Original Article

Is iron deficiency related with increased body weight? A cross-sectional study

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Summary. *Aim:* To investigate the relationship between iron deficiency and obesity through dietary intake and inflammation parameters in overweight and obese women. *Material/Methods:* A total of 619 women, aged 20-49 years were involved in the study. The biochemical parameters [whole blood count (WBC), anaemia parameters, C-reactive protein (CRP), high sensitive C-reactive protein (hsCRP), soluble transferrin receptor (sTfR)] and bioelectrical impedance analysis were analysed in all participants. *Results:* Iron deficiency was identified in 23.5% of women with normal weight, and was much common in obese (45.6%) and overweight (41.9%) women. Although dietary iron intake was higher in the overweight and obese women, the WBC, CRP, hsCRP and sTfR levels were lower in women with normal weight than overweight and obese women ($p<0.05$). *Conclusion:* Iron deficiency risk can be more likely to occur in obesity due to increased level of inflammation. Therefore, physicians may need to take a greater role in addressing iron deficiency in their obesity patients.

Key words: obesity, overweight, iron deficiency, women, inflammation

Introduction

The widely accepted classification of nutritional disorders include obesity and iron deficiency (ID). In the Turkish Nutrition and Health Survey 2010, obesity was highlighted as an important public health problem, since the obesity (Body Mass Index (BMI) ≥30 kg/m^2) and overweight (BMI=25.0-29.9 kg/m^2) was reported in 30.3% and 34.6% of the population, respectively (1). According to findings of various studies in Turkey, approximately 50.0% of children aged under 5 years, 30.0% of school-age children and 50.0% of lactating women have anaemia (1).

In 1960s, research studies highlighted an association between ID and obesity which can be more detrimental for health if occurred together (2, 3). Although there is no sufficient evidence to explain mechanism of this association, lower dietary iron intake (3, 4), reduced iron absorption in the small intestine or increased iron requirements due to larger blood volume and body surface area (5) would be the possible factors that cause iron deficiency in obese individuals. In addition, obesity is associated with a chronic low-grade inflammation which may led to decreased iron absorption from intestine or increased sequestration of iron (6-8).

These factors support the fact that obesity may have a significant association with ID. Hence, the present study was aimed to investigate the association between ID and obesity in women.

According to the best of our knowledge, this was the first study performed in Turkey aimed to investigate the relation between obesity and iron deficiency based on a hypothesis that the prevalence of ID would be higher in overweight and obese women than women with normal weight.

Materials and methods

Study population

The cross-sectional study was conducted between March 2012- November 2012. A total of 619 women, aged 20-49 years who visited the Izmir Bozyaka Training and Research Hospital, Internal Medicine, Endocrinology and Diet Outpatients Clinics. Written consent was obtained from all participants at the beginning of study which was approved by the Ethics Committee of the Faculty of Medicine, Hacettepe University, Ankara, Turkey (Approval number 431-1305). This study was supported by Hacettepe University Scientific Research Unit (Approval number 012DO6401002).

Sample

Minimum 377 individuals were calculated with the 80% power at the NCSS-PASS package while prevalence of anaemia in women of normal weight, overweight and obese women were estimated as 40%, 50%, 60%, respectively (9).

Exclusion criteria

Women having any of the following conditions that can effect body iron stores were excluded from the study: women aged under 20 years and over 49 years, pregnant, lactating, postmenopausal, underweight (BMI<18.5 kg/m^2), taking dietary iron supplements, with haemorrhage (clinical evidence in the previous 6 months), consumption of alcohol over 50 g/day, more than 3% changes of body weight or taking drugs which can effect body weight in the previous 3 months, with cancer and bariatric surgery history, gastrointestinal haemorrhage, inflammatory diseases, diabetes mellitus, bone, renal and liver failure, and gallbladder problems.

Data collection

Design of the questionnaire

In the study, a general questionnaire was applied to all women via a face-to-face interview. The questionnaire was validated, and designed to probe for detail on dietary nutrient intakes, physical activity levels and anthropometric status.

Anthropometric measurements

BMI calculations (10) and anthropometric measurements (11) were performed by a researcher dietitian using the criteria suggested by World Health Organisation (WHO). Bioelectrical impedance analyser (TANITA TBF 300, Tanita Corp., Tokyo, Japan) was used to measure body weight. Subjects were instructed to avoid food or liquid intake and vigorous exercise for 4 hours prior to the measurement, and asked not to wear any metallic objects during the measurement. Body height was measured using a tape measure while women standing without shoes, keeping their shoulders in a relaxed position, arms hanging freely and head in Frankfort horizontal plane.

