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Correlation between Iron Status and Heart Failure Severity in Patients with Chronic Heart Failure

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Abstract

Background: Iron deficiency anemia is widely present in patients with heart failure (HF) with an estimated prevalence of over 50% in ambulatory patients. It is an independent predictor of worse functional capacity and survival.

Aim of Study: To measure serum hepicidin in patients with chronic systolic HF, compare this with healthy subjects and correlate iron status (serum ferritin and transferrin saturation) and serum hepicidin and the stages of HF severity.

Patients and Methods: Sixty patients with systolic HF diagnosed at the outpatient clinic or Cardiology Department at the Mansoura Specialized Internal Medicine Hospital, Mansoura University, Mansoura, Egypt, were included in the study.

Results: The mean age of the patients was 62.33 ±7.48 years, and in the control group was 61.13 ±7.50 years. In the cases group, there was 83.3% males and 16.7% females versus 63.3% males and 36.7% females in the control group. Forty five cases (75%) presented with class II NYHA classification HF and 15 cases (25%) with class III NYHA classification heart failure. The median levels of hemoglobin concentration, mean corpuscular volume, serum iron, serum ferritin and hepcidin level were higher in NYHA class II cases as compared to class III. The best cutoff point of hepcidin to identify HF cases from the control was below 9.15ng/ml with 63.3% sensitivity and 89.3% specificity. The AUC was 0.772 with high statistically difference (p<0.001).

Conclusion: Heart failure is associated with anemia, especially iron deficiency anemia, Serum hepcidin could be utilized as a marker for heart failure as it is increased with the disease severity.

Key Words: *Chronic heart failure – Iron deficiency anemia – Serum hepicidin.*

Introduction

HEART failure (HF) is a common condition and one that is projected to increase significantly in the coming decades due to the ageing population and increased survival of patients with complications of coronary artery disease [1]. It is estimated that the number of patients living with HF in the USA will increase by ~50% by 2030 [2]. Heart failure is a major cause of death with current 1 year mortality rates of 25-35% after an initial hospitalization with HF [3].

Iron deficiency anemia is widely present in patients with HF with an estimated prevalence of over 50% in ambulatory patients. It is an independent predictor of worse functional capacity and survival [3].

Definition of iron deficiency in HF differs from other conditions of chronic inflammation and is defined as: Ferritin <100 µg/L or ferritin of 100-299 µg/L with a transferrin saturation (TS) <20% [4]. At present, intravenous iron is the preferred route for treatment in heart failure patients [5].

Hepcidin is the iron regulatory hormone produced in the liver in response to both the fluctuating level of iron in the hepatocyte, and elevated cytokine (IL6) levels induced by inflammation or infection [6]. The postulated role of hepcidin in inflammation and infection is to deprive pathogens of essential iron, forming part of our innate immunity [7].

Inappropriate elevation of hepcidin is an important mechanism of anaemia of chronic disease [8]. In inflammatory conditions, higher levels of hepcidin coincide with elevated inflammatory markers (IL6) and more severe disease [9].

In HF, the opposite relationship has been demonstrated, with hepcidin levels found to be inversely related to the severity of disease [10]. In a case-control study of 321 patients with chronic HF, high hepcidin levels were found in patients with mild symptoms of HF (NYHA I/II) [11]. So, the aim of this study was tomeasure serum hepicidin in patients with chronic systolic HF, compare this with

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healthy subjects and correlate iron status (serum ferritin and TS) and serum hepicidin and stage of HF severity.

Patients and Methods

This study included 60 patients with systolic HF who were diagnosed at the outpatient clinic or Cardiology Department at the Mansoura Specialized Internal Medicine Hospital, Mansoura University, Mansoura, Egypt for a duration of one year between January 2019 and December 2019.

Ethical consideration:

Written informed consents were obtained from all participants before inclusion in the study. The whole study design was approved by the Institutional Review Board (IRB), Faculty of Medicine, Mansoura University, confidentiality, and personal privacy will be respected in all levels of the study, patients feel free to withdraw from the study at any time without any consequences and collected data will not be used for any other purpose.

Inclusion criteria:

Both sexes, age ≥18 years, a documented history of HF of ≥6 months, symptoms and signs of left side HF and left ventricular ejection fraction (LVEF) ≤45% as assessed by echocardiography (performed at the time of the study) [1] were the inclusion criteria.

