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Serum Hepcidin Level in Obese Children and Adolescents: It's Association with Iron Deficiency Anemia

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Authors' contributions

All authors had equal role in design, work, statistical analysis and manuscript writing. All authors have approved the final article work.

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ABSTRACT

Background: Childhood obesity is a worldwide chronic public health problem. It was found that obesity is associated with iron deficiency and iron profile abnormalities, which appear to be caused by several factors such as decreased intake, insufficient bioavailability, and deficient intestinal iron uptake as well as iron release from stores because of an over expression of hepcidin.
Aim of the Work: Was to estimate serum hepcidin levels in obese children and adolescents and to evaluate its relation with iron deficiency anemia in these children.
Subjects and Methods: The current study included 50 patients recruited from the Nutrition Clinic of Pediatric Department at Tanta University Hospital, 25 of them were obese with iron deficiency anemia and the other 25 were obese without iron deficiency anemia and 25 healthy children and adolescents of matched age and sex enrolled as controls. All studied children were subjected to complete history taking, thorough clinical examination including anthropometric measures (Weight, height, Body mass index), assessment of pubertal status using Tanner criteria and laboratory investigations including: CBC, BUN, creatinine, ALT, AST, stool analysis, occult blood in stool, CRP, iron profile, Serum Hepcidin, abdominal ultrasound.

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Results: There were significant differences between patients and control group as regard Weight, BMI and their z scores. Significantly lower levels of hemoglobin, serum ferritin, serum iron and transferrin saturation in obese children with IDA than obese children without IDA and controls and significantly higher levels of TIBC were found in obese children with IDA compared to obese children without IDA and controls. As regard CRP it was significantly higher in obese children than controls. Serum hepcidin was significantly higher in obese children than controls but there is no significant difference between obese children with IDA and obese children without IDA. Significant positive correlation between Serum hepcidin levels and BMI in obese children was found. **Conclusion:** Serum hepcidin level was significantly higher in obese children and adolescents in comparison with healthy lean control with no significant difference between obese children with IDA and obese children without IDA. So, estimation of serum hepcidin level is not diagnostic but it may be beneficial in screening of iron deficiency anemia in pediatric obese individuals. Further studies with larger sample size are needed to verify these findings.

Keywords: Hepcidin; iron deficiency anemia; obesity.

1. INTRODUCTION

Obesity is an energy metabolism disorder resulting in the excessive storage of fat and that may also lead to physical and psychological problems [1]. Obesity is a growing public health problem; the prevalence of this condition has increased dramatically in recent years. Incorrect eating habits along with a sedentary lifestyle can lead to overweight and obesity among children and adolescents [2].

Obesity and iron deficiency are two of the most common nutritional disorders worldwide [2].

Due to the epidemic increase in obesity worldwide, numerous studies were conducted in the past decade, specifically examining the association between obesity and iron deficiency (ID). Classic explanations for ID among obese individuals include inadequate dietary iron intake, increased iron requirements due to increased blood volume and physical inactivity. However, the latest research suggests that obesity-associated low-grade inflammation and the iron-regulatory protein hepcidin play a principal role in the regulation of endogenous iron homeostasis [3].

Hepcidin is a 25 amino acid peptide which is present in human serum and urine. It acts as a key regulator of iron metabolism [3]. It controls both iron entering to plasma from absorptive sites and iron released from stores. It performs its function through binding and degrading the sole cell iron exporter ferroportin, which is highly expressed on baso-lateral membrane of enterocytes and macrophage surface [4].

So, it decreases iron absorption from the small intestine, reduces the transport of iron from macrophages to the plasma and/or prevents mobilization of stored iron from the liver [5].

Considering that obesity represents a low grade chronic inflammatory state, the hypoferremia associated with obesity could be attributed to certain metabolic and molecular adaptations induced by inflammation. This hypothesis is supported by the significant inverse correlations observed in overweight and obese individuals between serum iron concentration and the concentrations of a variety of adipocytokines and pro inflammatory cytokines after controlling for iron intake, iron needs and iron losses. Furthermore, certain acute phase peptides (e.g. hepcidin) that are usually elevated in obesity have also been found to regulate iron homeostasis [6].

