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Original Article

Inflammation-Driven Iron Deficiency in Obese Children: The Role of Hepcidin and IL-6

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ABSTRACT

Aim: Obesity and iron deficiency represent two of the most prevalent nutritional disorders worldwide. Obesity is accompanied by chronic low-grade inflammation, with elevated circulating levels of pro-inflammatory cytokines, particularly interleukin 6 (IL-6). Obesity-related inflammatory pathways promote hepatic hepcidin synthesis, with IL-6 serving as a central mediator of hepcidin transcription under inflammatory conditions. Hepcidin is the principal regulator of intestinal iron absorption, and its increased expression contributes to impaired iron availability in obese individuals. This study aimed to examine the association between obesity and iron deficiency and to clarify the role of hepcidin in iron homeostasis among obese children.

Materials and Methods: This case-control study enrolled 50 children with obesity [body mass index (BMI) >95th percentile] and 50 healthy non-obese children (BMI between the 5th and 95th percentiles), aged 8-18 years. The evaluated parameters included hemoglobin (Hb), mean corpuscular volume (MCV), serum iron, ferritin, total iron-binding capacity, transferrin saturation (TS), as well as serum hepcidin and IL-6 levels.

Results: Obese children had significantly lower serum iron, Hb, MCV, ferritin, and TS (all $p<0.05$), and higher hepcidin and IL-6 levels ($p=0.024$ and $p=0.032$, respectively), compared to the controls. Hepcidin levels were directly correlated with IL-6 ($p<0.001$) and BMI standard deviation scores ($p=0.019$). Inverse correlations were observed between hepcidin and iron ($p=0.024$), hepcidin and Hb ($p=0.001$), and hepcidin and MCV ($p=0.02$).

Conclusion: Chronic inflammation of obesity and elevated hepcidin levels result in the low iron states in obese children.

Keywords: Obesity, hepcidin, interleukin-6, iron deficiency

Introduction

Obesity is characterized by the excessive accumulation of adipose tissue which adversely affects health outcomes. Over recent decades, it has emerged as one of the most important public health challenges worldwide and is recognized by the World Health Organization as a global epidemic (1). Obesity develops through both adipocyte hypertrophy and hyperplasia, leading to an expansion of adipose tissue mass (2). Obesity substantially increases susceptibility to metabolic complications which include insulin resistance, abnormalities in lipid metabolism, and the development of hypertension. Emerging evidence suggests that obesity also contributes to disturbances in iron metabolism, leading to iron deficiency anemia (IDA) (3).

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Iron plays a fundamental role in human biology by supporting cellular energy pathways, erythropoiesis, and immune competence. In addition, it is required for heme synthesis, enzymatic reactions such as cytochrome P450 activity, deoxyribonucleic acid replication and repair, and normal neurodevelopment (2). The relationship between hepcidin, obesity, and iron metabolism is complex and multifaceted, involving various metabolic and inflammatory pathways.

In obesity, chronic low-grade inflammation is associated with increased circulating hepcidin levels, which in turn contributes to disturbances in systemic iron regulation (4). Increased hepcidin activity limits intestinal iron uptake and restricts iron mobilization from storage sites, which can reduce iron availability even when total body iron stores are preserved (5,6). Studies have shown that obese individuals, including children and adolescents, exhibit higher hepcidin levels compared to their normal-weight counterparts. Higher hepcidin levels have been shown to be associated with elevated concentrations of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α) (7-9). This inflammatory response is primarily driven by dysfunctional adipose tissue, which secretes pro-inflammatory cytokines which in turn stimulate hepcidin production (10,11).

The present study was designed to examine the association between serum hepcidin and IL-6 concentrations and markers of iron status in children with obesity.

Materials and Methods

Ethical Consideration

This study was prepared based on a thesis into research into childhood obesity causing IDA which was completed in 2012. The study received approval from the Ethics Committee of Marmara University Faculty of Medicine (approval no.: MAR-AEK-09-2010-0066, date: 28.09.2010).

Study Population

A total of 100 children between 8 and 18 years of age were enrolled, including 50 children with obesity and 50 age-matched healthy controls. Recruitment was conducted through the well-child and pediatric outpatient clinics of Marmara University Faculty of Medicine Hospital. Written informed consent was obtained from both the participants and their legal guardians.

Obesity was classified using age- and sex-specific body mass index (BMI) percentiles, with values above the 95th percentile and a corresponding BMI standard deviation score (SDS) greater than +2 SD based on local growth references (12). The obese group consisted of children with no known systemic, endocrine, neurologic, or chronic diseases and who were not on any medications. The control group included healthy children matched for age and sex, whose BMI values ranged between the 5th and 95th percentiles according to age- and sex-specific reference standards.