Exclusion criteria:

- Acute coronary syndrome and/or coronary revascularization within the 3 months preceding the study.
- Unplanned hospitalization due to HF deterioration or any other cardiovascular reason within 1 month preceding the study.
- Any acute or chronic illness that might influence iron metabolism (including malignancy, infection, severe renal disease requiring dialysis, and hematological diseases).

Full detailed history:

As age, sex, special habits, associated chronic diseases (diabetes mellitus, hypertension, liver diseases, endocrinal diseases), state of current disease (causes, onset and duration) and risk factors for heart diseases, history of palpitation or easy fatigability, history of cardiac, hepatic or renal diseases, Family history and drug history.

Full clinical examination:

Focusing on general examination: Vital signs (blood pressure, pulse,respiratory rate, temperature) and Cardiac examination.

Investigation:

Hemoglobin levels, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC), serum creatinine, serum level of high-sensitivity C-reactive protein (hs-C reactive protein, mg/L) (CRP) was assessed using kinetic nephelometry), assessment of iron status, assessment of serum hepcidin [6], 12-lead electrocardiography (ECG) (rate, rhythm, ischemic changes, pattern of p waves, T waves & PR segments) and Echocardiography (ejection fraction, left ventricular internal dimensions and severity of mitral regurge).

Statistical analysis:

The collected data were coded, processed and analyzed using the SPSS (Statistical Package for Social Sciences) version 22 for Windows® (IBM SPSS Inc, Chicago, IL, USA).Data were tested for normal distribution using the Shapiro Walk test. Qualitative data represented as frequencies and relative percentages. Chi square test (χ^2) and Fisher exact used to calculate difference between qualitative variables as indicated. Quantitative data were expressed as mean ± SD (Standard deviation) or median (range). Independent samples *t*-test used to compare between two independent groups of normally distributed variables (parametric data) while Mann Whitney U test used for non-normally distributed Data (non-parametric data). One way analysis of the variance (one way ANOVA) used to compare between more than two independent groups of normally distributed variables (parametric data) while Kruskal Wallis test was used for non-normally distributed Data (non-parametric data). Spearman's correlation was used to test the correlation between two variables with non-parametric quantitative data and receiver operating characteristic (ROC) analysis. For all the above-mentioned tests, the level of significance was tested, expressed as the probability of (*p*-value). Significant if the *p*-value is ≤0.05.

Results

A CONSORT flowchart of the study population is shown in Fig. (1), where 60 of 75 patients with systolic HF were included in this study. They attended at Mansoura Specialized Hospital in Egypt, for a duration of one year between January 2019 and December 2019, and were willing to participate, and gave their consent. Of the fifteen patients who were excluded from the study, eight did not meet the inclusion criteria and seven declined. As a result, 60 patients were examined and divided into three groups: 42 patients with ischemic HF (group I) and 10 patients with valvular HF

(group II) and 8 patients with idiopathic HF (group III).

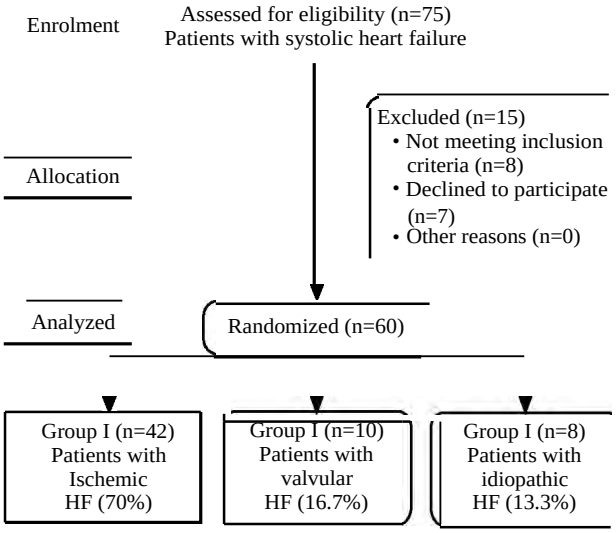

Fig. (1): Flowchart of the studied patients.

