The Relationship between Ferritin and Anemia Parameters in Females with Iron Deficiency Anemia and Inflammation

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Abstract

Objective: Iron deficiency is the leading cause of anemia worldwide, and measuring serum ferritin concentration is recognized as the gold standard test for iron deficiency anemia. In inflammation, however, hyperferritinemia occurs without evidence of iron overload. The aim of this study was to investigate the relationship between ferritin and other anemia parameters in female patients with a combination of iron deficiency anemia and inflammation.

Methods: A total of 314 girls and women with anemia (Hb <12.5 g/dL) were selected from users of primary health care centers in Shif port. The participants were divided into a study and a control group. The study group included females with anemia and inflammation, and the control group included anemia without inflammation. Complete blood count, serum ferritin, iron and hemoglobin concentration, hematocrit, mean cell volume, mean cell hemoglobin concentration, red cell distribution width, transferrin saturation, total iron binding capacity and C-reactive protein were measured by autoanalyzer and ELISA kits. The relationship between ferritin and hemostatic markers was estimated with Pearson's correlation coefficient and multiple linear regression models.

Results: There was a significant positive correlation between serum ferritin and serum iron concentration, hemoglobin, hematocrit and mean cell hemoglobin concentration before and after adjustment for age in both groups. A negative association between serum ferritin level and total iron binding capacity was also found in both groups.

Conclusions: We conclude that ferritin is a reliable noninvasive standard test to diagnose iron status in females with iron deficiency anemia even in the context of inflammation.

Keywords: Ferritin, Inflammation, Anemia.

(J Med J 2015; Vol. 49 (4):205-214)

Received	***Accepted***
Nov. 9, 2014	*Aug. 13, 2015*

Introduction

Anemia has been recognized as a contributing factor in social, economical and educational backwardness in developing countries, particularly among preschool-aged children and women[1,2]. The leading cause of anemia is iron deficiency[3]. In women, anemia

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is defined as a serum hemoglobin concentration below 12 mg/dL[4]; however, iron deficiency anemia (IDA) is diagnosed by measuring serum ferritin concentration[5,6]. Ferritin, an iron storage protein which contains 3000-4500 iron molecules[7,8], is found in all cells but is abundant in the liver, spleen and bone marrow[9]. Serum iron regulates the synthesis of ferritin. Reportedly, iron loading in the body increases ferritin synthesis in the liver and hence serum ferritin concentration[10].

Inflammation causes a type of anemia termed anemia of chronic disease (ACD), which usually occurs in association with inflammatory diseases[8,9]. The characteristics of ACD are similar to IDA; however, in ACD (but not in IDA) the concentration of ferritin, as an acute-phase inflammatory protein increases independent of serum iron level[8]. Inflammation reportedly increases hepcidin mRNA expression[11]. Hepcidin decreases iron release from reticuloendothelial cell (RECs) and from the gastrointestinal tract[12,13], and increases ferritin concentration in the liver and serum[14].

Several studies found a relationship between poverty, anemia and inflammation[1, 15]. In poor countries, inflammation can occur during infection with a microbial pathogen, including bacterial, viral, or yeast infections concurrent with iron deficiency anemia. In other words, in low-income settings IDA and ACD may tend to co-occur. Accordingly, the present study was designed to investigate the relationship between serum ferritin concentration and other anemia indices in females with concomitant IDA and inflammation. Additionally, we investigated the association between serum ferritin concentration and socioeconomic markers such as income, education, family size, internet usage and marital status of the participants.

Materials and Methods

This study was conducted between January and June 2009 at Kheibar Primary Health Care Center, Bushehr University of Medical Sciences and Health Care, located in Shif port in northeast Bushehr, Iran. We recruited 314 females with anemia (based on their medical records) who were seen at the Kheibar Primary Health Care Center. They were selected from eligible patients and who were willing to participate in the study.

