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Evaluation of Iron Status in Cats with Hypertrophic Cardiomyopathy with and without Congestive Heart Failure

Seyed Farzin Seyednejad, Dariush Shirani*, Saeid Bokai[2], Seyed Mehdi Nasiri[1]

[1]**Department of Internal Medicine, Faculty of Veterinary Medicine, University of Tehran, Tehran, Tehran, Iran**

[2]**Department of Epidemiology and Zoonoses, Faculty of Veterinary Medicine, University of Tehran, Tehran, Iran**

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ABSTRACT

BACKGROUND: All organisms need iron for their life and metabolic activity, and the healing process of patients depends on this element. Hence, its deficiency can negatively affect patients' quality of life and lead to disorders. Although Iron deficiency is proved as an important co-morbidity in human and canine

* **Corresponding author:** Dariush Shirani, Department of Internal Medicine, Faculty of Veterinary Medicine, University of Tehran, Tehran, Iran. P. O. Box: 14155-6453, Tehran, Iran. Tel: +98 21 61117149, Fax: +982166933222, email: dshirani@ut.ac.ir

patients with heart failure, there is also no published research on the role of iron in feline hypertrophic cardiomyopathy.

OBJECTIVES: The goal of this research was to determine and compare iron status of cats with hypertrophic cardiomyopathy with and without congestive heart failure.

METHODES: Based on laboratory, radiographic and echocardiographic findings, 45 client owned cats were divided into three groups: control, hypertrophic cardiomyopathy (HCM) without congestive heart failure, and hypertrophic cardiomyopathy with congestive heart failure. Iron concentration, ferritin, total binding-iron capacity (TIBC), and serum transferrin saturation percentage were measured and compared on all cats. Statistical nonparametric testing was used to analyse the data.

RESULTS: No groups illustrate any statistically significant difference for iron concentration (P=0.3), ferritin concentration (P=0.853), TIBC (P=0.1), and TSAT (P=0.639). The highest iron concentration, and the lowest transferrin level and lowest the transferrin saturation percentage were observed in the HCM group with congestive heart failure. Also, cats without congestive heart failure had the lowest TIBC compared to other groups.

CONCLUSIONS: Unlike previous studies in dogs and humans, our study did not show a significant difference between the iron status in cats with hypertrophic cardiomyopathy.

KEYWORDS: Hypertrophic Cardiomyopathy; Iron deficiency; Iron status; Feline

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Introduction:

Iron deficiency anemia in dogs and cats is usually caused by decreased intake, impaired absorption, or chronic bleeding (Dev and Babitt, 2017; Naigamwalla *et al*., 2012). The most common type of anemia following iron deficiency is inflammatory anemia (after chronic diseases). Various mechanisms may be responsible for this type of anemia, including alterations in iron homeostasis,

erythroid progenitor cell proliferation, erythropoietin synthesis, and reduced red blood cell life cycle. Furthermore, the production of cytokines such as interferon omega, tissue necrosis factor, interleukin 1, interleukin 6, and interleukin 10 is responsible for changes in the body's access to iron (McCown and Specht, 2011)

Unlike the various ways in which iron enters the heart cells, cardiomyocytes have only one way to release iron. This makes heart cells prone to iron accumulation (Kazory and Ross, 2009). A study on mice showed that by inhibiting the removal of iron from the heart, a deadly dilated cardiomyopathy occurs and contrary to systemic iron levels, iron levels in heart cells increased (Opasich *et al*., 2005). A significant factor in the auto-regulation of systemic iron and iron in cardiomyocytes is Hepcidin, which is produced by cardiomyocyte. Unlike systemic hepcidin, cardiac cell`s hepcidin levels increase due to iron deficiency (to maintain intracellular iron) (Van der Meer *et al*., 2004). Certain drugs (angiotensin-converting inhibitors and antiplatelet drugs), dietary iron deficiency, insufficient intestinal absorption of iron (due to inflammation of the intestinal mucosa), and increased hepcidin expression could be the cause of absolute iron deficiency in the context of heart failure (Von Haehling *et al*., 2019). The prevalence of iron deficiency in dogs with chronic mitral valve disorders with no congestive symptoms is reported to be 18.5% (Savarese *et al*., 2018) Moreover, Iron deficiency has been proven in human patients with acute heart failure and has been shown to improve clinical signs with intravenous iron administration (Anker et al., 2009; Kang *et al*., 2017).

