today's world. Moreover, the rising incidence of OW and obesity is no longer confined to urban centers but is also increasingly evident in rural areas. A recent study in Tala District, Menoufiya Governorate, Egypt, exhibited a striking rise in obesity rates among preschool children (2-5 years), with 21% of children categorized as OW or obese⁽⁴⁾.

This troubling trend reveals that nearly one in five children under the age of five in this region are struggling with weight-related health challenges. Although undernutrition remains a significant issue in low- and middle-income countries, the worldwide prevalence of OW and obesity is steadily increasing⁽⁵⁾.

This upward trajectory is largely driven by rapid shifts in the global food landscape, with the proliferation of inexpensive, ultra-processed foods and a marked decline in physical activity⁽⁶⁾.

These factors significantly contribute to the rising prevalence of the double burden of malnutrition, a complex issue recognized by the World Health Organization (WHO). This phenomenon refers to the simultaneous existence of undernutrition alongside obesity or diet-related noncommunicable diseases within individuals, families, and broader communities. As both conditions coexist, they pose unique challenges to public health, creating a paradox where communities face both nutrient deficiencies and the growing threat of chronic diseases linked to poor dietary patterns⁽⁷⁾.

The WHO's concept of double burden of malnutrition extends across various titres, individual (e.g., concurrent obesity and nutritional anemia), household (e.g., maternal nutritional anemia and child obesity), and population (e.g., regions where both undernutrition and obesity co-exist). While Double Burden of Malnutrition (DBM) presents a global challenge, it disproportionately affects Low- and Middle-Income Countries (LMICs), particularly those undergoing rapid transitions in dietary patterns⁽⁶⁾.

This research aims to explore and quantify the prevalence of ID, OW, and obesity in young children at Benha University Hospital, examining their interrelationship and implications for public health strategies.

Patients and methods:

Patients:

This combination of cross sectional and case control study was done on 100 children aged 1 to 10 years, diagnosed with obesity or OW, at Benha University Hospital and its outpatient clinics. The research unfolded over a 10-month period, from January 2024 to October 2024. Ethical approval was secured from the Research Ethics Committee of Benha Faculty of Medicine, and written informed consent was obtained from all parents prior to their children's participation,

ensuring ethical transparency and participant safety throughout the research.

Approval code: MS 20-12-2023

Inclusion criteria were age 1-10 years of age, children from the outpatient health care units of Banha University Hospital and both sexes diagnosed with obesity or OW according to the body mass index (BMI) ⁽⁸⁾.

Exclusion criteria were children more than 10 years old or less than 1 year, with inborn errors of metabolism, with chronic diseases and parents who refused to share in the research.

Sample size calculation

The sample size was calculated using Epi-info software version 7.2.5.0 based on a previous study by Hamiel et al., which reported a prevalence of iron deficiency anemia of 38.8% in overweight and obese children. The total sample size needed to detect such a prevalence was determined to be 91 children, and to increase precision, the sample was increased to 100 children. Confidence level and margin of error were adjusted at 95% and 10%, respectively ⁽⁸⁾.

Methods:

All studied cases were subjected to the following: Lab measurements, including [Complete blood count for selected cases, Iron profile (Serum Iron, Serum Ferritin & Total Iron-Binding Capacity (TIBC).), kidney and liver function test].

Anthropometric measurements: Weight, Height, the body mass index (BMI).

Lab measurements: 5 ml of venous blood was withdrawn under complete aseptic conditions, samples were divided into 2 parts: 1st part: 2 ml was added into EDTA vacutainer tube and used for Complete Blood Count (CBC) and 2nd part was added into plain vacutainer tube and then left for 15-20 minutes till clotting, centrifuged at 1500 rpm for 15 minutes. The resultant serum will be separated and used for subsequent analysis of (kidney and liver function tests and iron profile).

The body mass index (BMI): was estimated by the formula $BMI = \frac{Wt.}{height^2}$ in meters ⁽⁹⁾.

- BMI of $\geq 97^{th}$ percentile for age and gender or BMI of > 30 (whichever is smaller) considered obese.
- BMI of $\geq 90^{th}$ percentile but $< 97^{th}$ percentile for age and gender or BMI of > 25 considered OW.