The participants were invited to come to the primary health care center on the following morning in a letter delivered to their homes by the research team. The objectives of the study were explained and written, signed informed consent was obtained from each participant. Then a blood sample was obtained from each participant and transferred to the laboratory. The criteria for inclusion in the study were: (1) hemoglobin level < 12.5 g/dL, (2) female gender, and (3) ferritin < 30 ng/dL[16]. iron deficiency is defined by ferritin concentrations <25 μg/L in adults and <15 μg/L in children[17]. However, ferritin concentration increases in the presence of inflammation which is potentially prevalent in developing countries[1, 15]. Therefore WHO has emphasized that serum ferritin should be measured with acute-phase CRP in order to account for inflammation, and this cutoff value should be increased to 30 μg/dL[16]. Potential participants were excluded if they were pregnant, had been breastfeeding during the previous 3 months or longer, had an abortion, were in the postpartum or post-hysterectomy period, or had been taking iron tablets during the previous 6 months. The presence or absence of inflammation was determined by measuring serum C-reactive protein (CRP). Based on the CRP results, participants with anemia were assigned to one of two groups. Those with a positive CRP

result were assigned to the study group, and those with a negative CRP result were assigned to the control group. A validated questionnaire that included items on economic, educational and social characteristics was completed during an interview with a member of the research group.

The study protocol was approved by the Ethics Committee of Bushehr University of Medical Sciences, and the research reported here was carried out in accordance with the principles of the Declaration of Helsinki as revised in 2000.

Measurement of serum and plasma parameters

Venous blood samples were drawn between 8:00 and 9:00 hours. The blood samples were divided into two tubes; one tube contained 5 mL blood which was centrifuged at 3000 g for 15 min at 4 °C to determine iron, total iron binding capacity (TIBC), ferritin and CRP. The second tube, which contained EDTA, was used for a 1.5-mL blood sample to determine complete blood count (CBC), hemoglobin concentration, hematocrit (Htc), mean cell volume (MCV), mean cell hemoglobin concentration (MCHC) and red cell distribution width (RDW). All samples were kept at 4 °C until analysis. All analyses were carried out at the laboratory of the Persian Gulf Tropical Medicine Research Center on the day of blood collection.

One hematologist was assigned to analyze and record the hematological data. Anemia was defined as hemoglobin <12.5 g/dL. Microcytic anemia was defined as MCV <80 fL. Iron deficiency anemia was defined as anemia with a ferritin level ≤30 ng/dL[16]. Serum ferritin was measured by enzyme-linked immunosorbent assay (ELISA) (Stat Fax, Kit: Monobind, Palm City, FL, USA). Red cell distribution width was calculated as the standard deviation of MCV / means MCV× 100 and was reported as a percentage[18]. The laboratory values were obtained with a Sysmex-1000 autoanalyzer (Sysmex, San Diego, CA, USA). C-reactive protein was measured with a Prestige autoanalyzer (Bionik, Swadlincote, Derbyshire, UK).

Statistical analysis

The distribution of variables was studied with probability plots and the Shapiro-Wilks test. The relationship between ferritin and hemostatic markers was determined by calculating Pearson's correlation coefficient. Partial correlation analyses were done with adjustment for age. Multiple linear regression models were also used to determine the association between ferritin and hemostatic markers. Agreement between pairs of tests was calculated with the kappa statistic. To identify differences between the two groups in ferritin levels and serum anemia parameters, we used the general linear model with adjustment for age. A value of $p<0.05$ was accepted as significant.

All statistical analyses were done with an IBM computer using the SPSS 15 statistical software package (SPSS Inc., Chicago, IL, USA).