In the current study, the mean age in the cases group was 62.33±7.48 years and in the control group was 61.13±7.50 years, with no significant difference between the two groups. There was 83.3% males and 16.7% females, while in the control group there was 63.3% males and 36.7% females, with statistically significant difference between the two groups (p=0.034). There was no statistically significant difference between the two groups in the mean BMI. There were 40% of the cases group with DM, while only 20% of the control group had DM with statistically significant difference between the two groups. The CRP levels were significantly higher in the heart failure group as compared with control group (2.6mg/l vs 0.9mg/l respectively) (Table 1).

Table (1): Demographic data between the two study groups.

	Groups		Test of	
	Cases group (N=60)	Control group (N=30)	Signif-icance	*p*-value
Age (years)	62.33±7.48	61.13±7.50	t= 0.717	0.475
Sex:				
Males	50 (83.3%)	19 (63.3%)	χ^2=4.427	0.034*
Females	10 (16.7%)	11 (36.7%)		
BMI (kg/m^2)	27.32±1.92	26.07±2.50	t=1.626	0.273
DM	24 (40%)	6 (20%)	χ^2=3.601	0.038*
Serum creatinine	0.99±0.23	0.89±0.21	t=1.875	0.066
CRP (mg/L)	2.6 (1.3-6.8)	0.9 (0.5-1.3)	z=–3.485	0.011*

p : Probability.
Continuous data expressed as mean ± SD.
Qualitative data are expressed as number (percentage).
t_2 : Independent samples *t*-test.
χ^2 : Chi-square test.
* : Statistically significant.

Regarding the cause of HF in the cases group, the most common cause was the ischemic heart failure in 70% of the cases followed by 16.7% of the cases had valvular heart disease and idiopathic HF (non-ischemic and non-valvular) was detected in 13.3% of the cases (Fig. 2).

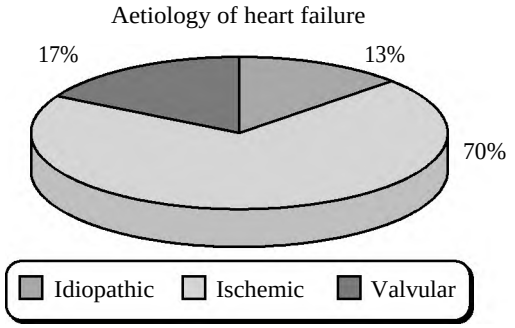

Fig. (2): Aetiology of heart failure in the cases group.

There was mild mitral regurgitation (MR) in 26 cases (43.3%), moderate MR in 21 cases (35%) and severe MR in 13 cases (21.7%). The percentage of cases with atrial fibrillation (AF) in the cases group was 23.3% while only 1 case in the cases group had implantable cardiovascular defibrillator (ICD) (Table 2).

Table (2): Analysis of degree of MR, AF, and ICD in the cases group.

Cases group (N=60)	Number (%)
Degree of MR:	
Mild	26 (43.3%)
Moderate	21 (35%)
Severe	13 (21.7%)
Atrial fibrillation (AF)	14 (23.3%)
Implantable cardiovascular defibrillator (ICD)	1 (1.7%)

Qualitative data are expressed as number (%).
MR : Mitral Regurgitation.
AF : Atrial Fibrillation.
ICD: Implantable Cardioverter Defibrillator.

There were 45 cases (75%) with class II NYHA classification HF and 15 cases (25%) with class III NYHA classification heart failure (Fig. 3).

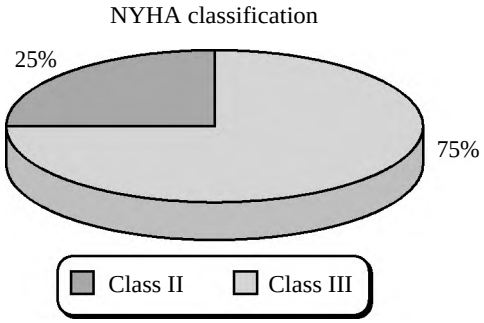

Fig. (3): Analysis of NYHA classification of heart failure in the cases group.

Additionally, the EF in the cases group was 40.70±2.80% which was statistically significant lower as compared with the control group (67 ± 4.74%) (p<0.001). The mean hemoglobin concentration, mean MCV and mean MCH were statistically significantly lower in the cases group as compared with the control group. The mean MCHC was lower in the cases group, but it didn't reach a statistically significant value and the percentage of cases with anemia in the cases group was 45% vs 6.7% in the control group with high statistically significant difference between the two groups (p<0.001), (Table 3).