2. SUBJECTS AND METHODS

This study (Case-Controlled study) was done after approval from ethical committee of research center of Tanta University and written consent from the parents of all children included in this study. The study was conducted on 50 obese children and adolescents, recruited from the Nutrition Clinic, Pediatric Department, Tanta University, in the period from October 2018 to September 2019 divided into two subgroups: 25 obese children and adolescents with iron deficiency anemia including 13 males and 12 females with their age ranged from 6 to 16 years and mean age value of 10.84 ± 3.0 (Group A) and 25 obese children and adolescents without iron deficiency anemia including 13 males and 12 females with their age ranged from 6 to 16 years and mean age value of 10.60 ± 3.15 (Group B)

This study included also 25 healthy children as a control group including 12 males and 13 females

with their age ranged from 6 to 15 years and mean age value of 10.44 ± 2.86.

2.1 Inclusion Criteria

our study included children and adolescent with age ranging from 6 to 18 years, with body mass index (BMI) equal to or greater than the 95th percentile for gender and age and with serum ferritin lower than 30 µg/dl, Transferrin saturation lower than 16% and hemoglobin (Hb) concentration lower than 11.5 g/dl for children 5-11 years of age, lower than 12 g/dl for those 12-15 years of age and for girls older than 15 years and lower than 13g/dl in boys older than 15 years in group A.

2.2 Exclusion Criteria

Children with syndromatic obesity (Prader Willi, Laurence-Moon Biedle syndrome, etc), endocrinal obesity causes such as Cushing's syndrome or hypothyroidism, systemic diseases including liver diseases, malignancy, drug use (corticosteroids, antithyroid drugs, anti-psychotic) , infection or collagen disease, diabetes mellitus or hypertension were excluded from the study. In addition, children at higher risk of iron deficiency anemia (drinking large amounts of cow milk and parasitic infestation), with hemoglobin lower than 8 gm/dl and those receiving iron therapy within the previous 6 months were also excluded.

All the children in the study were subjected to the following:

1. Full history taking with special emphasis on: Past history for systemic diseases ,Family history of obesity, hypertension and diabetes , Drug administration (corticosteroids, anti-thyroid drugs, anti-psychotic, use of other drugs),Dietary History especially that of iron intake and social history including (number of children in the family, educational level and job of mother)
2. Clinical examination: Including medical examination of (head & neck, chest, heart, abdomen, upper and lower limbs).
3. Anthropometric measures and Z-score calculation for (Weight, Height and Body mass index (BMI).
4. Assessment of pubertal status using Tanner criteria [7].
5. Investigations including: Complete blood count, stool analysis, occult blood in stool, CRP, iron profile [serum ferritin, serum iron, total iron binding capacity (TIBC), transferring saturation] and serum hepcidin.

Statistical analysis of the present study was conducted by using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). Qualitative data were described using number and percent. The Kolmogorov-Smirnov test was used to verify the normality of distribution. Quantitative data were described using range (minimum and maximum), mean and standard deviation, median and interquartile range (IQR). Significance of the obtained results was judged at the 5% level.

The used tests were (Chi-square test, Monte Carlo correction, F-test (ANOVA), Mann Whitney test, Kruskal Wallis test and Spearman coefficient).

3. RESULTS

There were no statistically significant differences between obese children and controls as regard (Age, sex, residence, family history of diabetes and hypertension, dietetic history of iron intake, social history and tanner staging) while there was statistically significant difference between them as regard family history of obesity as shown in Table 1.

Weight, BMI, and their corresponding z-scores were significantly higher in obese children (groups A and B) than controls, with no statistically significant difference between obese children with iron deficiency anemia (group A) and obese children without iron deficiency anemia (group B). On the other hand, height and the corresponding z-scores did not show a significant difference between obese children and controls Table 2.