Results

A total of 314 females were evaluated. Of the 314 participants with anemia in our sample, 87 (27.7%) had inflammation. The mean age (±SD) was 29 (±14) years. The age distribution in our sample was 10.2% between 2 and 10 years, 16.2% between 10 and 20 years, 29.3% between 20 and 30 years, 23.6% between 30 and 40 years, 11.5% between 40 and 50 years, 6.4% between 50 and 60 years, and 2.2% >60 years. Table 1 shows the basic

characteristics of the participants. The average family income was less than 3 US$/day in 78%, between 3 and 6 US$/day in 32% and more than 6 US$/day in 4% of the participants. The educational level among participants was 44% illiterate, 40% up to elementary school, 33% up to high school and 11% up to diploma. The employment rate of participants was 2.2%.

Table 1. Baseline characteristics of the women (n=314). Means ±SD

Characteristic NO	**Anemic women (n=314) mean**	**SD**
Age (years)	29.00	14.625
BMI (kg/m□^{2})	23.41	1.62
Hb (g/dL)	11.29	1.04
RBC (x10^6/µL)	4.25	0.34
Fe (µg/dL)	62.33	16.29
Ferritin (ng/dL)	13.29	5.46
Htc (%)	35.98	3.98
TIBC (µg/dL)	429.28	58.07
Transferin saturation (%)	15.14	5.49
MCV(fL)	73.63	7.26
MCHC (%)	25.86	4.23
RDW (%)	14.44	2.36
CRP (µg/dL)	3.08	3.16

BMI; body mass index, Hb; hemoglobin, RBC; red blood count, Fe; iron, Htc; hematocrit, TIBC; total iron binding capacity, MCV; mean corpuscular volume, MCHC; Mean corpuscular hemoglobin concentration, RDW; red distribution width, CRP; C reactive protein.

Fifty-five percent of the participants had one child, 2% had two; 23% had three and 12% had four or more children. Only 34% of the women reported using the internet. Forty-six percent of them were married, 35% were single and 33% were widows. These characteristics did not differ significantly between the two groups (data not shown).

A significant positive correlation was found between ferritin level and educational level, and a negative association was observed between ferritin concentration and the number of children.

Bivariate correlation analysis showed a positive correlation between serum ferritin level and serum iron concentration, hemoglobin, hematocrit and mean cell hemoglobin concentration in participants with anemia and inflammation. An inverse association was found between serum ferritin level and TIBC in the study group (Table 2). This significant relationship persisted after adjustment for age in the partial bivariate correlation analysis (Table 2). There was no significant correlation between serum ferritin level and Red Blood Cell (RBC), MCHC,

RDW or transferin saturation in the study group. In the control group ferritin level was predictive of the level of Hb, RBC, Htc, RDW and transferin saturation before and after adjustment for age (Table 2).

Table 2. Unadjusted, and age adjusted correlation between ferritin and serum anemia parameters in women

	Study group				Control group			
	Unadjusted		Adjusted		Unadjusted		Adjusted	
	r	p	r	p	r	p	r	
Age (year)	-0.04	0.73			-0.03	0.57		
BMI (kg/m□2)	.040	0.72			-0.01	0.80		
Fe (µg/dL)	0.82	0.001	0.83	0.001	0.81	0.001	0.81	0.001
Hb (g/dL)	0.23	0.04	0.20	0.05	0.41	0.001	0.40	0.001
RBC (x106/µL)	0.06	0.62	0.08	0.51	0.38	0.001	0.39	0.001
Htc (%)	0.26	0.03	0.25	0.04	0.16	0.01	0.17	0.007
TIBC(µg/dL)	-0.76	0.001	-0.76	0.001	-0.72	0.001	-0.71	0.001
MCV (fL)	0.21	0.08	0.18	0.15	-0.03	0.59	-0.04	0.45
MCHC (%)	0.25	0.04	0.25	0.04	0.02	0.69	0.02	0.68
RDW(%)	-0.13	0.3	-0.10	0.41	-0.74	0.001	-.074	0.001
Transferin (%)	0.07	0.55	0.13	0.28	0.85	0.001	0.85	0.001
Children	-0.18	0.15	-0.03	0.80	-0.16	0.009	-0.08	0.19
Education	0.25	0.04	0.23	0.05	0.18	0.005	0.15	0.01
income	0.09	0.45	0.09	0.47	0.03	0.61	0.02	0.71

BMI; body mass index, Hb; hemoglobin, RBC; red blood count, Fe; iron, Htc; hematocrit, TIBC; total iron binding capacity, MCV; mean corpuscular volume, MCHC; Mean corpuscular hemoglobin concentration, RDW; red distribution width.