Table (3): Analysis of EF and the laboratory parameters in the two study groups.

	Groups		Test of	
	Cases group (N=60)	Control group (N=30)	t	p-value
EF	40.70±2.80	67±4.74	33.094	<0.001 *
HGB (mg/dl)	12.03±1.79	13.60±1.24	4.289	<0.001 *
MCV	80.77±5.54	85.40±4.19	4.036	<0.001 *
MCH	27.68±3.32	29±2.10	1.994	0.049*
MCHC	31.53±2.29	32.13±1.89	1.240	0.218
Presence of anemia	27 (45%)	2 (6.7%)	χ^2=14.421	<0.001 *

p : Probability. t : Independent samples t-test.
Continuous data expressed as mean ± SD. χ^2 : Chi-square test.
Qualitative data are expressed as number (percentage). * : Statistically significant (p<0.05).

Moreover, there was high statistically significant difference between the control group, cases with class II NYHA classification and cases with class III NYHA classification as regards hemoglobin (HGB) concentration, mean corpuscular volume (MCV), serum iron, serum ferritin, total iron binding capacity (TIBC) and hepcidin level. The median levels of HGB concentration, MCV, serum iron, serum ferritin and hepcidin level were higher in NYHA class II cases as compared to class III. The mean value of TIBC was statistically significantly lower in class II NYHA class HF cases as compared with cases in class III. Table (4).

Table (4): Analysis of HGB, MCV and iron profile in the study groups.

	Groups			Test of	
	Control group (N=30)	NYHA II (N=45)	NYHA III (N=15)	Significance	p-value
HGB (mg/dl)	13.60±1.24 **A**	12.15±1.69 **B**	11.69± 2.10 **B**	F=9.652	<0.001 *
MCV	85.40±4.19 **A**	80.80±5.90 **B**	80.67±4.45 **B**	F=8.055	<0.001 *
Serum iron (mcg/dl)	96 (69.75-114.75) **A**	62 (44-70.75) **B**	59 (38-79) v	KW=30.390	<0.001 *
Serum ferritin (ng/ml)	96 (57-147) **A**	33 (27.5-62.5) v	28 (24-80) **B**	KW=25.447	<0.001 *
TIBC	279.40±44.92 **A**	300.42±47.94 **A**	322.06±22.5 v	F=4.236	0.018*
Hepcidin (ng/ml)	10.25 (7.27-12.75) **A**	7.4 (5.2-8.6) **B**	5.1 (3.1-7.5) **B**	KW=20.195	<0.001 *

p: Probability. t: independent samples t-test.
Continuous data expressed as mean ± SD or median (IQR). χ^2 : Chi-square test.
Qualitative data are expressed as number (percentage). z : Mann-Whitney U-test.
A,A: Similar letters indicates no statistically significant difference between the two adjacent subgroups A.
B : Different letters indicates a statistically significant difference between the two adjacent subgroups.

Furthermore, there was statistically significant moderate positive correlation between serum hepcidin levels with EF, HGB, MCV, MCH, MCHC, serum iron and serum ferritin levels. Other parameters didn't show statistically significant correlation with serum hepcidin level. Table (5).

The best cutoff point of hepcidin to identify HF cases from the control is below 9.15ng/ml with 63.3% sensitivity and 89.3% specificity. The AUC is 0.772 with high statistically difference (<0.001). (Table 6, Fig. 4).

Table (5): Correlation between hepcidin and other parameters in the study.

Variables	Hepcidin	
	r	*p*-value
Age	–0.016	0.882
BMI	–0.101	0.349
EF	0.452	<0.001*
HGB	0.517	<0.001*
MCV	0.391	<0.001*
MCH	0.292	0.005*
MCHC	0.244	0.020*
Creatinine	–0.160	0.133
Iron	0.460	<0.001*
Ferritin	0.397	<0.001*
TIBC	–0.191	0.072

r: Spearman's correlation.
p: Probability.
*: Statistically significant ($p<0.05$).

Table (6): Analysis of the diagnostic ability of hepcidin to differentiate CHF cases from the controls.