If the causes of left ventricular hypertrophy, such as systemic hypertension, hyperthyroidism, and congenital aortic stenosis, are ruled out in tests, primary hypertrophic cardiomyopathy (HCM) is diagnosed by an increase in heart size due to ventricular hypertrophy (Fox, 2003). It is the most prevalent cardiac disease in cats with a prevalence of 14-16% (Paige *et al*., 2009). Arterial thromboembolism, congestive heart failure, left atrial dilatation, systolic dysfunction of the atrium and

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left ventricle, and left ventricular hypertrophy are various prognostic factors for this disease that have been reported so far (Payne *et al*., 2015; Roderick *et al*., 2017)

In previous studies, measurement of iron and its associated factors in cats with chronic renal failure and chronic gastrointestinal disease showed that these patients had functional iron deficiency (Hunt and Jugan, 2021; Javard *et al*., 2017).

Based on what we know, there is no study on iron levels in cats with cardiomyopathy. Here, we compare the iron levels of cats with HCM with and without congestive heart failure and evaluate the possible relationship between the levels of iron and its related factors with the occurrence of HCM.

Material and Methods

This cross-sectional observational study included cats owned by clients who were presenting to small animal hospital between September 2019 to April 2020. An complete physical examination by a veterinarian was performed for each individual cats. Obtaining of blood was done with informed owner permission. We enrolled 45 client-owned cats and divided them into three equal groups: 1. Control, 2. HCM with congestive heart failure (A) and 3. HCM without congestive heart failure (B) (15 cats per group). Cats who had client were included as a control group if their physical examination were unremarkable Additionally, there is no dental disease, no history of underlying illness, and no clinical or biochemical abnormalities on a previous CBC and biochemistry tests within recent 30 days. Except for worming and vaccination, these cats were not being treated with any medication.

Hypertrophic cardiomyopathy patients had one of the clinical signs of arrhythmia, murmur, tachypnea, muffle heart sound, cardiogenic pulmonary congestion (edema) or pleural effusion. Thoracic radiography and echocardiography were performed for suspected cats and based on the results; cats with HCM echocardiographic characteristic (thickness of left ventricular wall or interventricular septum greater than 6 mm) were included in the patient group. Radiographic and echocardiographic manifestations of cardiogenic pulmonary edema (alveolo-interstitial lung pattern) or pleural effusion

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with moderate to severe left atrial dilatation (left atrium to aorta ratio greater than or equal to 1.8) or furosemide-responsive tachypnea associated with moderate to severe left atrial dilatation (ratio of the left atrium to aorta greater than or equal to 1.8) was considered congestive heart failure (Luis Fuentes *et al*., 2020; Rauch *et al*., 2020).Exclusion criteria included evidence of systemic hypertension (systolic blood pressure greater than 180 mmHg or greater than 160 mmHg with ocular manifestation), hyperthyroidism, acromegaly, and aortic stenosis. Other exclusion criteria were the presence a systemic illness (kidney, liver and encocrinopathies), previous supplementation with iron, recent blood transfusion. A Doppler ultrasound device (EICKEMEYER) were used to measure blood pressure.

The collection of blood samples an iron panel and a full complete blood count was done at the same time. 1 milliliter of blood for CBC was obtained in a tube with EDTA and the test was done with an auto analyzer (Celtac alpha, MEK-6550J/ K). 5 milliliters of whole blood were drawn and then collected in a tube separates serum and immediately centrifuged in order to determine the biochemical test, serum iron concentrations, total iron binding capacity (TIBC), and ferritin concentrations. The samples were separated, frozen and preserved at 80°C. Biochemical tests, serum iron concentration and TIBC were evaluated routinely (Pars Azmoun Company Kit) by Vitalab Selectra E auto analyzer. Ferritin concentrations were evaluated using a quantitative enzyme linked immunosorbent assay using anti-ferritin monoclonal antibodies in a sandwich arrangement (Gest *et al*., 2015). Transferrin saturation percentage (TSAT) was estimated according on this information as [serum iron (mg/dL)/TIBC(mg/dL)]. MyBioSource kit (MBS705772 made in the USA) was used through the Immunoassay method to measure plasma thyroxine levels in healthy cats older than 7 years and all patient cats.