As shown in Table 3, both hemoglobin and MCV were significantly lower in obese children with iron deficiency anemia (group A) than obese children without iron deficiency anemia (group B) and controls with no statistically significant difference between group B and controls. Also, no statistically significant difference between obese children (groups A and B) and controls as regard WBCS and Platelets.

As regard iron profile, serum ferritin is significantly lower in obese children with iron deficiency anemia (group A) than controls than obese children without iron deficiency anemia

Table 1. Characteristics of the studied groups

	Groups						Test of sig.	P
	Patients				Controls			
	Group A (n = 25)		Group B (n = 25)		Group C (n = 25)			
	No.	%	No.	%	No.	%		
Sex								
Male	13	52.0	13	52.0	12	48.0	χ^2= 0.107	0.948
Female	12	48.0	12	48.0	13	52.0		
Age (years)								
Min. – Max.	6.0 – 16.0		6.0 – 16.0		6.0 – 15.0		F= 0.112	0.894
Mean ± SD.	10.84 ± 3.0		10.60 ± 3.15		10.44 ± 2.86			
Residence								
Rural	11	44.0	10	40.0	13	52.0	χ^2= 0.753	0.686
Urban	14	56.0	15	60.0	12	48.0		
Family history								
Obesity	14	56.0	13	52.0	0	0.0	χ^2= 21.181*	
Diabetes	12	48.0	8	32.0	9	36.0	1.462	<0.001*
Hypertension	12	48.0	9	36.0	6	24.0	3.125	0.481
								0.210
Nutritional history of iron intake								
Heme iron							χ^2= 3.360	
	16	64.0	20	80.0	14	56.0	0.321	0.186
Non heme iron	12	48.0	11	44.0	13	52.0		0.852
Social history								
Number of children								
IQR	3.0 – 4.0		3.0 – 4.0		2.0 – 3.0		H= 5.117	0.077
Median	3.0		3.0		3.0			
Educational level of mother								
Illiterate	5	20.0	3	12.0	1	4.0	χ^2= 9.950	^{MC}p=
Primary	10	40.0	8	32.0	4	16.0		0.118
Average	6	24.0	10	40.0	10	40.0		
High	4	16.0	4	16.0	10	40.0		

	Groups						Test of sig.	P
	Patients				Controls			
	Group A (n = 25)		Group B (n = 25)		Group C (n = 25)			
	No.	%	No.	%	No.	%		
Job of mother								
Not worker	19	76.0	17	68.0	14	56.0	χ^2= 2.280	0.320
Worker	6	24.0	8	32.0	11	44.0		
Tanner staging								
Pre pubertal	13	52.0	14	56.0	14	56.0	χ^2= 0.108	0.948
Pubertal	12	48.0	11	44.0	11	44.0		

χ^2: Chi square test F: F for ANOVA test; MC: Monte Carlo H: H for Kruskal Wallis test
p: p value for comparing between the studied groups
Group A: Obese with iron deficiency anemia.
Group B: Obese without iron deficiency anemia; Group C: Healthy children

Table 2. Anthropometric measures of the studied groups

	Groups			Test of Sig.	P
	Patients		Controls		
	Group A (n = 25)	Group B (n = 25)	Group C (n = 25)		
Weight (kg)					
Min. – Max.	32.0 – 94.50	34.0 – 99.0	20.0 – 63.0	F= 18.591*	<0.001*
Mean ± SD.	58.86 ± 17.22	62.85 ± 18.62	36.28 ± 13.61		p_1=0.674 p_2<0.001* p_3<0.001*
Z score of weight					
IQR	4.20 – 5.60	4.60 – 7.40	0.0 – 1.0	H= 52.059*	<0.001*
Median	5.0	5.80	0.50		p_1=0.104 p_2<0.001* p_3<0.001*
Height (cm)					
Min. – Max.	116.0 – 167.0	110.0 – 174.0	108.0 – 160.0	F= 1.198	0.308
Mean ± SD.	141.2 ± 14.89	140.4 ± 15.80	135.0 ± 15.42		