Exclusion criteria for both groups included: (1) the use of iron supplements within the past six months; (2) the presence of an active infection; (3) the presence of any inflammatory condition (e.g., inflammatory bowel disease, autoimmune disease) or a history of cancer therapy within the past year; (4) the presence of any significant risk factors for iron deficiency (e.g., chronic blood loss from heavy menstruation or gastrointestinal bleeding, or adherence to a vegetarian diet); and (5) the presence of any known disorders of erythrocyte function (e.g., thalassemia, lead poisoning, sickle cell disease, or sideroblastic anemia).

Instruments and Laboratory Methods

Each participant underwent a detailed medical evaluation. All blood samples for hematologic indices and biochemical parameters were collected in the morning after an overnight fast in order to minimize potential diurnal and dietary variations. Complete blood count, including hemoglobin (Hb) and mean corpuscular volume (MCV), was performed on a Beckman Coulter LH 780 analyzer. Serum iron and total iron-binding capacity were assessed using a colorimetric method on the Roche Cobas C 502 analyzer, while ferritin concentrations were determined by an electrochemiluminescence immunoassay on the Roche Modular Analytics E170 platform. Transferrin saturation (TS) was calculated as [serum iron ÷ total iron binding capacity (TIBC)] × 100. For inflammatory markers, serum samples were frozen at -20 °C until analysis. Serum hepcidin concentrations were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (DRG® Hepcidin ELISA, DRG Diagnostics, Germany) according to the manufacturer's instructions. The analytical sensitivity of the assay was approximately 0.9 ng/mL, with a detection range of 0-80 ng/mL. The intra-assay coefficient of variation was below 8%, whereas the inter-assay coefficient of variation remained below 10%. Serum IL-6 levels were determined using a commercially available ELISA kit (eBioscience®, Vienna, Austria) following the manufacturer's protocol. The analytical sensitivity of the assay was approximately 0.9 pg/mL, with a detection range of 2-200 pg/mL. Both intra-assay and inter-assay coefficients of variation were below 10%. For both assays,

all samples were analyzed in duplicate and measured within the same assay run in order to minimize analytical variability.

Statistical Analysis

All statistical evaluations were carried out with GraphPad Prism software (version 5.00; GraphPad Software, San Diego, CA, USA). Continuous variables were summarized using appropriate descriptive measures, including means with SDs or medians with interquartile ranges, depending on the data distribution.

Comparisons between groups were performed according to the data distribution. The Mann-Whitney U test was applied for non-normally distributed variables, including IL-6 and hepcidin, whereas normally distributed continuous variables were analyzed using the independent samples t-test. Categorical variables were evaluated with the chi-square test. Associations between variables were examined using Pearson's correlation coefficient for normally distributed data and Spearman's rank correlation coefficient for variables with non-normal distributions, including hepcidin and IL-6. Statistical significance was defined as a p value below 0.05.

Results

The study population comprised 50 children and adolescents with obesity and 50 healthy non-obese controls. The median age was comparable between the obese and control groups [12.0 (8.0-18) vs. 12.1 (8.3-18) years], with a similar male distribution in both groups (60% vs. 52%). No significant differences were observed between the groups with respect to age or sex. The group comparisons of the demographic characteristics, anthropometric measurements, and iron-related parameters are summarized in Table I. The mean BMI-SDS of the obese group was 2.6±0.4 versus -0.6±0.9 for the non-obese group. Compared with the healthy controls, the children with obesity had significantly reduced serum iron (p=0.024), Hb (p=0.001), MCV (p=0.02), ferritin (p<0.05), and TS levels (p<0.05). Conversely, the children with obesity showed significantly elevated IL-6 and hepcidin concentrations compared with the controls (p=0.024 and p=0.032, respectively) (Figure 1). TIBC was much higher in the obese group compared with the control group (p=0.001). Although ferritin levels were significantly lower in the obese group compared with the controls (p=0.03), mean ferritin values in both groups remained within the age-appropriate reference ranges. Hepcidin levels were strongly and positively associated with IL-6 concentrations (p<0.0001). Figure 2 supporting the hypothesis that chronic inflammation in obesity stimulates hepcidin production. Hepcidin concentrations demonstrated significant negative associations with serum iron (p=0.024), Hb (p=0.001), and MCV (p=0.02), indicating that elevated hepcidin levels contribute to impaired iron availability and anemia in obese children (Table II). No significant