In order to statistically analyze the data, SPSS software (version 18) was used. $P \leq 0.05$ was considered statistically significant. After confirming the normality of the data by the Shapiro-Wilk test ($P>0.05$), an ANOVA test was used to evaluate the differences between study groups. The Kruskal-Wallis test for iron

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and ferritin and unilateral analysis of variance for TSAT and TIBC were performed, as the distribution in these groups was not normal.

Results

As a control group, fifteen cats were investigated. The mean age (based on the month) was 89.73 months (range 60-146). Of 15 cats of the this group, 10 were male and included Persian (12), DSH (2), and Siamese (1) breeds. For inclusion in the group, fifteen cats were part of the group A. The mean age was 108.80 months (range 65-164). 10 of the 15 cats in the group of patients with congestive heart failure (Group A) were male and included Persian (9), DSH (3), Himalayan (2), and Maine Coon (1) breeds. Also, the mean age of group B was 101.66 months (range 67-154). Of the 15 cats in the group of patients without congestive heart failure (Group B), 11 were male and included Persian (11), DSH (3), and Himalayan (1) breeds. The age difference between these three groups was insignificant. (*P>0.05*)

Tables 1 and 2 illustrate radiographic and echocardiographic findings of three groups. Hematocrit, heart rate and blood pressure of three groups were recorded. All cats in all groups were nonanemic with a hematocrit greater than 28.7%. One cat in control group and one in group A had tachycardia without arrhythmia and they were considered due to stress. No significant difference was found for hematocrit (P=0.077), heart rate (P=0.371) and blood pressure (P=0.057).

Figure 1 and 2 shows the results of iron and ferritin measurements. These two values did not follow the normal distribution. For iron (P=0.3) and ferritin (P=0.853), there were no statistically remarkable difference between any of the groups. Iron concentration range was 55-143, 34-148, and 55-148 µg/dL for control group, group A, and B, respectively.[1] All cats had normal iron concentration. Ferritin concentration range was 137-624, 129-580, and 105-505 ng/mL for control group, group A, and B, respectively.[2] In the control group one cat had an elevated level of of 624 ng/mL. Three cats from group

[1] Iron reference interval: 33-157 µg/dL
[2] Ferritin reference interval: 90-300 ng/mL

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A showed increased ferritin concentration, including: 490, 524, and 580 ng/mL and two cats in the group B had increased ferritin concentration of 485 and 505 ng/mL.

Figure 3 and 4 shows the results of TSAT and TIBC measurements. These two indices followed the normal distribution, and after analysis by ANOVA test, among study groups, no observable differences were found (*P>0.05*). TIBC concentration range was 198-450, 165-345, and 203-395 for control group, group A, and B, respectively (P=0.1).[3] Only one cat in each groups had increased TIBC. Also, one cat in group A had decreased TIBC concentration of 165 µg/dL. Transferrin saturation percentage range was 15-49, and 18-46 for control group, group A, and B, respectively.

Discussion

In human patients with severe heart failure, iron deficiency has been described (Hunt and Jugan, 2021). Improvement of clinical signs with iron supplementation in these patients has also been demonstrated (McCown and Specht, 2011; Acierno *et al*., 2020). According to the present study, no significant relationship was seen in the iron panel among all groups.

Mean ± standard deviation of TIBC was the lowest for group A (268.27±15.16 versus 311±11.33 and 314.07± 21.19 for group B and control, respectively). One of the most common causes of decreased TIBC in patients with chronic diseases is the presence of inflammatory processes. The presence of a congestion and an inflammatory process in the group (A) cats may be the reason for the low TIBC compared to other groups. According to previous studies in cats with renal failure and the significant difference between the patients and the control group, other factors may have been effective in reducing this value in HCM cats (Gest *et al*., 2015).