	Groups			Test of Sig.	P
	Patients		Controls		
	Group A (n = 25)	**Group B (n = 25)**	**Group C (n = 25)**		
Z score of Height					
IQR	-0.20 – 0.50	-0.20 – 1.0	-1.0 – 0.20	H= 5.369	0.068
Median	0.20	0.20	0.0		
BMI (kg/m^2)					
Min. – Max.	22.90 – 36.40	24.60 – 39.0	14.50 – 24.60	F= 76.722*	<0.001*
Mean ± SD.	28.81 ± 3.29	31.23 ± 4.46	19.10 ± 3.09		p_1=0.058
					p_2<0.001*
					p_3<0.001*
Z score of BMI					
IQR	2.01 – 3.20	2.02 – 3.90	-0.26 – 0.32	H= 49.995*	<0.001*
Median	2.47	3.05	-0.10		p_1=0.507
					p_2<0.001*
					p_3<0.001*

F: F for ANOVA test, Pairwise comparison bet. each 2 groups was done using Post Hoc Test (Tukey)
p: p value for comparing between the studied groups
p_1: p value for comparing between Group A and Group B
p_2: p value for comparing between Group A and Group C
p_3: p value for comparing between Group B and Group C
**: Statistically significant at p ≤ 0.05*
Group A: Obese with iron deficiency anemia.
Group B: Obese without iron deficiency anemia.
Group C: Healthy children

Table 3. Aboratory investigations in the studied groups

	Groups			Test of Sig.	P
	Patients		Controls		
	Group A (n = 25)	Group B (n = 25)	Group C (n = 25)		
Hemoglobin (gm/dl)					
Min. – Max.	9.10 – 12.60	10.80 – 14.10	11.40 – 14.10	F= 23.173*	p <0.001*
Mean ± SD.	11.08 ± 1.0	12.56 ± 0.91	12.61 ± 0.77		p_1<0.001*
					p_2<0.001*
					p_3=0.984
MCV (fl)					
Min. – Max.	55.60 – 79.50	61.80 – 86.20	75.0 – 85.0	F= 13.304*	p <0.001*
Mean ± SD.	72.16 ± 6.64	77.40 ± 5.91	79.94 ± 3.12		p_1=0.003*
					p_2<0.001*
					p_3=0.232
MCH					
Min. – Max.	17.20 – 26.60	20.50 – 30.30	25.0 – 27.50	F= 21.123*	p <0.001*
Mean ± SD.	22.93 ± 2.70	25.49 ± 1.86	26.36 ± 0.72		p_1<0.001*
					p_2<0.001*
					p_3=0.260
S. ferritin (ng/ml)					
Min. – Max.	10.70 – 29.70	31.60 – 85.90	31.30 – 69.50	F= 45.832*	p <0.001*
Mean ± SD.	21.03 ± 6.27	53.16 ± 17.58	43.66 ± 9.86		p_1 <0.001*
					p_2<0.001*
					p_3=0.020*
S. iron (µg/dl)					
Min. – Max.	20.0 – 29.0	50.0 – 108.0	60.0 – 152.0	F= 107.674*	p <0.001*
Mean ± SD.	26.32 ± 2.63	75.08 ± 19.21	94.68 ± 22.08		p_1 <0.001*
					p_2<0.001*
					p_3=0.001*
TIBC (µg/dl)					
Min. – Max.	351.0 – 471.0	200.0 – 360.0	270.0 – 350.0	F= 32.179*	p <0.001*
Mean ± SD.	380.60 ± 31.62	316.0 ± 34.09	322.0 ± 28.41		p_1 <0.001*
					p_2<0.001*
					p_3=0.779