Table 3. Multiple linear regression analysis for the association between ferritin (independent variable) and Fe as well as Hg(dependent variables) in women

		Ferritin Study group		Ferritin Control group	
		β	p	β	p
Fe	Age-Adjusted	2.3	0.001	0.30	0.001
Hb	Age-Adjusted	0.03	0.05	2.18	0.001
Htc	Age-Adjusted	0.12	0.03	0.49	0.001
TIBC	Age-Adjusted	-6.42	0.001	-0.07	0.001

Fe; iron, Hb; hemoglobin, Htc; hematocrit, TIBC; total iron binding capacity

In the multiple regression analyses, serum ferritin level showed a significant correlation with serum iron (β=2.3, p< 0.001), Hb (β=0.03, p=0.05), Hct (β=0.12, p=0.03) and TIBC (β=-6.42, p>0.001) after controlling for age in the study group (Table 3) (Figure 1, Figure 2). In the control group, a significant association was observed between serum ferritin and serum iron, Hb, Hct and TIBC after controlling for age (Table 3). The level of agreement between the ferritin test and iron test in the study group was 0.82 (p<0.001).

The unpaired *t* test showed a significantly higher mean concentration of ferritin, iron, Hb, Hct and RDW in the study group compared to the control group. However, there were no

significant differences in serum MCV, MCHC, RBC or transferrin saturation between the two groups (Table 4).

Table 4. Differences of variables between two groups

Group / Characteristics	Study group	Control group	P value
Age (years)	32.20±15.08	27.99±14.91	0.13
BMI (kg/m□^{2})	23.41±1.46	23.41±1.65	0.98
Hb (g/dL)	11.83±0.69	11.18±1.06	0.001
RBC (x10^6/µL)	4.34±0.29	4.23±0.34	0.07
Fe (µg/dL)	66.88±15.65	61.41±16.31	0.06
Ferittin (ng/dL)	16.15±5.52	14.38±6.4	0.04
Htc (%)	67.86±14.63	56.76±20.85	0.003
TIBC (µg/dL)	416.22±48.00	431.93±59.68	0.13
Transferin saturation	16.58±5.33	14.85±5.50	0.08
MCV(fL)	72.61±9.15	73.84±6.83	0.35
MCHC (%)	25.16±3.41	26.01±4.37	0.27
RDW (%)	13.59±1.77	14.62±2.43	0.01

BMI; body mass index, Hb; hemoglobin, RBC; red blood count, Fe; iron, Htc; hematocrit, TIBC; total iron binding capacity, MCV; mean corpuscular volume, MCHC; Mean corpuscular hemoglobin concentration, RDW; red distribution width.