Diagnostic parameter	Hepcidin levels
AUC	0.772
Cut off point	>9.15 (for control)
Sensitivity	63.3%
Specificity	89.3%
PPV	68.4%
NPV	90.6%
Accuracy	82.8%
p-value	<0.001*

AUC: Area under the curve. PPV : Positive predictive value.
p: Probability. NPV: Negative predictive value.
*: Significant *p*-value (<0.05).

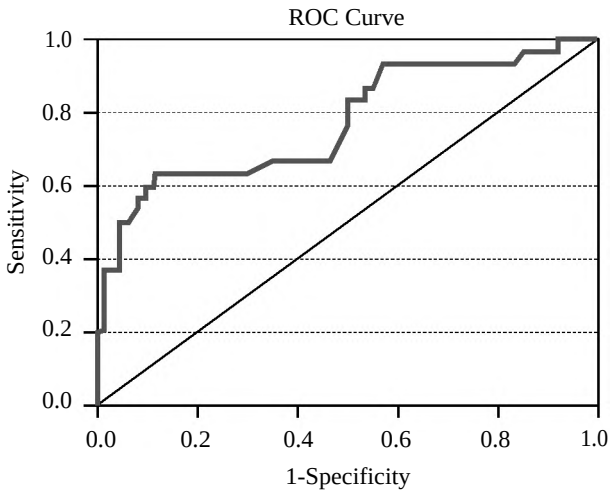

Fig. (4): Roc curve for hepcidin level to differentiate between CHF cases and control.

Discussion

Heart failure (HF) is a chronic disabling syndrome with a prevalence of 1% to 2% in the global population and ≥10% in those age ≥65 years, and is being increasingly recognized in younger patients **[12]**. Acute on top of chronic HF is the leading cause of recurrent hospitalizations and early readmissions, which accounts for its notably high morbidity and costs. Patients have a lower quality of life, and their survival is gravely compromised **[13,14]**.

Iron deficiency (ID) is frequent among patients with stable systolic heart failure (HF) and has serious unfavorable clinical and prognostic consequences **[15]**. Iron deficiency (ID) is overwhelmingly recognized in over 30% to 50% of patients with stable chronic HF **[16]**. Intravenous iron therapy administered in patients with HF and ID lessens the symptoms and improves exercise capacity and quality of life. However, the sequence of changes in iron status occurring during the natural history of HF and the pathomechanisms triggering ID in these patients remain not fully understood **[17]**.

Hepcidin controls the activity of ferroportin, a transmembrane iron exporter out of different cell types, that is, at the site of iron absorption (gut mucosa cells in the duodenum), as well as at the site of iron storage (hepatocytes, macrophages). Once ferroportin is bound by hepcidin, it is destroyed in the lysosome, leading to reduced iron release **[18]** as hepcidin is commonly and predominantly acknowledged as the key regulator of iron metabolism, an analysis of the pattern of its changes may allow one to understand the pathomechanisms leading to iron deficiency, which characterizes the HF syndrome. So this study was conducted to determine the changes in iron status in parallel with disease progression. The study included 60 patients diagnosed as systolic HF and 30 healthy control subjects.

In our study, the mean age in the cases group was 62.33±7.48 years and in the control group was 61.13±7.50 years with no significant difference between the two groups. This came opposite to Yildirim et al., **[19]** who reported that in the cases with HF, the mean age of the patients was 62.5 ± 10.5 years and the mean age in the control group was 51.7±9.2 years. The age of the patients in the case group was significantly higher than the control group ($p<0.05$) **[19]**. In general, the prevalence of HF is around 2% in the 40-59 age group, while about 5% in the 60-69 age group is approximately 10% in the age group of 70 years **[20]**.

In the current study, in the cases group, there was 83.3% males and 16.7% females while in the control group there was 63.3% males and 36.7% females with statistically significant difference between the two groups ($p=0.034$). This came in accordance with Yildirim et al., **[19]** who showed

that thirty-three (66%) of the cases with HF were male and 17 (34%) were female while six of the control group (30%) were male and 14 (70%) were female. The rate of male patients in the case group was significantly higher than that in the control group (p<0.05).

According to our study, the serum creatinine level was higher in the HF cases group as compared with the healthy controls, however it didn't reach a statistically significant value. In the study conducted by Yildirim et al., [19] urea and creatinine concentrations were found to be significantly higher in patients with heart failure than normal controls [19]. The most important reason for this is cardiorenal syndrome. Ronco, et al., in their study revealed that there was a communication between the heart and kidney as two aspects and classified the cardiorenal syndrome [21].