The anemia cut-offs of Hb (gm/L):

- **Children 6 - 59 months of age:** mild (100:109), moderate (70:99) and severe (lower than 70)
- **Children 5 - 11 years of age:** mild (110:114), moderate (80:109) and severe (lower than 80)

Serum Ferritin cut-offs in Children (ug/L):

- **Serum Ferritin < 12 :** diagnostic of iron deficiency
- **Serum Ferritin 12 - 20:** possible iron deficiency

Transferrin cut-offs:

- **Transferrin saturations:** of less than 20% indicate iron deficiency
- **Male:** 20-50%
- **Female:** 15-50%

The assessment of the children's growth was conducted via the WHO child growth standards, employing the Z-score system for classification. A Z-score above +2 was used to identify children who were OW, reflecting a weight that exceeded the typical range for their age group. Those with a Z-score above +3 were classified as obese, indicating a more significant deviation from the expected weight range. In contrast, children whose height-for-age Z-score was below -2 were categorized as stunted, indicating a deficiency in growth. Additionally, children whose weight-for-age (W/A) and weight-for-height (W/H) Z-scores were less than -2 were classified as underweight, pointing to a deficiency in weight relative to their age and/or height. This method of classification offers valuable insights into the growth patterns and nutritional status of children, enabling the early identification of potential health

concerns and the implementation of targeted interventions⁽¹⁰⁾.

Statistical analysis

The data were analyzed via SPSS version 28 (IBM, Armonk, New York, USA). For continuous variables, the mean and standard deviation (SD) were calculated to summarize the data, and intergroup comparisons were performed via the unpaired Student's t-test and ANOVA (F) test, based on the nature of the data. Categorical variables were represented as frequencies and percentages, and relationships between these variables were examined via either the Chi-square test or Fisher's exact test, depending on the data characteristics. Statistical significance was determined by a two-tailed P-value of less than 0.05, which was considered indicative of a meaningful difference between the groups.

Results:

A significant association was exhibited between gender and ID anemia, with an elevated proportion of males in the non-deficient group as opposed to the iron-deficient one ($P=0.017$). The iron-deficient group exhibited significantly elevated titres of BMI, serum urea, and serum creatinine as opposed to the non-deficient one ($zP<0.05$). Additionally, the distribution of BMI categories exhibited a significantly larger proportion of the iron-deficient group classified as obese as opposed to the non-deficient one ($P = 0.001, 0.867, 0.438, 0.372, < 0.001$) respectively. **Table 1**

Table 2 illustrates a moderate negative correlation between BMI and Mean Corpuscular Hemoglobin (MCH) ($r=-0.310, P=0.002$). Additionally, BMI exhibited significant negative correlations with serum iron ($r=-0.236, P=0.019$) and transferrin saturation ($r=-0.239, P=0.017$).

Strong positive correlations were exhibited between BMI and Alanine Aminotransferase (ALT) ($r=0.455, P<0.001$), Aspartate Aminotransferase (AST) ($r=0.421, P<0.001$), and serum creatinine ($r=0.491, P<0.001$), while serum urea also exhibited a positive correlation with BMI ($r=0.205, P=0.042$). Other variables, such as Hemoglobin (Hgb), Mean Corpuscular Volume (MCV), serum ferritin, and TIBC, did not exhibit any significant correlation with BMI.

Table 3 shows the ROC analysis conducted to predict ID anemia via BMI. The analysis revealed an AUC of 0.766, with a 95% confidence interval of 0.660 - 0.871. The optimal BMI cutoff point was determined to be >28.2 , where sensitivity, specificity, PPV, and NPV were 75.8%, 60%, 44%, and 85.7%, respectively.

Figure 1 depicts the multivariate logistic regression analysis aimed at predicting ID anemia. The model revealed that a one-unit increase in BMI was associated with a 55% heightened risk of ID anemia ($OR=1.559, 95\% CI=1.261 - 1.927, P<0.001$), after controlling for age and gender. Additionally, female gender was linked to nearly a threefold increased risk of ID anemia ($OR=3.204, 95\% CI=1.102 - 9.312, P=0.032$).

Table 5 depicts the observations of another multivariate logistic regression analysis examining the relationship between obesity and ID anemia. Obesity exhibited a fivefold increase in the risk of developing ID anemia ($OR=5.007, 95\% CI=1.801 - 13.923, P=0.002$), after controlling for age and gender. Female gender was again associated with a threefold increased risk of ID anemia ($OR=2.894, 95\% CI=1.089 - 7.695, P=0.033$).