Table I. Anthropometrical and biochemical features of the obese group versus the control group

		Control		Obese	p value
Age		12.9±2.9		12.7±2.7	0.767
Sex					
Girls	20	40%	24	48%	0.420
Boys	30	60%	26	52%	
BMI (kg/m^2)		17.98±2.09		30.85±3.63	0.0001
BMI-SDS		-0.60±0.92		2.60±0.47	<0.0001
Ferritin (ng/mL)		35.97±13.62		29.59±15.26	0.03
Serum iron (ug/dL)		93.78±28.17		69.28±23.59	0.0001
TIBC (mg/dL)		363.16±40.4		402.56±43.17	0.0001
Transferrin saturation (%)		26.24±8.9		17.45±6.35	0.0001
Hb (g/dL)		13.05±0.84		12.63±0.89	0.019
MCV (fL)		84.65±2.78		82.68±3.46	0.002
Hepcidin (ng/mL)		8.61±1.97		11.12±5.48	0.024
IL-6 (pg/mL)		1.99±0.80		3.09±3.99	0.032

BMI: Body mass index, SDS: Standard deviation score, TIBC: Total iron binding capacity, Hb: Hemoglobin, MCV: Mean corpuscular volume, IL-6: Interleukin-6

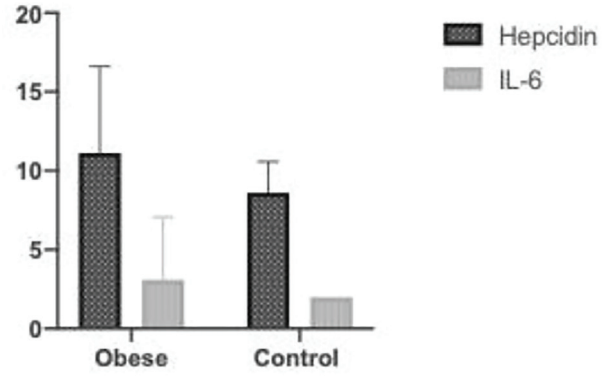

Figure 1. Comparisons of serum hepcidin and IL-6 between the two group
IL-6: Interleukin-6

Table II. Correlations between IL-6, serum hepcidin and iron profiles in all children

		IL-6	Hepcidin
Ferritin	r	-0.076	-0.155
	p	0.452	0.122
Iron	r	**-0.219**	**-0.226**
	p	**0.03 2**	**0.024**
TIBC	r	-0.039	0.091
	p	0.704	0.369
Hemoglobin	r	**-0.236**	**-0.328**
	p	**0.018**	**0.001**
MCV	r	**-0.221**	**-0.233**
	p	**0.029**	**0.02**
TS	r	0.015	-0.042
	p	0.881	0.674

IL-6: Interleukin-6, TIBC: Total iron binding capacity, MCV: Mean corpuscular volume, TS: Transferrin saturation

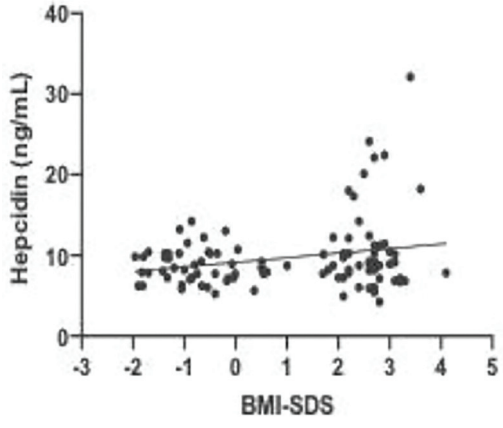

Figure 3. Correlation between BMI-SDS and serum hepcidin
BMI: Body mass index, SDS: Standard deviation score