	Groups			Test of Sig.	P
	Patients		Controls		
	Group A (n = 25)	**Group B (n = 25)**	**Group C (n = 25)**		
Transferrin saturation (%)					
Min. – Max.	5.30 – 8.20	16.0 – 33.80	17.10 – 49.60	F= 118.368*	$p < 0.001^*$
Mean ± SD.	6.91 ± 0.85	23.35 ± 5.81	29.56 ± 7.23		$p_1 < 0.001^*$
					$p_2 < 0.001^*$
					$p_3 < 0.001^*$
CRP (mg/L)					
IQR				H= 49.630*	$p < 0.001^*$
	7.40–11.80	7.55 – 11.80	1.50–3.0		$p_1 = 0.969$
Median	8.40	8.50	1.90		$p_2 < 0.001^*$
					$p_3 < 0.001^*$
Hepcidin (ng/ml)					
IQR.	175.6 – 304.7	144.5 – 267.7	102.6 – 174.9	H= 19.463*	$p < 0.001^*$
Median	203.7	208.5	123.6		$p_1 = 0.555$
					$p_2 < 0.001^*$
					$p_3 < 0.001^*$

F: F for ANOVA test, Pairwise comparison bet. each 2 groups was done using Post Hoc Test (Tukey)
H: H for Kruskal Wallis test, Pairwise comparison bet. each 2 groups was done using Post Hoc Test (Dunn's for multiple comparisons test)
p: p value for comparing between the studied groups
p_1: p value for comparing between Group A and Group B
p_2: p value for comparing between Group A and Group II
p_3: p value for comparing between Group B and Group II
**: Statistically significant at p ≤ 0.05*
Group A: Obese with iron deficiency anemia.
Group B: Obese without iron deficiency anemia.
Group II: Healthy children

(group B) while serum iron and transferrin saturation are significantly lower in obese children with iron deficiency anemia (group A) than obese children without iron deficiency anemia (group B) than controls and on the other hand TIBC is significantly higher in obese children with iron deficiency anemia (Group A) than obese children without iron deficiency anemia (Group B) and controls with no statistically significant difference between group B and controls.

Both CRP and serum hepcidin were significantly higher in obese children (groups A and B) than controls with no statistically significant differences between obese children with iron deficiency anemia (group A) and obese children without iron deficiency anemia (group B).

Serum hepcidin levels showed significant positive correlations with BMI of obese children (groups A and B) (r=0.302, p-value = 0.033), and BMI in obese children with iron deficiency anemia (group A) (r=0.637, p-value = 0.001).

As regard serum hepcidin, there was no statistically significant differences between pre-pubertal and pubertal children within each of the studied groups (obese group (A and B): Mean ± SD= 279.03 ± 204.51 versus 230.39 ± 114.43 respectively with p-value = 0.579; obese group with iron deficiency anemia (group A): Mean ± SD= 329.77 ± 269.17 versus 239.29 ± 126.03 respectively with p-value = 0.810; obese group without iron deficiency anemia (group B): Mean ± SD= 231.92 ± 108.64 versus 220.67 ± 105.52 respectively with p-value = 0.647; controls: Mean ± SD= 130.03 ± 39.06 versus 182.44 ± 109.57 respectively with p-value = 0.183).

There was also no statistically significant differences between male and female children within any of the studied groups (obese group (A and B): Mean ± SD= 234.38 ± 158.09 versus 280.78 ± 180.97 respectively with p-value = 0.103; obese group with iron deficiency anemia (group A): Mean ± SD= 246.12 ± 195.33 versus 329.91 ± 232.30 respectively with p-value = 0.123; obese group without iron deficiency anemia (group B): Mean ± SD= 222.65 ± 116.67 versus 231.65 ± 96.15 respectively with p-value = 0.574; controls: Mean ± SD= 124.18 ± 29.68 versus 179.77 ± 103.21 respectively with p-value = 0.270).