Table 1: Demographic characteristics, CBC findings, Iron profile, kidney and liver functions of the studied cases

Demographics		
Age (Years)	Median (range)	6 (1 - 10)
Gender		
Males	n (%)	69 (69)
Females	n (%)	31 (31)
Weight (Kg)	Median (range)	39 (14 - 96)
Height (cm)	Mean $\pm$ SD	118 $\pm$ 24
BMI	Mean $\pm$ SD	28.75 $\pm$ 2.95
BMI categories		
Overweight	n (%)	50 (50)
Obese	n (%)	50 (50)
CBC findings		
Hgb (g/dl)		11.24 $\pm$ 1.29
MCV (fl)		76.11 $\pm$ 7.51
MCH (pg)		25.28 $\pm$ 2.77
Iron profile		
Serum Iron (ug/dl)	Median (range)	58 (13 - 137)
Serum ferritin (ng/ml)	Median (range)	30 (2.9 - 103)
TIBC (mg/dl)	Mean $\pm$ SD	334 $\pm$ 51
Transferrin Saturation	Median (range)	18.4 (5.4 - 41.5)
Iron deficiency anemia	n (%)	29 (29)
kidney and liver functions		
Serum urea (mg/dl)	Mean $\pm$ SD	25.8 $\pm$ 7.3
Serum creatinine (mg/dl)	Mean $\pm$ SD	0.77 $\pm$ 0.19
ALT (U/L)	Median (range)	25 (7 - 58)
AST (U/L)	Mean $\pm$ SD	33 $\pm$ 7

BMI: Body mass index; n: Number; Kg: Kilogram; cm: Centimeter. g/dl: Grams per deciliter; MCV: Mean corpuscular volume; fl: Femtoliter; MCH: Mean corpuscular Hgb; pg: Picogram, ALT: Alanine aminotransferase; U/L: Units per liter; AST: Aspartate aminotransferase.

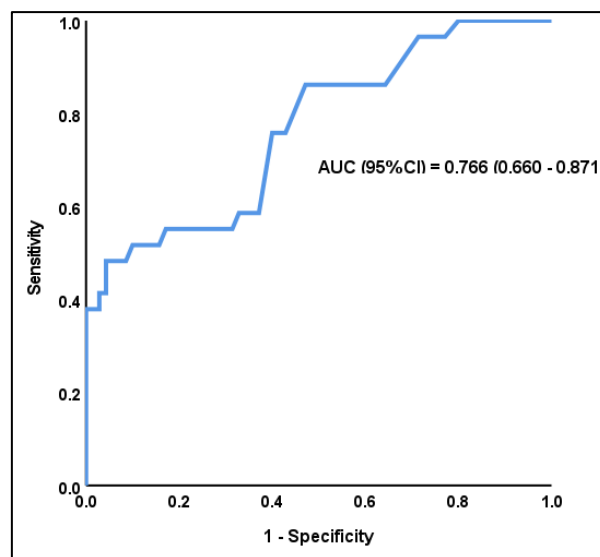
**Figure 1:** ROC analysis of BMI to predict iron deficiency anemia

Table 2: Demographic characteristics, CBC findings and Kidney and liver functions according to iron deficiency anemia

		Iron def anemia		P-value
		Yes (n=29)	No (n=71)	
Age (years)	Median (range)	6 (1 - 10)	6 (1.2 - 10)	0.867
Gender				
Males	n (%)	15 (51.7)	54 (76.1)	0.017*
Females	n (%)	14 (48.3)	17 (23.9)	
BMI	Mean $\pm$ SD	30.83 $\pm$ 3.22	27.89 $\pm$ 2.37	<0.001*
BMI category				
Overweight	n (%)	7 (24.1)	42 (60)	0.001*
Obese	n (%)	22 (75.9)	28 (40)	
CBC findings				
Hgb (g/dl)	Mean $\pm$ SD	9.85 $\pm$ 1.49	11.81 $\pm$ 0.56	<0.001*
MCV (fl)	Mean $\pm$ SD	68.11 $\pm$ 7.51	79.38 $\pm$ 4.45	<0.001*
MCH (pg)	Mean $\pm$ SD	22.06 $\pm$ 2.54	26.59 $\pm$ 1.5	<0.001*
Kidney and liver functions				
Serum Urea (mg/dl)	Median (range)	28.1 $\pm$ 8.2	24.8 $\pm$ 6.7	0.042*
Serum creatinine (mg/dl)	Mean $\pm$ SD	0.84 $\pm$ 0.2	0.75 $\pm$ 0.17	0.021*
ALT (U/L)	Median (range)	27 (11 - 38)	23 (7 - 58)	0.438
AST (U/L)	Mean $\pm$ SD	34 $\pm$ 8	32 $\pm$ 7	0.372

*Significant P-value; n: Number; %: Percentage; BMI: Body mass index, /dL: Grams per deciliter; SD: Standard deviation; MCV: Mean corpuscular volume; fL: Femtoliter; MCH: Mean corpuscular Hgb; pg: Picogram, ALT: Alanine aminotransferase; U/L: Units per liter; AST: Aspartate aminotransferase.