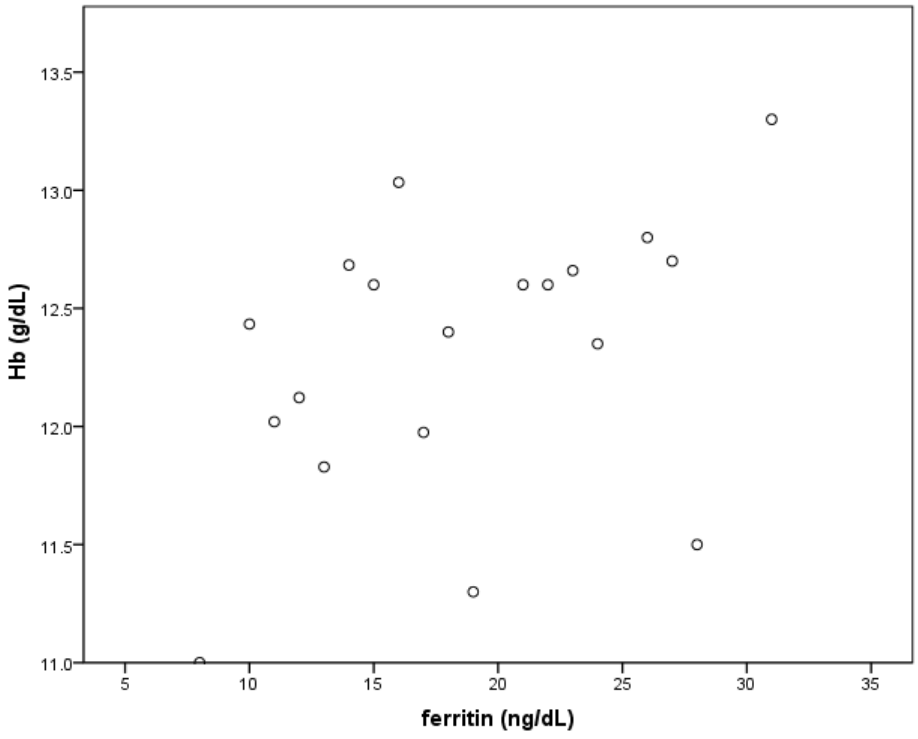

Figure 1. Scatterplot diagram of linear regression between serum ferritin and hemoglobin in females with anemia and inflammation