Decreased appetite and decreased gastrointestinal absorption of iron due to inflammation of the gastrointestinal tract are the main reasons of iron concentration level. No significant difference was detected for iron concentration among all groups; nevertheless, Mean ± standard deviation of iron

[3] TIBC reference interval: 169-325 µg/mL

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concentration was the lowest for group A (76±9.36 versus 90.6±8.28 and 92.13± 6.91 for group B and control, respectively. In our study group A cats presented in acute phase of disease and they had no appetite at least for a day. It may the cause of decreasing iron concentration in the more severe phase of this disease. With increasing erythropoiesis following a decrease in intracellular iron, serum iron decreases, so it is not possible to obtain accurate information about the total amount of iron in the body just by evaluating the level of serum iron. Since iron concentration is regarded as being objectively nonspecific, it should not be applied to evaluate total iron's body content. Based on our research, no difference was found for iron concentration among any of the cats populations which confirms its incompetence to determine total iron body stores.

Despite no significant difference among groups, the highest Mean ± standard deviation of ferritin concentration was observed in group A. The best way to identify iron deficiency in humans without performing bone marrow biopsy is to measure ferritin concentration, has a strong association with total body stores. Serum ferritin levels, are the best predictor of whole iron body stores in cats when measured in conjunction with serum iron levels (Gest *et al*., 2015). No association was detected between tissue iron stores and total iron binding capacity or serum iron concentration (Andrews *et al., 1994*). Pro-inflammatory cytokines such as IL-1, IL-2 and TNF-α enhance the release of ferritin which is acute phase protein. (McCown and Specht., 2011). Increased ferritin concentration was observed in 63.2% of cats with inflammatory disease (Freedman *et al*,. 1983). Three cats in control group had increased ferritin concentration. All imaging and laboratory findings were normal in these cats and no historical evidence of systemic disease were described; thus, the significance of these findings are unclear.

It is difficult to distinguish between true and functional iron deficiency. A decrease in iron and ferritin along with an increase in total iron-binding capacity is defined as true iron deficiency, while in functional

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iron deficiency, ferritin is normal or even increased, and iron-binding capacity is reduced (Anker *et al*., 2009; Bohn, 2013)

According to the present study, functional iron deficiency can be seen more in the advanced stages of heart disease, such as congestive heart failure, than in the early stages of the disease and the control group.

TAST is a calculated value according to TIBC and provides a determination of transferrin amount has bound iron. Due to insufficient iron preserves, TSAT is normal to decreased in an inflammatory disease related anemia and it is often reduced in absolute iron deficiency anemia. Although there is no reported reference index for cats, in human patients, a normal range is between 25 and 45 percent. Usually, due to insufficient iron storage and anemia, the TSAT index decreases in absolute iron deficiency. As iron content in group (A) was lower than the other two groups, transferrin saturation was also reduced.

Functional iron deficiency has been demonstrated in cats and dogs with renal insufficiency, and the use of injectable iron supplements (iron dextran 50 mg/cat, intramuscular) is recommended. The injectable form of the drug is preferable to the oral form (Payne *et al*., 2015). With this in mind, one study could examine the effects of iron intake in cats and dogs with congestive heart failure.

The results obtained may have been affected by some limitations in our study. Firstly, no described gold standard for noninvasive estimation of iron status in cats in order to diagnosis of iron deficiency exists. Storage of iron in feline liver and spleen have been studied; however, it is not possible due to invasive method of evaluation. The Second limitation to this study was the number of samples. More accurate results can be obtained with a larger statistical population. Also, in this study, acute phase proteins such as hepcidin and SAA were not measured. Measurement of these factors can accurately show the inflammatory process's effect on the iron panel. Moreover, using symmetric dimethylarginine (SDMA) could more definitively rule out kidney disease.

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In this study, despite the lack of significant differences between patient and control groups, it can be concluded that there is functional iron deficiency in patients. More studies to assess the effect of iron on improving the clinical condition are warranted.

Conflict of Interest Declaration

The authors declare they have no conflicting interests.