All the anthropometric measurements of the subjects were measured three times and the mean values were obtained. Based on participants' calculated BMI values, women were classified as normal weight (BMI=18.5-24.9 kg/m^2), overweight (BMI=25.0-29.9 kg/m^2) and obese (BMI=≥30 kg/m^2) using criteria suggested by WHO (10).

Assessment of biochemical parameters

Biochemical indicators of iron status, including red blood cell count (RBC), haemoglobin, haematocrit, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), serum iron, total iron binding capacity (TIBC), ferritin, transferrin saturation and sTfR were analysed in all subjects. CRP and hsCRP were assessed to describe inflammation.

ID was defined as either 1) iron level <49 mcg/dL or 2) elevated total iron binding capacity (TIBC, 428 mcg/dL) and low transferrin saturation (TS<16%) (12).

After an 8 hours of overnight fast, blood samples were collected between 08.30-10.30 am. The bio-

chemical analysis of RBC, haemoglobin, haematocrit, MCV, MCH, and MCHC parameters were performed in the Izmir Bozyaka Training and Research Hospital Laboratories.

The serum was separated by centrifugation and stored at -20°C, and shipped on dry ice to Duzen Laboratory (accredited) in Ankara, where sTfR and hsCRP analyses took place. Transferrin saturation (TSAT) was calculated by using the formula (Serum iron/TIBC) × 100). sTfR was measured using Elisa assay (Biovendor, Czech Republic) and hsCRP was measured using a particle enhanced immunoturbidimetric assay (Roche, Germany).

Dietary assessment

The dietary intake of the participants was assessed by food consumption frequencies and 24-hour dietary assessment tool. Participants were asked to record all the foods and beverages consumed within two consecutive weekdays and one weekend day. A Nutrient Database (BeBiS, Ebispro for Windows, Germany; Turkish Version/BeBiS 7) was used to produce dietary data. Energy and nutrient intake data were compared with the Dietary Guidelines of Turkey (13, 14). Portion sizes were estimated with 2-dimensional food models and a food atlas including 3 to 5 portion size images of 120 foods (13, 15).

PA assessment

The participants asked to record their PA pattern by using a log method which captures the time individuals spend in a range of activity (sleeping, very light, light, moderate, high and very high). To determine energy expenditure at per activity type, duration of activity was multiplied by the PA ratio and PA levels. Population groups with less, similar or more than average PA were classified as sedentary (<1.40), light (1.40-1.60), moderate (1.70-1.99) or vigorous (2.00-2.40) PA status, respectively (16).

Statistical analysis

SPSS version 15.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Normality of the data distribution was determined with the Kolmogorov-Smirnov test. Descriptive analysis (mean±standard deviation, median, frequencies (%) and range) were performed. One way analysis of variance (ANOVA, for normally distributed variables) and Kruskal-Wallis test (for non-normally distributed variables) were used to compare continuous variables. According to Tukey or Conover's multiple comparisons tests, Pearson chi-square and analysis of variance were conducted to determine groups differences for overall factors. The odds ratios (OR, 95% confidence intervals) were calculated to test the iron deficiency risk in overweight and obesity. Two-sided p values were calculated and $p<0.05$ was noted as statistically significant.

Results

The socio-demographic data collected with questionnaires showed that older age, being married, being illiterate or literate, and higher number of pregnancies were all associated with higher prevalence of being overweight or obese. Overweight and obese women had lower income, and a higher number of people per household compared to normal weight individuals. No differences in menses density, regular menstruation and average duration of menses were observed between the study groups.