Due to the lack of a normal reference range for serum hepcidin in the pediatric age group, a ROC curve was done. Accordingly, the optimal cutoff point of serum hepcidin to discriminate obese children with iron deficiency from healthy individuals was >155 ng/ml (area under curve (AUC) of 0.826, specificity 72%, sensitivity 88%, positive predictive value 75.9% and negative predictive value 85.7%) while serum hepcidin level of 166 ng/ml or more was the optimal cutoff point to discriminate obese with iron deficiency anemia from obese without iron deficiency anemia (area under curve (AUC) of 0.558, specificity 32%, sensitivity 88%, positive predictive value 56.4% and negative predictive value 72.7%) (Fig. 1).

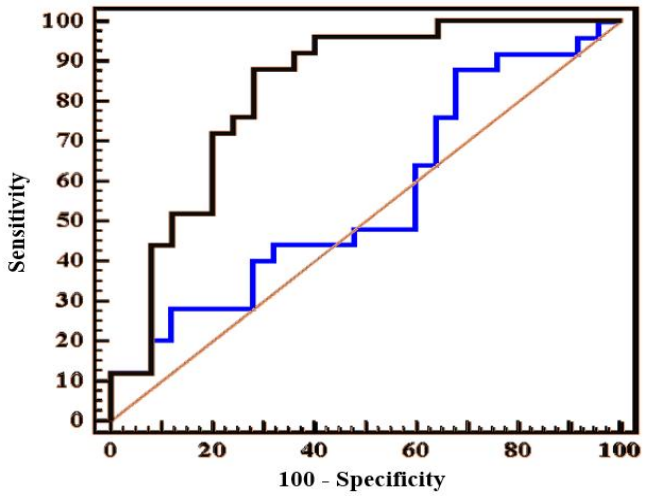

Fig. 1. ROC curve for hepcidin to diagnose obese children with iron deficiency anemia from healthy children (black curve) and from obese children without iron deficiency anemia (Group B) (Blue curve)

4. DISCUSSION

It has been suggested that obesity may be associated with a low-quality diet containing little iron. However, comparisons of dietary iron intake of normal and obese children have found no significant differences in iron intake, so hypoferremia associated with obesity is not related to iron intake [8].

In our study, it was found that there was no statistically significant difference between obese children and controls as regard nutritional history of heme iron intake (p= 0.186) and non heme iron intake (p= 0.852) while in Gajewska et al. [9] study iron intake was significantly higher in obese children than in controls.

As regard hematological data in the present study, hemoglobin and MCV were significantly lower in obese children with iron deficiency anemia (group A) than obese children without iron deficiency anemia (group B) and controls with no statistically significant difference between obese children without iron deficiency anemia and controls. This agrees with Sanad et al. [8] study while Gajewska et al. [9] study had found no significant difference between patients and controls.

Various hypotheses have attributed the association between obesity and iron deficiency to several factors. These include iron deficiency developing due to imbalanced nutrition in obese subjects, an increase in iron requirements due to increased blood volume, a decrease in myoglobin that binds iron in the muscles due to a decrease in physical activity and genetic predisposition. However, iron intake through diet does not differ between the obese and the non-obese subjects [3].

As regard CRP in the present study, it was significantly higher in obese children (groups A and B) than controls (p<0.001) while there was no statistically significant difference between obese children with iron deficiency anemia (group A) and obese children without iron deficiency anemia (group B) (p= 0.969). Sanad et al. [8], Sal E. et al. [1], Gajewska et al. [9] and Emam et al. [10] studies had found that there was statistically significant difference between patients and controls.

It was found that obese children had elevated levels of CRP which is acute phase protein. This is explained by the fact that excess fat deposited in visceral organs leads to low-grade chronic inflammation [11].

As regard iron profile data in our study, serum ferritin was significantly lower in obese children with iron deficiency anemia (group A) than obese children without iron deficiency anemia (group B) (p<0.001) and controls (p<0.001). It was significantly higher in obese children without iron deficiency anemia (group B) than controls (p= 0.003), although it was within normal level in both groups. Sanad et al. [8], Nazif HK et al. [12] and Emam et al. [10] studies observed that serum ferritin was significantly lower in obese children than controls while Gajewska et al. [9] and Sal E. et al. [1] studies had found that there was no significant difference between patients and controls as regard serum ferritin.