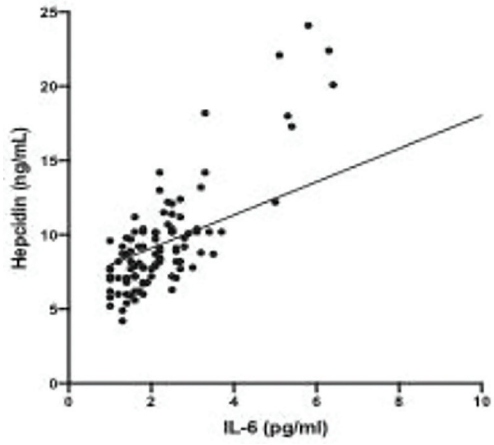

Figure 2. Correlation between serum hepcidin and IL-6
IL-6: Interleukin-6

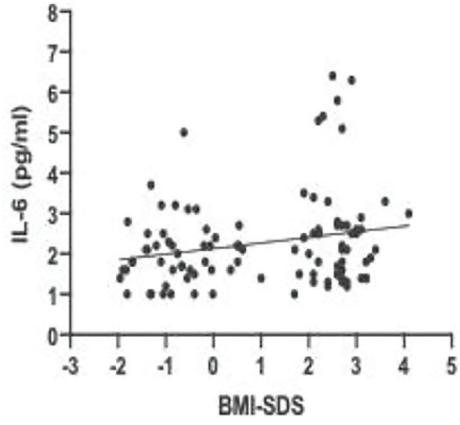

Figure 4. Correlation between BMI-SDS and serum IL-6
BMI: Body mass index, SDS: Standard deviation score, IL-6: Interleukin-6

correlations were observed between TS and either hepcidin (p=0.674) or IL-6 (p=0.881). Positive correlations between BMI-SDS and hepcidin (p=0.001) and also between BMI-SDS and IL-6 (p=0.03) are shown in Figures 3 and 4. Hepcidin and IL-6 were negatively correlated with Hb, MCV and iron levels (Table II).Higher BMI-SDS values were associated with increased IL-6, hepcidin, and total iron-binding capacity levels, while inverse relationships were observed with Hb, serum iron, and MCV (Table III). Separate subgroup analyses showed no statistically significant associations between any inflammatory markers, including hepcidin and IL-6, and iron-related parameters in either the obese group or the non-obese group.

Table III. Correlations between BMI-SDS and serum Hepcidin, IL-6, and iron profiles in all children

		BMI-SDS
IL-6	r	0.2098
	p	0.0361
Hepcidin	r	0.2339
	p	0.0192
Iron	r	-0.3659
	p	0.0002
TIBC	r	0.4219
	p	<0.0001
Hemoglobin	r	-0.3625
	p	0,0002
MCV	r	-0.3010
	p	0.0023
Table III. Continued		
		BMI-SDS
TS	r	-0.4451
	p	<0.0001
Ferritin	r	-0.1638
	p	0.1033

BMI: Body mass index, SDS: Standard deviation score, IL-6: Interleukin-6, TIBC: Total iron binding capacity, MCV: Mean corpuscular volume, TS: Transferrin saturation

Discussion

Iron plays a critical role in fundamental biological processes across almost all forms of life, and disturbances in iron balances have widespread clinical implications. According to global health data, iron deficiency remains the most prevalent nutritional disorder worldwide (13). In childhood obesity, contributing factors include genetic predisposition, imbalanced diet, reduced physical activity with lower myoglobin synthesis, and increased iron needs due to greater blood volume (14). However, studies have shown no major differences in dietary iron intake or in enhancers/inhibitors of absorption between obese and non-obese children, suggesting diet alone does not explain the higher prevalence of deficiency (15).

The inverse association between adiposity and iron status was initially described by Wenzel et al. (16) in 1962; obese adolescents had significantly lower mean serum iron than their non-obese peers. Later studies supported this observation. In the Third National Health and Nutrition Examination Survey, data from 9,698 children aged 2-16 years were analyzed, revealing that 10.2% of participants had BMI values exceeding the 95th percentile. Within this subgroup, iron deficiency prevalence was 6.2% in children aged 2-5 years and 9.1% in those aged 12-16 years (17). In an Iranian study of 1,675 university students, Hb and MCV decreased with rising BMI (18). The authors concluded that overweight children and adolescents have a higher prevalence of iron deficiency than their normal-weight peers, consistent with our findings. Moafi et al. (18) also noted that anemia risk increased with age, whereas we found no correlation between serum iron and age (p=0.7).

Cepeda-Lopez et al. (19) demonstrated that iron deficiency in obesity is driven not only by low dietary intake but also by inflammation. Previous studies have reported that, even with comparable dietary iron intake, obese women exhibit significantly reduced serum iron levels compared with their non-obese counterparts (p=0.014). In contrast, obese children have been shown to display increased TIBC (p<0.001), suggesting the presence of a compensatory mechanism. C-reaktif protein (CRP) levels were about fourfold higher in obese participants, reflecting chronic inflammation which may impair iron metabolism. Similarly, the obese children in our study had lower serum iron levels and higher total iron-binding capacity than the non-obese children. Richardson et al. (20) demonstrated that obese children aged 2-19 years (BMI >95th percentile) had lower serum iron, Hb, ferritin, and TS than age-matched controls, in findings which parallel those observed in our cohort.