The biochemical and haematological results which are classified according to BMI values are presented in Table 1. Serum iron, haemoglobin and transferrin saturation levels of obese (65.4±33.7 mcg/dL, 12.6±1.3 g/dL, 17.4%±10.0, respectively) and overweight (73.8±36.4 mcg/dL, 12.7±1.3 g/dL, 19.4%±11.0, respectively) women were lower than in normal weight (85.6±37.5 mcg/dL, 13.0±1.2 g/dL, 23.8%±11.0, respectively) women ($p<0.05$). The white blood cell (WBC), CRP, high sensitive C- reactive protein (hsCRP) and sTfR levels of obese (7.3±1.6 mm^3, 0.7±0.6 mg/dL, 13.6±16.8 mg/L, 1.9±0.7 mcg/mL, respectively) and overweight (6.9±1.7 mm^3, 0.5±0.3 mg/dL, 2.4±2.6 mg/L, 1.7±0.9 mcg/mL, respectively) women were higher than in women with normal weight (6.4 ±1.5 mm^3, 0.2±0.1 mg/dL, 1.5±0.9 mg/L, 1.5±0.8 mcg/mL, respectively) ($p<0.05$, Table 1).

As shown in Table 2, 52.0% of obese and 49.7% of overweight women had a TSAT index below 16%. It was seen that 75.3% of those with CRP levels in the

top quartile were obese. It was also observed that there was a significant interaction among BMI, TSAT and CRP (p<0.05, Table 2).

In overweight and obese women, the prevalence of ID was significantly higher than in normal weight women. The prevalence of ID in the obese, overweight and normal weight women were 45.6%, 41.9% and 23.5%, respectively. A significant inverse relationship was found between iron deficiency and BMI (p<0.05) (Table 3).

Table 3 shows that obese and overweight women were 2.7 (95% confidence interval, Cl=1.8-4.1; p<0.001) and 2.32 (95% confidence interval, Cl=1.46-3.7; p<0.001) times more likely to be iron deficient than normal weight women.

PA levels according to BMI categories are described in Table 4. In total, 93.0% of obese women had a light activity level (Table 4). A significant relationship was found between BMI and physical activity levels (p<0.05).

Table 5 shows the dietary nutrient intake of women. Obese women had a significantly higher intake of iron (13.9±2.5 mg; p<0.001), than overweight (12.6±2.6 mg/day; p<0.001) or normal weight women (12.8±2.2 mg/day; p<0.001). Anthropometric findings were showed in Table 6.

Discussion

The study findings indicated that the prevalence of ID was higher in overweight and obese women than in women with normal weight. The prevalence of ID among obese, overweight and normal weight women were 45.6%; 41.9% and 23.5%, respectively. The results of our study suggests that in the study population, obesity was a strong negative predictor of iron status overweight and obese women were much likely to be iron deficient than women with normal weight (Table 3). Similar to this study, Cepeda-Lopez and colleagues (10) reported that obese women and their children had an increased risk of iron deficiency with odd ratios of 1.92 and 3.96, respectively. A potential explanation for the relation between obesity and impairment in iron status is that increased adipose tissue in obese women might increase the inflammatory activity (17, 18).

The serum iron, haemoglobin and transferrin saturation levels of obese and overweight women were lower than in normal weight women (p<0.05) (Table 1). The WBC, CRP and hsCRP levels of obese and overweight women were higher than normal weight individuals. These findings support the association between obesity and inflammation. sTFR was one of the parameters used as a definitive marker of iron deficiency as, it is not an acute-phase reactant (19). Freix-

Table 1. Haematological and biochemical parameters of iron status according to BMI classification

Biochemical parameters	BMI (kg/m²)			
	18.5-24.9 (n=170)	**25.0-29.9 (n=179)**	**≥30.0 (n=270)**	**p value**
Serum iron (mcg/dL)	85.6±37.5	73.8±36.4	65.4±33.7	<0.001*
TIBC (mcg/dL)	374.0±61.6	399.1±59.9	393.0±59.7	<0.001*
TSAT (%)	23.8±11.0	19.4±11.0	17.4±10.0	<0.001**
WBC (mm³)	6.4±1.5	6.9±1.7	7.3±1.6	<0.001*
Hb (g/dL)	13.0±1.2	12.7±1.3	12.6±1.3	0.002**
Ferritin (ng/mL)	16.7±14.1	14.9±12.8	16.3±13.9	0.288
CRP (mg/dL)	0.2±0.1	0.5±0.3	0.7±0.6	<0.001**
hsCRP (mg/L)	1.5±0.9	2.4±2.6	13.6±16.8	<0.001**
sTfR (mcg/mL)	1.5±0.8	1.7±0.9	1.9±0.7	<0.001**