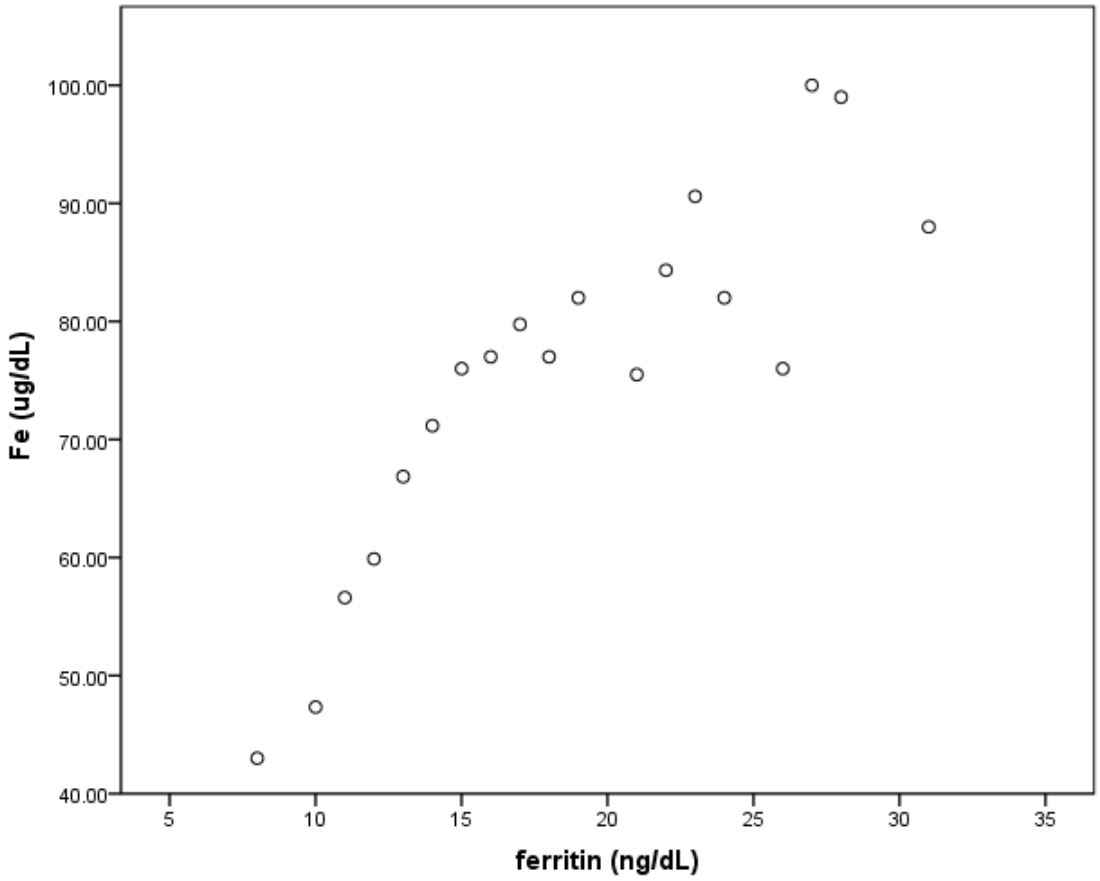

Figure 2. Scatterplot diagram of linear regression between serum ferritin and iron in females with anemia and inflammation

Discussion

The participants in this study were from low-income and illiterate sectors of the population. The results showed a significant inverse correlation between serum ferritin concentration and educational level in both groups (with or without inflammation). Moreover, there was a nonsignificant association between income (economic status) and serum ferritin concentration, hemoglobin level and CBC in both groups. Poverty is apparently a leading cause of IDA and inflammation in underdeveloped regions. In such areas, inflammation can occur during infections with pathogenic microorganisms. As in the present work, several earlier studies reported a significant relationship between poverty, anemia and inflammation[1, 2].

Ferritin measurement is the gold standard for diagnosing iron deficiency. The results of several studies have shown that ferritin is the best predictor of iron deficiency even in individuals who have inflammation (high CRP)[19]. The present study also detected a significant relationship between serum ferritin concentration and iron, Hb, Hct and TIBC in participants with anemia and inflammation. In contrast, several reports found that inflammation negated the correlation between serum ferritin and other anemia parameters[19-21]. Nemeth et al. and Ganz et al. reported that inflammatory cytokines such as interleukin-6 can rapidly induce hepcidin synthesis, which in turn promotes iron deposition in RECs by

favoring ferritin synthesis[12, 13]. In contrast, some studies have shown a transitory effect of inflammation on ferritin status, an effect which reverts after a short time[22]. This temporary impact was seen in turpentine-induced chronic inflammation mice, in which hepatic hepcidin mRNA expression increased during the early period and then returned to normal[23]. In addition, it was reported that in ACD mice with experimentally transplanted melanoma tumors, hepatic hepcidin mRNA expression increase only in the early period[24]. Fry et al. reported that after primary hepcidin gene expression due to inflammation in dogs, hepcidin expression decreased after experimentally induced nutritional iron deficiency[25]. Iron deficiency thus appears to suppress ferritin synthesis even in the context of inflammation. Vokurka et al. concluded that iron deficiency was the reason for decreased hepcidin gene expression after a primary increase in ACD mice[24].

The other explanation for decreased ferritin in IDA in the context of inflammation is that upregulation of hepcidin and the resulting increase in ferritin production is not the only pathway that inflammation uses to decrease serum iron. In chronic inflammation, a large part of iron is absorbed through the macrophage membrane by divalent metal transporter 1 (DMT1)[26]. Interferon-γ and tumor necrosis factor-α upregulate the expression of DMT1. In addition, these proinflammatory cytokines deposit iron in macrophages by decreasing the expression of ferroportin[27]. Accordingly, the main iron storage depots, i.e. the liver and bone marrow macrophages, use different pathways for iron storage: the former acts through ferritin production and the later functions through macrophage deposition. Consequently, in inflammation the organ responsible for iron storage differs. Therefore in inflammation, iron may be deposited in macrophages, where it is not associated with the liver. Hypothetically, therefore, the induction of ferritin synthesis is not the only pathway inflammation uses to decrease serum iron.

In the present study serum ferritin concentration was higher in the study group than the control group. WHO has reported that the cutoff point to diagnose depletion of iron reserves is a serum ferritin level less <10 mg/L. WHO has also emphasized that serum ferritin should be measured with acute-phase CRP in order to account for inflammation [28], and this cutoff value should be increased to 30 μg/dL[16].