Adipose tissue contributes to chronic low-grade inflammation through the secretion of cytokines such as IL-1, IL-6, and TNF-α, as well as adipokines including leptin, adiponectin, hepcidin, and resistin (21). IL-6 and hepcidin levels were significantly increased in the obese children relative to the controls in our study. IL-6–mediated activation of the Janus kinase/signal transducer and activator of transcription signaling cascade plays a central role in the regulation of hepcidin expression. In obesity, chronic inflammation elevates IL-6, stimulating hepatic hepcidin production, a mechanism important for iron homeostasis, but which also contributes to iron deficiency (22). In the overall cohort, IL-6 levels were positively associated with hepcidin, whereas hepcidin showed an inverse relationship with serum iron, supporting inflammation-mediated hepcidin regulation of iron metabolism. However, when correlation analyses between inflammatory markers (IL-6 and hepcidin) and iron parameters were performed separately within the obese and non-obese groups, no significant associations were identified. This pattern suggests that obesity-related disturbances in iron metabolism may primarily reflect

a group-level difference between obese and non-obese children rather than a simple linear association at an individual level.

Chronic inflammation in obesity is thought to disrupt iron homeostasis by reducing intestinal absorption and increasing iron sequestration in macrophages and the reticuloendothelial system (23). In anemia of inflammation, bone marrow and iron stores are usually adequate, yet serum iron levels remain low (24). Ferritin levels may be normal or elevated in this context, reflecting both iron storage and its role as an acute-phase reactant (25). However, in childhood obesity, the interpretation of ferritin is complex due to the coexistence of low-grade chronic inflammation and disturbances in iron homeostasis. Obesity-related inflammation may increase hepcidin and restrict iron availability through the hepcidin–ferroportin axis without necessarily inducing a proportional increase in ferritin (26,27). Accordingly, studies on ferritin in obesity are heterogeneous: some report impaired iron status with higher hepcidin and reduced iron absorption despite comparable ferritin levels (8), whereas others demonstrate lower ferritin concentrations in obese children, particularly when true iron deficiency predominates (28). In our study, mean Hb, MCV, iron, TS, and ferritin were all significantly lower in the obese group, although ferritin values remained within age-appropriate normal ranges.

Weight reduction improves hepcidin levels and iron status in obesity by reducing chronic inflammation. In a randomized controlled trial, young women with obesity and IDA showed significant reductions in serum hepcidin levels, accompanied by increases in Hb and ferritin following diet-induced weight loss (29). Similarly, obese children participating in a weight loss program exhibited decreased hepcidin concentrations, which were correlated with lower leptin and IL-6 levels, highlighting the role of reduced inflammation in improving iron absorption (30).

Elevated hepcidin in obesity alters iron distribution and contributes to IDA, particularly in children, who often respond poorly to oral iron intake due to persistently high hepcidin (31). Thus, standard supplementation may be insufficient, and strategies targeting inflammation or modulating hepcidin may be required.

Study Limitations

This study had several limitations, including its single-center design and relatively small sample size, which may restrict the generalizability of the results. Dietary intake and physical activity were not evaluated in detail, and additional inflammatory markers such as CRP or adipokines, which could have provided a more comprehensive picture, were not measured. These factors should be considered when interpreting the results, and future multicenter longitudinal studies are needed in order to confirm and expand our observations.

Conclusion

Our study shows that obesity-related inflammation, reflected by elevated IL-6 and hepcidin, contributes to iron deficiency in children with obesity. These findings underscore the need for a comprehensive approach to management which addresses both nutritional and inflammatory factors. Weight control and anti-inflammatory strategies may help reduce hepcidin overexpression and improve iron availability. Future studies are warranted in order to explore targeted interventions to lower chronic inflammation or inhibit hepcidin activity, thereby restoring normal iron metabolism in this vulnerable group.

Ethics

Ethics Committee Approval: This study was prepared on the basis of the thesis on the research of childhood obesity causing IDA, completed in 2012. The study received approval from the Ethics Committee of Marmara University Faculty of Medicine (approval no.: MAR-AEK-09-2010-0066, date: 28.09.2010).

Informed Consent: Written informed consent was obtained from both the participants and their legal guardians.

Footnotes

Authorship Contributions

Concept: D.H., A.B., Design: D.H., B.Y., A.B., Data Collection or Processing: D.H., Analysis or Interpretation: D.H., B.Y., A.B., Literature Search: D.H., B.Y., A.B., Writing: D.H., A.B.

Conflict of Interest: The authors declare that they have no competing interests.

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