**ANOVA test, p<0.001; ** Kruskal Wallis Test, p<0.001 - Analyses number is different for sTfR and hsCRP. Because they were measured from collected serum in Duzen Laboratory. TIBC: Total iron binding capacity; TSAT: Transferrin saturation, Hb: Haemoglobin; CRP: C-reactive protein; hsCRP: high sensitive C reactive protein; sTfR: Soluble transferrin receptor*

Table 2. Biochemical parameters of women according to BMI classifications

Biochemical parameters	18.5-24.9 (kg/m²)		25.0-29.9 (kg/m²)		≥30 (kg/m²)			
	n	C%	n	C%	n	C%	L%	p value
Iron deficiency								
No	130	76.5	104	58.1	147	54.4	38.6	<0.001*
Yes	40	23.5	75	41.9	123	45.6	51.7	
TSAT (%)								<0.001*
<16	41	24.1	89	49.7	140	52.0	51.9	
≥16	129	75.9	90	50.3	129	48.0	37.1	
CRP (quartile)								<0.001*
1. quartile	141	82.9	58	32.4	58	21.5	22.6	
2. quartile	23	13.5	50	27.9	51	18.9	41.1	
3. quartile	5	2.9	34	19.0	45	16.7	53.6	
4. quartile	1	0.6	37	20.7	116	43.0	75.3	

**Pearson chi-square; C %: Percentage of column, L%: Percentage of line*

Table 3. Odds Ratios (ORs) of iron deficiency according to BMI in women

Variable	Iron Deficiency					
	No		Yes		p value	Odds Ratio (OR)
	n	%	n	%		(95% CI)
Normal and Obesity					< 0.001*	2.70 (1.8-4.1)
18.5-24.9	130	76.3	40	23.7		
≥30.0	147	54.4	123	45.6		
Normal and Overweight					< 0.001*	2.32 (1.46-3.7)
18.5-24.9	130	76.3	40	23.7		
25.0-29.9	104	58.1	75	41.9		

**Pearson chi-square, # Iron deficiency defined as either serum iron <49 mcg/dL, or elevated total iron binding capacity>428 mcg/dL and low percentage transferrin saturation (<16%) (12).*

Table 4. Physical activity levels of women according to BMI classifications

PAL	BMI (kg/m²)						p value
	18.5-24.9		25.0-29.9		≥30		
	n	%	n	%	n	%	
<1.40 Sedentary	-	-	3	1.7	5	1.9	
1.40-1.69 Light activity	62	36.5	110	61.5	251	93.0	<0.001*
1.70-1.99 Moderate activity	108	63.5	66	36.9	14	5.2	

**Fisher Exact Test*

Table 5. Dietary intake of nutrients according to BMI classifications

Energy and nutrients	**BMI (kg/m²)**			
	18.5-24.9 (n=170)	**25.0-29.9 (n=179)**	**≥30.0 (n=270)**	**p value**
Energy (kkal/d)	1620.5±153.2	1916.4±232.3	2069.5±278.3	<0.001**
Protein (g/d)	65.0±11.3	65.0±12.0	69.0±11.2	<0.001*
Animal protein (g/d)	35.4±10.9	33.4±11.3	33.1±10.7	0.090*
Vegetable protein (g/d)	29.6±5.3	31.6±7.0	35.6±6.3	< 0.001**
Fibre (g/d))	23.9±5.2	24.4±5.7	26.4±5.5	< 0.001**
Vitamin C (mg/d)	157.6±70.3	169.5±64.8	180.8±66.9	0.002*
Calcium (mg/d))	753.1±182.2	799.5±208.1	802.2±182.5	<0.022*
Iron (mg/d)	12.8±2.2	12.6±2.6	13.9±2.5	<0.001*
Zinc (mg/d)	9.8±1.7	10.0±1.7	10.7±1.8	<0.001*

**ANOVA; **Welch ANOVA; $\bar{x}$±S: mean±standard deviation*

Table 6. Anthropometric measurements according to BMI classifications

Variables	**BMI (kg/m²)**			
	18.5-24.9 (n=170)	**25.0-29.9 (n=179)**	**≥30.0 (n=270)**	**p value**
Height (cm)	163±5.9	161±6.6	160±6.5	<0.001*
Weight (kg)	61.2±6.0	71.3±6.5	91.4±14.0	<0.001***
BMI (kg/m²)	22.8±1.6	27.4±1.4	35.4±4.8	<0.001*
BMR (kcal/day)	1404±75.4	1491±87.9	1665±148.7	<0.001**
Fat (%)	26.4±4.4	33.5±3.6	41.2±4.2	<0.001*
Fat mass (kg)	16.3±3.9	24.1±4.5	38.1±9.5	<0.001***
Fat free mass (kg)	44.7±2.9	47.2±2.9	53.1±5.3	<0.001***
Total body water (kg)	32.7±2.0	34.5±2.1	38.9±3.9	<0.001***