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مطالعه پانل آهن گربه‌های مبتلا به کاردیومیوپاتی هیپرتروفیک همراه و بدون نارسائی احتقانی قلب

سید فرزین سیدنژاد[1]، داریوش شیرانی[1*]، سعید بکائی[2]، سید مهدی نصیری[1]

[1]گروه بیماری‌های داخلی، دانشکده دامپزشکی دانشگاه تهران، تهران، ایران

[2]گروه اپیدمیولوژی و بیماری‌های مشترک گروه بهداشت و کنترل مواد غذایی، دانشکده دامپزشکی دانشگاه تهران، تهران، ایران

چکیده

زمینه مطالعه: آهن در عمل برای حیات تمامی ارگانیسم‌ها و بدون شک برای عملکردهای مختلف متابولیک ضروری است و کمبود آن می‌تواند بر روی کیفیت زندگی بیماران تأثیر بگذارد.

هدف: این مطالعه با هدف بررسی و مقایسه سطح آهن گربه‌های مبتلا به کاردیومیوپاتی هایپرتروفیک همراه و بدون نارسائی احتقانی قلب و تفاوت آن باگروه سالم انجام پذیرفته است.

روش کار: 45 گربه، براساس یافته‌های آزمایشگاهی، رادیولوژی و اکوکاردیوگرافی به سه گروه سالم، مبتلا به کاردیومیوپاتی هایپرتروفیک بدون نارسایی احتقانی قلب و مبتلا به کاردیومیوپاتی هایپرتروفیک همراه با نارسایی احتقانی قلب تقسیم شدند. در این گربه‌ها چهار فاکتور آهن، فریتین، مجموع ظرفیت اتصال به آهن و درصد اشباع ترنسفرین سرم اندازه‌گیری و با یکدیگر مقایسه شد.

* نویسنده مسئول: تلفن: 982161111749 نمابر: 982166933222 Email:dshirani@ut.ac.ir

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نتایج: در گربه‌های مبتلا به کاردیومیوپاتی هایپرتروفیک همراه با نارسایی احتقانی قلب، بیشترین غلظت آهن، کمترین میزان فریتین و درصد اشباع ترنسفرین را در بین سه گروه دارا بودند. همچنین مجموع ظرفیت اتصال به آهن در گروه بیماران بدون نارسائی احتقانی قلب کمترین بود.

نتیجه‌گیری نهایی: نتیجه این مطالعه پژوهشی حاکی از آن است که با وجود اختلاف معناداری که در مطالعات انسانی و سگ بین پانل آهن وجود دارد، در بین این سه گروه مورد مطالعه اختلاف معناداری مشاهده نگردید.

کلمات کلیدی: کاردیومیوپاتی هایپرتروفیک، فقر آهن، سطح آهن، گربه‌سانان

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Index	Control Medium; (min-max)	(A)* Medium; (min-max)	(B)* Medium; (min-max)

Table 1. Information on chest and abdomen imaging in control group, HCM group with and without congestive heart failure.

Interval wall thickness (mm)	4.25; (2.9-5.7)	7.3; (6.4-9.5)	6.5; (6.1-7.8)
Left ventricular wall thickness (mm)	4.3; (1.3-5.5)	7.4; (6.6-10.1)	6.7; (6.3-8.3)
The ratio of the left atrium to the aorta	1.2; (0.9-1.45)	1.81; (1.5-2.3)	1.45; (1.1-1.63)
Largest left atrium diameter in right longitudinal view (mm)	11.8; (9.5-12.6)	17.8; (15-23.2)	12.6; (11.1-14.5)
The presence of Spontaneous Echo Contrast	0	3.15	1.15

(A)*. HCM group with congestive heart failure.

(B)*. HCM group without congestive heart failure.

Index	Control Medium; (min-max)	(A)* Medium; (min-max)	(B)* Medium; (min-max)

Table 2. Echocardiographic findings in control group, HCM group with and without congestive heart failure

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Interval wall thickness (mm)	4.25; (2.9-5.7)	7.3; (6.4-9.5)	6.5; (6.1-7.8)
Left ventricular wall thickness (mm)	4.3; (1.3-5.5)	7.4; (6.6-10.1)	6.7; (6.3-8.3)
The ratio of the left atrium to the aorta	1.2; (0.9-1.45)	1.81; (1.5-2.3)	1.45; (1.1-1.63)
Largest left atrium diameter in right longitudinal view (mm)	11.8; (9.5-12.6)	17.8; (15-23.2)	12.6; (11.1-14.5)
The presence of Spontaneous Echo Contrast	0	3.15	1.15

(A)*. HCM group with congestive heart failure.