Table 3: Correlation between BMI and other parameters

	BMI	
	r	P
Hgb (g/dl)	-0.182	0.072
MCV (fl)	-0.161	0.112
MCH (pg)	-.310	0.002*
Serum iron	-0.236	0.019
Serum ferritin	-0.152	0.133
TIBC (mcg/dl)	0.178	0.078
Transferrin saturation	-0.239	0.017*
ALT	0.455	<0.001*
Serum urea (mg/dl)	.205	0.042*
Serum creatinine (mg/dl)	.491	<0.001*
AST (UIL)	.421	<0.001*

g/dL: Grams per deciliter; MCV: Mean corpuscular volume; fL: Femtoliter; MCH: Mean corpuscular Hgb; pg: Picogram; TIBC: Total iron-binding capacity; mcg/dL: Micrograms per deciliter; ALT: Alanine aminotransferase; U/L: Units per liter; mg/dL: Milligrams per deciliter; SD: Standard deviation; AST: Aspartate aminotransferase.

*: statistically significant as P value <0.05

Table 4: Multivariate logistic regression analysis to predict iron deficiency anemia via BMI

	OR (95% CI)	P-value
Age (years)	0.837 (0.689 - 1.017)	0.074
Gender	3.204 (1.102 - 9.312)	0.032*
BMI	1.559 (1.261 - 1.927)	<0.001*

*Significant P-value; OR: Odds ratio; CI: Confidence interval; BMI: Body mass index.

Table 5: Multivariate logistic regression analysis to predict iron deficiency anemia via obesity

	OR (95% CI)	P-value
Age (Years)	0.914 (0.769 - 1.085)	0.303
Gender	2.894 (1.089 - 7.695)	0.033*
Obesity	5.007 (1.801 - 13.923)	0.002*

*Significant P-value; OR: Odds ratio; CI: Confidence interval.

Discussion:

In our investigation, we observed a significant relationship between gender and ID anemia, with an elevated proportion of males found in the non-iron-deficient group when in contrast with those in the iron-deficient group ($P = 0.017$). Furthermore, BMI was notably elevated in individuals who were iron-deficient, in contrast to those who were not. The noticed disparity was statistically significant ($P < 0.001$), indicating that individuals with ID may be more likely to have elevated BMI values. A closer look at the BMI categories revealed even more interesting insights: a significantly larger percentage of individuals with iron-deficiency anemia were classified as obese in contrast with those without ID. However, when we analyzed age, we did not observe any significant differences ($P = 0.867$), suggesting that age does not have a strong influence on the likelihood of experiencing iron-deficiency anemia in this population.

In a parallel study, Lisboa and colleagues explored the prevalence of iron-deficiency anemia (ID) among children under 60 months in Minas Gerais, Brazil. Their research, which included a random sample of 725 children, found a clear gender-based association with ID anemia: a higher percentage of males (66.6%) were in the non-iron-deficient group, compared to only 58.9% in the iron-deficient group ($P = 0.038$). These findings align with previous research by Nead and colleagues, which similarly highlighted the central role of gender and BMI in the distribution of ID anemia. These studies collectively emphasize that factors like gender and body weight are not secondary influences

but essential to understanding the prevalence of ID anemia across different populations^(11,12).

In contrast, a study conducted by Ranchana Khemphet and colleagues delved into the association between different weight categories, specifically OW, obesity, and morbid obesity, and iron titres in children. The research involved a cohort of 99 pediatric outpatients, aged between 5 and 15 years, who were categorized into three distinct groups: those with normal iron titres, those with ID, and those with ID anemia. The observations of the study revealed that both gender and BMI exhibited similar patterns between the healthy and iron-deficient groups, with P of 0.445 and 0.862, respectively⁽¹³⁾. These findings differ from those of prior studies, possibly owing to variations in study methodology, the demographic characteristics of the participants, and other variables that could have influenced the outcomes. This stands in contrast to the consistent associations between BMI and ID observed in earlier research.