Nel et al. suggested using greater cutoffs for ferritin than the WHO criteria[29]. The discrepancies between different reports regarding the serum ferritin cutoff value in inflammation can be attributed to variations in the clinical manifestations, which may in turn be related to the duration or severity of inflammation or to the differential expression of various proinflammatory cytokines that induce the inflammatory response[30].

A potential limitation of this study is that we did not estimate the sensitivity and specificity of the ferritin test to diagnose anemia in either group. The gold standard to determine sensitivity and specificity is bone marrow aspiration, which is an invasive procedure for which appropriate resources were not available[31].

Conclusions

We conclude that ferritin is a reliable noninvasive standard test to diagnose iron deficiency and iron deficiency anemia in females even in the context of inflammation.

Bone marrow aspiration remains the gold standard to predict iron deficiency, but is an invasive, costly and complicated procedure.

Acknowledgments

We thank K. Shashok (AuthorAID in the Eastern Mediterranean) for improving the use of English in the manuscript.

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العلاقة بين مستوى الفيريتين ومعالم فقر الدم بسبب عوز الحديد في الإناث اللاتي يعانين من الأمراض الإلتهابية

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الملخص

يعد عوز الحديد السبب الرئيس لفقر الدم في جميع أنحاء العالم، كما يعد قياس مستوى الفيريتين في مصل الدم المعيار الذهبي لتشخيص هدا المرض. ولكن في حالات الالتهاب، فإن مستوى الفيريتين يرتفع بدون وجود دليل على تكدس الحديد في الجسم.

لذا؛ كان الهدف من هذه الدراسة، البحث عن العلاقة مابين مستوى الفيريتين ومعالم فقر الدم الأخرى في الإناث اللاتي يعانين من فقر الدم الناتج عن عوز الحديد وكذلك من الأمراض الإلتهابية.

طريقة ومواد البحث: تم اختيار 314 من البنات والنساء اللاتي بعانين من فقر الدم (هيموجلوبين /12.5 <ديسيلتر) من مرتادي المراكز الطبية الأولية في ميناء شيف. تم تقسيم المرضى إلى مجموعتين: مجموعة الدراسة وتتكون من مرضى فقر الدم وأحد الأمراض الإلتهابية، ومجموعة المراقبة وتتالف من مرضى فقر الدم بدون أية أمراض إلتهابية. تم قياس تعداد الدم الكامل، الفيريتين، الحديد، الهيموجلوبين، الهيماتوكريت، معدل حجم الخلايا، معدل تركيز الهيموجلوبين الخلوي، عرض الخلايا الحمراء، الترانسفيرين، قدرة ارتباط الحديد الكلية، وكذلك البروتين التفاعلي ج، وذلك باستعمال المحلل التلقائي وعدة اليسا. تم تحديد العلاقة بين مستوى الفيريتين وعلامات الدم الأخرى باستعمال معامل ارتباط بيرسون وكذلك نماذج الانحدار الخطي المتعدد.

النتائج: كان هناك علاقة إيجابية مهمة مابين مستوى الفيريتين وتركيز الحديد في مصل الدم وكذلك مستويات الهيموجلوبين والهيماتوكريت ومعدل تركيز الهيموجلوبين الخلوي، وذلك قبل وبعد التعديل بالنسبة للعمر في المجموعتين. وكانت هناك علاقة سلبية ما بين مستوى الفيريتين وقدرة ارتباط الحديد الكلية ما بين المجموعتين.

الاستنتاج: نستنتج أن الفريتين هو فحص معياري موثوق لتشخيص حالة الحديد في الإناث اللاتي يعانين من فقر الدم الناتج عن عوز الحديد حتى بوجود الأمراض الإلتهابية.

الكلمات الدالة: فيريتين، إلتهاب، فقر الدم.