Uncorrected Proof

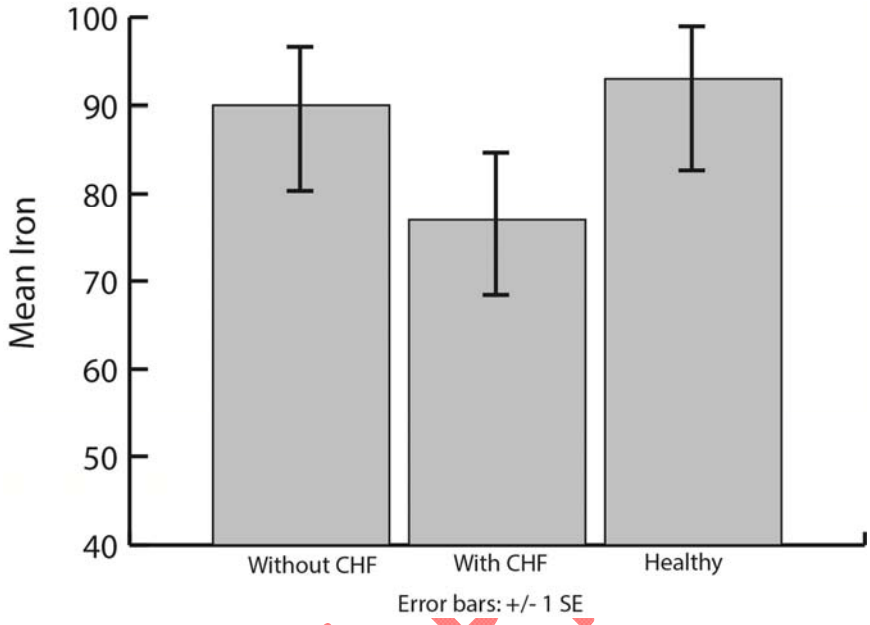

Figure 1. Mean Iron concentration

360

Uncorrected P

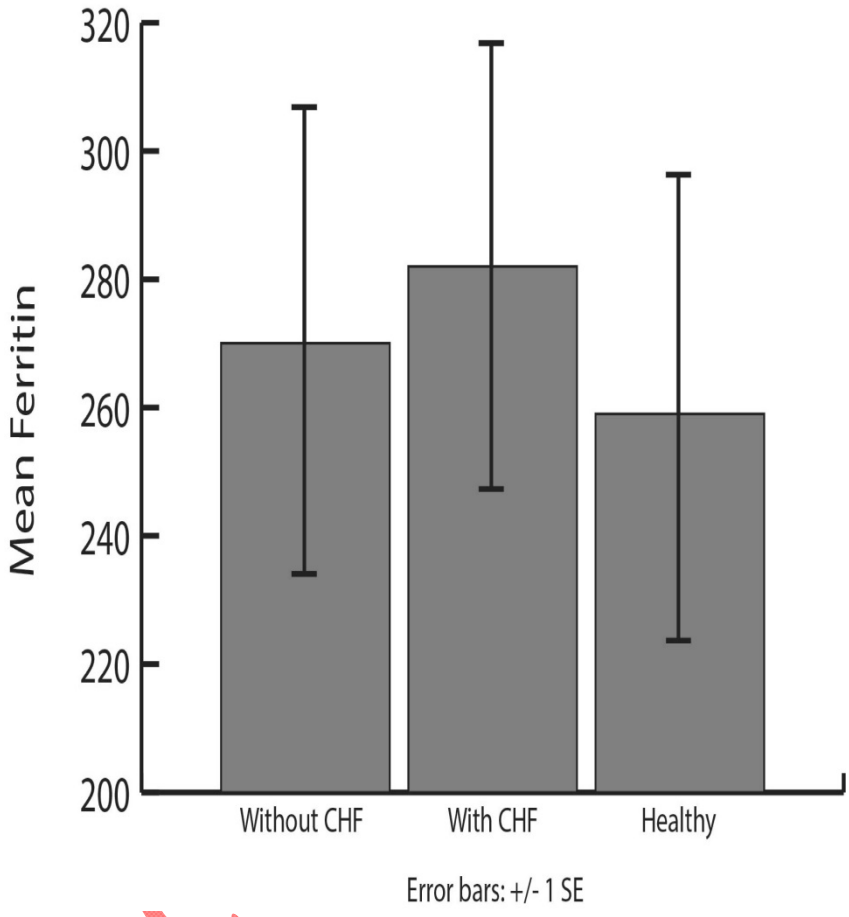

Figure 2. Mean Ferritin concentration