In our study, we observed significantly lower levels of hemoglobin (Hgb), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH) in the iron-deficient (ID) group compared to the non-deficient group, with all differences being statistically significant ($P < 0.001$). These findings are consistent with previous research, such as that by Khalifa and colleagues, who also reported reduced values of Hgb, MCV, and MCH in individuals with ID anemia. The consistency across multiple studies highlights the importance of these blood parameters in diagnosing ID anemia and

underscores the need for targeted interventions to address this condition.⁽¹⁴⁾

Our study revealed significant differences in kidney function markers between the ID and non-deficient groups. Specifically, serum urea levels were elevated in the ID group ($P = 0.042$), and serum creatinine levels were slightly higher in the ID group ($P = 0.021$), suggesting that ID may impact kidney function. In contrast, no significant differences were observed in liver function markers, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), between the two groups ($P = 0.438$ for ALT and $P = 0.372$ for AST). These findings are in line with Khalifa's study, which also reported no significant differences in liver enzyme levels between individuals with ID anemia and controls ($P = 0.074$ for ALT and $P = 0.83$ for AST). However, Güneş and colleagues found no significant differences in kidney function markers between the ID and control groups ($P > 0.05$ for both), indicating that the impact of ID on kidney function may vary across studies.^(15,16)

In our study, we observed a moderate negative correlation between BMI and MCH ($r = -0.310$, $P = 0.002$). Additionally, both serum iron ($r = -0.236$, $P = 0.019$) and transferrin saturation ($r = -0.239$, $P = 0.017$) exhibited significant negative correlations with BMI, indicating a consistent pattern between higher BMI and lower iron levels. These findings align with research by Ghadimi and colleagues, who also identified negative correlations between BMI and transferrin saturation in a cohort of 206 elementary school students ($r = -0.24$, $P < 0.001$)⁽¹⁶⁾. Furthermore, Chambers and co-authors found an inverse correlation between BMI and iron levels in their study of 670 participants ($r = -0.23$, $P < 0.001$), further supporting the link between increased BMI and diminished iron concentrations. These consistent correlations suggest that higher BMI may impair iron metabolism, contributing to iron deficiency⁽¹⁷⁾.

The negative correlation between BMI and MCH indicates that as an individual's BMI increases, the Hgb content in the red blood cells tends to diminish. This suggests that obesity is influencing iron metabolism in the body, likely owing to the inflammation associated with excess body weight, which impairs the availability and utilization of iron. A diminished MCH is a characteristic sign of ID anemia, a condition that is often exhibited in individuals with elevated BMI. This condition is typically marked by a reduction in the ability of red blood cells to carry oxygen, reflecting the compromised iron metabolism in these individuals^(18, 19). As Obesity is associated with low-grade chronic inflammation. Several pro-inflammatory cytokines are secreted by adipose tissues, including interleukin-6 and tumor necrosis factor alpha. Indeed about one-third of interleukin-6 in the circulation is released from adipose tissue . The principal mechanism that links obesity and iron deficiency is low-grade systemic inflammation, observed in people with obesity . In people with overweight and obesity, serum hepcidin and serum interleukin-6 are significantly higher than those with normal weight .Hepcidin which is synthesized in the liver is stimulated by pro-inflammatory cytokines such as interleukin-6). A recent study reported that overweight and obese women with central adiposity demonstrated higher serum hepcidin, higher inflammation level, lower iron status, and lower iron absorption when fed with supplemental iron.

Moreover, the inverse relationship exhibited between BMI and both serum iron titres and transferrin saturation suggests that elevated BMI is associated with a diminish in circulating iron titres, as well as reduced efficiency in iron utilization. This reduced iron availability may be owing to increased titres of hepcidin, a protein that is elevated in individuals with obesity. Hepcidin is a small peptide hormone that is considered a key regulator for body iron homeostasis

Hepcidin is synthesized mainly in the liver and produced at low levels in other organs like adipose tissue. Hepcidin regulates plasma iron level by binding to ferroportin leading to internalization and degradation of ferroportin through blockage of cellular iron transport. Consequently, dietary iron absorption from the small intestine is downregulated, and thus, serum iron concentration is dropped. In addition, hepcidin slows down the release of recycled iron by macrophages to peripheral and iron mobilization from iron stores in the liver or spleen. Hepcidin plays a crucial role in regulating iron absorption and recycling, and when its titres are high, it interferes with the body's ability to absorb and reuse iron from food sources. As a result, even when dietary iron intake is adequate, individuals with elevated BMI may experience functional ID, where the body's iron stores are insufficient for optimal red blood cell production and oxygen transport⁽²⁰⁾.