Ferritin is used as a marker of iron deficiency in various healthcare facilities across the globe. Being an acute phase reactant, serum ferritin level is prone to be higher in overweight and obese children, because of a state of subclinical, but generalized inflammation in them [13].

Serum iron and transferrin saturation levels were significantly lower in obese children with iron deficiency anemia (group A) than obese children without iron deficiency anemia (group B) (p<0.001) and controls (p<0.001). They were also significantly lower in obese children without iron deficiency anemia (group B) than controls (p= 0.001). This agrees with Sanad et al. [8] and Nazif HK et al. [12] studies in which serum iron was significantly lower in obese children than controls.

TIBC levels were significantly higher in obese children with iron deficiency anemia (group A) than obese children without iron deficiency anemia (group B) (p<0.001) and controls (p<0.001) with no statistically significant difference between obese children without iron deficiency anemia and controls (p= 0.779). Our results are consistent with Sanad et al. [8], Nazif HK et al. [12], Sal E et al. [1] and Emam et al. [10] studies which showed significantly higher TIBC levels in patients compared to controls.

Obesity is known to lead to hypoferremia. A similar picture was observed in our study. In their meta-analysis, Zhao et al. [3] showed that serum iron and transferrin are the best markers of iron metabolism in obese children. Similar findings were obtained in our study as well, and a significant positive correlation was observed

particularly between transferrin saturation and (hemoglobin, MCV, serum ferritin and serum iron) and significant negative correlation between it and TIBC in the obese groups. It should be kept in mind that ferritin-based tests could misdiagnose iron deficiency in obese patients.

Excessive adiposity is characterized by low-grade chronic inflammation. This leads to the production of certain inflammatory cytokines, such as IL-6 and tumor necrosis factor-alpha. IL6 is well known to stimulate hepatic hepcidin production. As hepcidin reduces the export of iron from macrophages, hepatocytes and enterocytes, which result in reduced iron absorption and sequestration of iron within splenic and hepatic macrophages. Thus, less iron is released and more iron is stored explaining the reduced iron use in the obese. In addition, it is likely that the adipose tissue produces minor levels of hepcidin, which further contributes to the iron deficiency state observed in obese children [1].

In the present study serum hepcidin level was significantly higher in obese children (groups A and B) than controls ($p<0.001$), while there was no statistically significant difference between obese children with iron deficiency anemia (group A) and obese children without iron deficiency anemia (group B) ($p= 0.555$). This finding is in agreement with Sanad et al. [8], Sal E. et al. [1] and Gajewska et al. [9] studies which found significantly higher serum hepcidin level in obese children compared to controls.

In the present study, there was significant positive correlation between serum hepcidin and BMI in obese children and obese children with iron deficiency anemia (group A). Sanad et al. [8] study had found positive correlation between serum hepcidin and (BMI and TIBC) and negative correlation between it and (hemoglobin, serum iron and transferrin saturation).

No significant correlation was observed between serum hepcidin and serum ferritin or transferrin saturation in our study and this agrees with Sal E. et al. [1] study.

This study showed that serum hepcidin had high sensitivity and low specificity. Thus estimation of serum hepcidin may be beneficial in screening for iron deficiency anemia among obese children and adolescents.

5. CONCLUSION

We concluded that serum hepcidin level was significantly higher in obese children and adolescents than healthy lean controls with no significant difference between obese children with IDA and obese children without IDA. So, estimation of serum hepcidin level is not diagnostic but it may be beneficial in screening of iron deficiency anemia in pediatric obese individuals. Further studies with larger sample size are needed to verify these findings.

CONSENT AND ETHICAL APPROVAL

Written consent was obtained from all parents or guardians of the children. The study was approved by the Ethical Committee of Faculty of Medicine, Tanta University.

Permission number is 32581/9/18.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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