**ANOVA; **Welch ANOVA Tamhane; ***Kruskal Wallis Test; $\bar{x}$±S: mean±standard deviation*

enet and colleagues (20) reported that an increase in serum concentration of sTfR is associated with central obesity in men. In this study, overweight and obese women had higher sTfR, indicator of iron deficiency, compared to women with normal weight.

There are various studies indicate an association between obesity and iron deficiency in women (4,6, 21-24). Serum ferritin is a widely accepted marker to evaluate iron status. Yanoff and colleagues (6) and Lecube and colleagues (25) reported that serum ferritin levels accurately reflects depleted iron stores in the absence of inflammation. However in inflammatory conditions, such as obesity; ferritin levels are elevated as an acute phase reactant, hence ferritin is a

less effective indicator of iron bioavailability (26). Elevated serum ferritin concentrations have been shown positively associated with obesity in adults (27-36) and possibly in children (7-37). In this study, no difference was found in ferritin levels among women with different BMI categories (Table 1). This might be because we excluded the participants from study who might have had conditions that could effect body iron stores.

Recent studies have indicated inverse relationship between iron status and obesity (4, 6, 8, 21-24). However, in most of these studies, intake of dietary iron was not compared across same BMI categories and inflammation associated with obesity was not assessed by using the hs-CRP or CRP tests. In this study, obese women with a significantly higher dietary iron intake were compared with overweight and normal weight women (Table 5). However, in a study conducted by Yanoff et al. (6) daily dietary iron intake was not different in obese (16.0±5.9 mg/day; p=0.91) and non-obese subjects (16.1±5.9 mg/day, p=0.91). Similar to Yanoff's study, Menzie et al. (4) indicated obese participants to have higher intake of animal protein (63.6±34.5 vs 55.7±32.5 g/day; p<0.001) and more heme iron (3.6±2.8 vs 2.7±2.6 mg/day; p<0.001). Cepeda-Lopez et al. (10) reported similar dietary iron intakes in obese and normal weight individuals. The results of this study showed that obese women had significantly higher dietary intake of vitamin C and calcium compared to overweight and normal weight participants (Table 5). A significant relationship was found between BMI groups and intake of vitamin C and calcium (p<0.05). In contrast to this study, Menzie et al. (4) indicated that obese participants reported to consume less vitamin C and calcium than non-obese participants.

Vitamin C, which enhances the absorption of dietary non-heme iron, aids in the absorption of non-heme iron by increasing its bioavailability and reducing it to the ferrous state (38, 39). The results from Cook and co-worker's study (39) found that there was no significant difference in iron absorption with vitamin C intake between 51–247 mg/day. Moreover, in another study, the addition of 2000 mg vitamin C/day into the diet for 2 years did not alter iron stores (40). This study show that despite of higher iron and vitamin C intakes, obese and overweight women were more iron deficient. For this reason, there is a need for additional information on the actual role of dietary vitamin C in iron metabolism. When muscles are broken down during exercise, myoglobin, a heme protein found mainly in muscle, is released suggesting that higher levels of exercise could result in increased iron bioavailability (41). In this study, 93.0% of obese women had light PA levels (Table 4). Physical inactivity could be another factor associated with iron deficiency in obesity.

This study had several strengths. It has a large sample size which includes women with low and middle income. It uses CRP and hs-CRP assay, which provided an estimate of systemic inflammation as a potential underlying mechanism.

The limitations of this study were it was not possible to estimate iron bioavailability as three-day food records were self-recorded which might not completely reflect habitual iron intake, besides heme and non-heme iron intake were not measured.

Ultimately, this study showed that obesity related inflammation may lead to iron deficiency. Despite higher dietary iron and vitamin C intake, overweight or obese women had an increased prevalence of iron deficiency compared to women with normal weight. Iron deficiency was significantly associated with BMI and inflammation which was measured by CRP. These evidence suggests that, iron deficiency is a condition that should be carefully assessed in obese women.

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