In our investigation, we exhibited robust positive correlations between BMI and various biomarkers, including ALT ($r=0.455$, $P<0.001$), AST ($r=0.421$, $P<0.001$), and serum creatinine ($r=0.491$, $P<0.001$). Additionally, a moderate positive correlation was identified between BMI and serum urea titres ($r=0.205$, $P=0.042$). In contrast, no significant correlations were exhibited between BMI and other variables, such as Hgb, MCV, serum ferritin, and TIBC.

The exhibited positive relationship between BMI and liver enzymes, specifically ALT and AST, underscores the detrimental effects of excess body weight on liver health. As BMI increases, it places more strain on the liver, raising the likelihood of developing NAFLD, a condition commonly seen in individuals with obesity. Elevated ALT and AST titres are indicative of liver inflammation or damage, signaling compromised liver function. Moreover, the connection between BMI and kidney function markers, like serum creatinine and urea,

emphasizes the added pressure obesity places on the renal system. An elevated BMI can result in increased filtration demands on the kidneys, potentially leading to early-stage kidney dysfunction, which is commonly exhibited in obese individuals. This highlights the importance of managing obesity to reduce the risk of both liver and kidney complications⁽²²⁾.

In the present analysis, ROC testing was employed to determine the effectiveness of BMI in predicting IDA. The findings exhibited an AUC of 0.766, with a 95% confidence interval ranging from 0.660 to 0.871. The best BMI cutoff was identified as >28.2 , with sensitivity, specificity, PPV, and NPV values of 75.8%, 60%, 44%, and 85.7%, respectively.

Meanwhile, Dutta and co-authors studied a cohort of 245 young adults (113 men and 132 women) aged between 18 and 30. Their ROC analysis revealed that BMI was the most reliable predictor of anemia in this group, with BMI cutoffs of 20.65 kg/m² for men (AUC: 0.889) and 19.7 kg/m² for women (AUC: 0.904) proving most effective. Additionally, the research highlighted an elevated prevalence of anemia among Malaysian Indians (26.22%), followed by Malays (21.54%), "Others" (20%), and Chinese (14.5%). Notably, the prevalence was significantly elevated in young women (21.96%) in contrast with men (18.6%) across Malaysia⁽²³⁾.

Multivariate logistic regression analysis revealed that both obesity and female gender were strong predictors of IDA. Specifically, each unit increase in BMI was linked to a 55% elevated likelihood of developing IDA (OR=1.559, 95% CI=1.261–1.927, $P<0.001$). Additionally, obesity was associated with a fivefold increase in risk (OR=5.007, 95% CI=1.801–13.923, $P=0.002$), while being female tripled the risk of IDA (OR=3.204, 95% CI=1.102–9.312, $P=0.032$). These findings emphasize the significant impact of obesity and gender on IDA risk,

underscoring the need for targeted screening in at-risk populations.

However, the research has certain limitations. It was conducted in a single center, which may limit the generalizability of the observations. The small sample size could reduce the statistical power, especially for subgroup analyses. The cross-sectional design prevents establishing causality between obesity and IDA. Additionally, potential confounders like physical activity (physically inactive, moderate activity level or high physical activity), diet (calorie intake, macronutrient balance, high sugar or fat intake and consumption of processed foods), and socioeconomic status (income, education, residence and occupation) were not assessed.

Conclusion:

IDA is prevalent among OW and obese children, with obesity and female gender identified as significant risk factors. The obtained observations emphasize the urgent need for proactive screening and customized interventions to effectively address the combined challenge of malnutrition in children.

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This study was conducted without receiving any dedicated financial support from public, private, or non-profit funding bodies. The research was carried out independently, ensuring that no external financial influences were involved in the design, execution, or analysis.

Author Contributions

The authors made equal and significant contributions to every aspect of this study, from the initial concept and design to the analysis of data and writing of the manuscript. Each author was fully involved in the research process, ensuring a collaborative and comprehensive approach to the research.

Conflicts of Interest

The authors declare that there are no conflicts of interest related to this research. The findings and interpretations presented

in this study are solely based on the authors' independent research and analysis.

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