In the current study, the mean hemoglobin concentration, mean MCV and mean MCH were statistically significant lower in the cases group as compared with the control group. The mean MCHC was lower in the cases group, but it didn't reach a statistically significant value. This came in accordance with Divakaran et al., [22] who studied 36 patients with heart failure and anemia, 61 patients with heart failure and no anemia, and 38 control subjects. Patients in the anemic group had a lower hemoglobin (11.64±0.19g/dL) compared to those in the nonanemic group (14.25 ± 0.15g/dL) or control subjects (14.14±0.27g/dL).

Our study showed the percentage of cases with anemia in the cases group was 45% vs 6.7% in the control group with high statistically significant difference between the two groups (p<0.001). Adlbrecht, et al., [23] described the cause of anemia in HF as a relative increase in plasma volume rather than a massive reduction in red blood cell [23]. The CRP levels were significantly higher in the heart failure group as compared with control group (2.6mg/l vs 0.9mg/l respectively) (p=0.011). Jankowska et al., [11] showed that proinflammatory activation was associated with HF severity, as evidenced by increased serum levels of hsC-reactive protein and IL-6 in patients with systolic HF in subsequent NYHA classes (both p<0.00 1).

In the present study, the median serum iron in the cases group is 62mcg/dl which was statistically significantly lower as compared with the control group (96mcg/dl). The median serum ferritin level in the cases group was 32.5 which was statistically significantly lower as compared with the control group (96). The mean TIBC in the cases group was 305.83±49.46 which was higher as compared with the control group (279.40±44.92) with significant difference between the two groups (p=0.016). This agreed with Matsumoto, et al. (2010), found that serum iron and transferrin saturation in HF patients with anemia were found to be within the normal range but significantly lower than control group [24].

Also, the current results were comparable to Yildirim et al., [19] who showed that iron concentrations of patients with HF were significantly lower and iron binding capacity was significantly higher than control group. In accordance with our results, Cabrera et al., showed that serum iron and ferritin levels were statistically significant lower in the control group as compared with heart failure patients with preserved and reduced ejection fraction [25].

The present study reported that, the median levels of serum iron and serum ferritin and serum hepcidin levels were higher in NYHA class II and TIBC was lower, however, it didn't achieve a statistically significant difference. This came in agreement with Jankowska et al., [11] who showed that with increasing HF severity, assessed by NYHA class, patients developed iron deficiency (evidenced by reduced ferritin and low iron levels).

The current study found that, the median serum hepcidin concentration in the cases group was 7.15ng/ml which was statistically significantly lower as compared with the control group (10.25). Also, the median levels of serum hepcidin levels were higher in NYHA class II cases as compared to class III, but it didn't reach a statistically significant value (p=0.061). This came in agreement with Yildirim et al., [19] who showed that the mean serum hepcidin in the HF cases was 21.5±17.4 ng/ml which was higher than the level in the control group (16.3±8.5), but it didn't reach a statistically significant value Matsumoto, et al., [24] examined serum hepcidin concentrations in patients with heart failure. 36 heart failure patients with anemia, 16 patients without heart failure and anemia were compared. They found that serum hepcidin concentrations in heart failure patients with anemia were lower than those in other groups and that the anemia of inflammation was a minor criteria in the development of anemia in heart failure patients.

Our current results came opposite to the results of Jankowska et al., [11] who showed that as compared with healthy controls, patients with systolic HF in NYHA class I had increased serum levels of hepcidin (p<0.001). Only the cases with NYHA

class IV had serum levels of hepcidin lower than the controls. However, the authors agreed with us as they reported with increasing HF severity, assessed by NYHA class, patients developed a marked decrease in circulating hepcidin (p<0.001 for differences across NYHA classes). The explanation of elevated serum hepcidin during early stage of HF is owed to that the liver is established as the major source of circulating hepcidin **[26,27]**. There is an alternative (so far hypothetical) theory about an extra-hepatic origin of circulating hepcidin in HF (e.g., from ischaemic and/or hypoperfused myocardium) **[28,29]**. Recent research conducted by Lakhal-Littleton et al., in mouse models with specific cardiac hepcidin knockout showed that loss of hepcidin was associated with fatal cardiac dysfunction. Loss of cardiac hepcidin resulted in very significant increase in left ventricle mass and apoptosis. This dysfunction caused a marked reduction in ejection fraction of rat hearts **[30]**.