Uncorrec

365

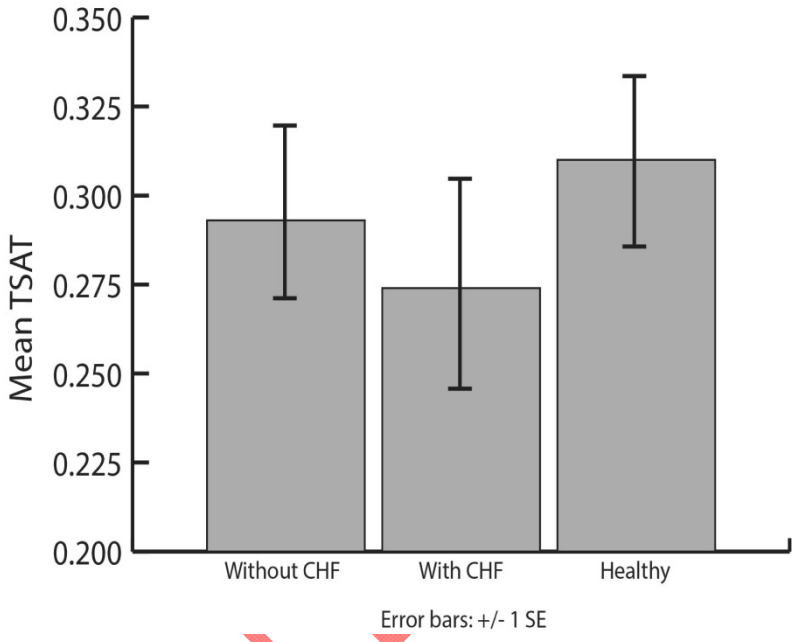

Figure 3. Mean TIBC

370

Uncorrected

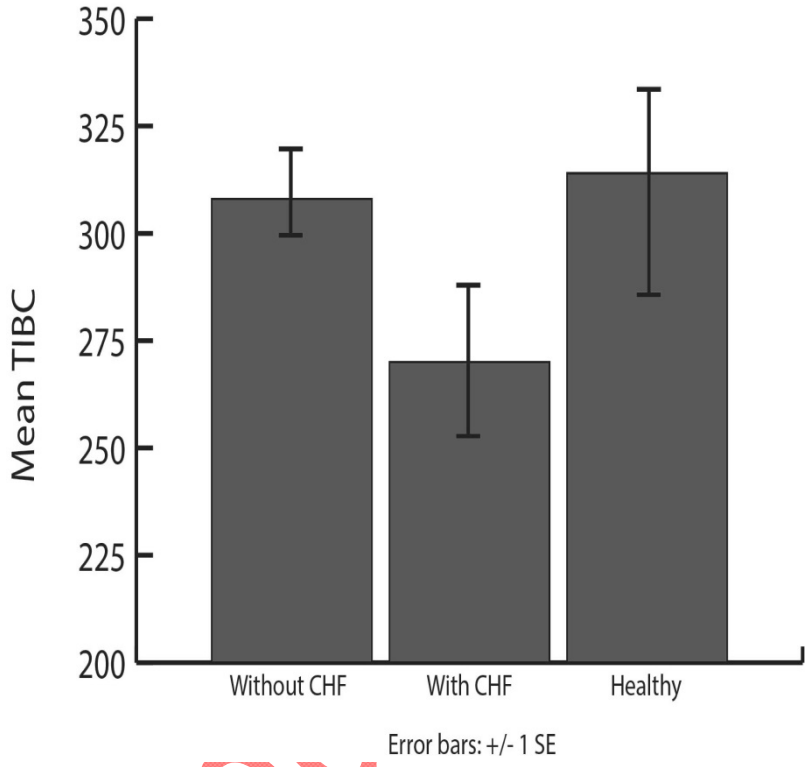

Figure 4. Mean TSAT

375

Uncorrected