According to our study, there was statistically significant moderate positive correlation between serum hepcidin levels and EF, HGB, MCV, MCH, MCHC, serum iron and serum ferritin levels. This came in accordance with Yildirim et al., **[19]** who reported that there was a positive correlation between hepcidin concentration and urea, ferritin, hemoglobin, hematocrite, C-reactive protein (p<0.05). In the same study, there was no correlation between hepcidin concentration and age, weight, creatinine, iron, vitamin B 12, folate, white blood cell (WBC), platelet, MCV, sedimentation rate, ejection fraction (EF) (p>0.05). In the study conducted by Jankwoska et al., **[11]** low hepcidin level in patients with systolic HF: Older age (r= –0.15, p<0.01), more prevalent ischaemic HF aetiology (t=2.05, p<0.05), high plasma NTproBNP (r=–0.12, p<0.05), high serum IL-6 (r=–0.33, p<0.001), low MCV (r=0.27, p<0.001), high RDW (r=–0.46, p<0.001), low serum ferritin (r=0.57, p<0.001), low Tsat (r=0.14, p<0.05), increased TIBC (r=–0.16, p<0.01), and high sTfR (r=–0.22, p<0.001).

In our study, the best cutoff point of hepcidin to identify HF cases from the control was below 9.15ng/ml, with 63.3% sensitivity and 89.3%. The AUC was 0.772 with high statistically significant difference (<0.001). This is one of the strength points of this study as no previous studies have reported the cutoff point of serum hepcidin to differentiate between HF and controls.

Conclusion:

Heart failure is associated with anemia and especially iron deficiency anemia. Anemia is present in increased prevalence with increasing the severity of heart failure. Serum hepcidin could be utilized as a marker for heart failure as it is increased with the disease severity except in NYHA III HF. Iron deficiency anemia is an extremely common comorbidity in patients with heart failure patients.

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المصابين بفشل مزمن فى عضلة القلب

فشل عضلة القلب هو متلازمة تتميز بالأعراض التالية ضيق فى التنفس وتورم فى الكاحل واجهاد التى تكون مصاحبة بارتفاع الضغط الوريدى الوداجى والخشخاش الرئوى والوذمة الطرفية بسبب خلل بنيوى و/أو وظيفى فى وظائف القلب، مما يؤدى إلى إنخفاض فى النتاج القلبى و/أو ارتفاع الضغوط داخل القلب أثناء الراحة أو أثناء الاجهاد. يقيد التعريف الحالى لفشل عضلة القلب إلى المراحل التى تظهر فيها الاعراض السريرية ويعتبر التعرف على السلائف مهماً لأنها ترتبط بالنتائج السيئة، وقد يؤدى بدء العالج فى مرحلة السلائف إلى خفض معدل الوفيات لدى المرضى الذين يعانون من خلل وظيفى فى ضغط الدم الانقباضى بدون أعراض.

الهدف من هذه الدراسة هو تحديد العلا قة بين الأنيميا فى مرضى فشل عضلة القلب المزمن والتى تقاس بنسبة الحديد فى الدم ومادة الهيبثدين المسئولة عن امتصاص الحديد والتى يزداد افرازها فى مرضى فشل عضلة القلب المزمن.

اشتمل هذا البحث على متابعة ٦٠ مريض يعانون من ضعف مزمن بعضلة القلب و ٣٠ من الأصحاء اعتماداً على أعراض ومضاعفات فشل عضلة القلب كل المرضى فى هذه الدراسة تم سحب عينات من الدم لمعرفة مستويات الحديد والهيبثدين فى الدم وتم مقارنتها بمدى شدة أعراض الفشل القلبى المزمن على مقياس كوهرت.

كانت المستويات المتوسطة للحديد والفيريتين وهيبسيدين المصل أقل بكثير فى مجموعة فشل عضلة القلب مقارنة مع المجموعة الضابطة وأظهرت انخفاضاً ملحوظاً مع زيادة شدة المرض. أفضل نقطة فاصلة للهيبسيدين لتحديد حالات فشل عضلة القلب هى أقل من ١٩.٥ نانو غرام/مل مع حساسية ٦٣.٣٪ وخصوصية.