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The Relationship of Iron Stores Biomarkers with Chronic Heart Failure in Nondialysis Chronic Kidney Disease Patients

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ABSTRACT

Background: Patients with chronic heart failure (CHF) benefit from treatment for iron deficiency by having better cardiac and renal function.

Objective: To evaluate the relationship of serum biomarkers of iron stores with CHF in nondialysis chronic kidney disease (CKD) patients, with or without anemia and to evaluate the therapeutic effect of iron sucrose administration for HF patients with iron deficiency.

Patients and Methods: This prospective study was conducted on 143 patients with CHF with eGFR < 60 ml/min/1.73 m^2 for evaluation the association of TSAT and serum ferretin level categories with all clinical, biochemical characteristics, NYHA functional class and echocardiographic findings. Only 100 patients were selected and randomized into two groups (control group and iron sucrose group) and followed up by echocardiography for 3 months.

Results: There were statistically significant differences in clinical, biochemical and echocardiographic parameters between iron stores level categories with better outcomes in higher TSAT and s. ferretin level categories. Intravenous iron was associated with improved renal function (both $p<0.01$ versus control). left ventricular systolic and diastolic diameters were decreased ($p<0.01$), global longitudinal systolic strain was significantly decreased ($p<0.01$) indicating improved left ventricular function.

Conclusion: Higher s. ferretin and TSAT level categories was associated with better outcomes involving clinical, biochemical and echocardiographic findings in comparison with lower s. ferretin and TSAT level categories in patients with heart failure and non-dialysis CKD. In patients with HF, intravenous iron therapy was linked to better myocardial functional measures and cardiac dimensions.

Keywords: Iron deficiency, HF, CKD, Ferritin, TSAT, Iron sucrose.

INTRODUCTION

Heart failure (HF), iron deficiency, and renal failure all have a strong link, and each of these concomitant conditions lowers these patients' chances of surviving. Up to 50% of people with HF have an iron deficit [(1)]. HF patients with iron deficiency also have greater rates of hospitalisation and death, in addition to having a lower quality of life and functional ability. Patients with and without anemia continue to experience the same results [(2,3)].

Several trials conducted in HF patients with lower ejection fraction and iron deficiency showed that IV iron supplementation, but not oral iron, improved outcomes, HF symptoms, functional class, and quality of life [(4-5)]. These advantages of IV iron treatment were found to exist regardless of the existence of anemia, according to a study [(5)].

More recently, research among patients with acute HF and iron deficiency showed a decrease in HF hospitalisations [(5)].

Given that iron plays a significant role in the cellular immune response and oxygen metabolism, particularly in cardiac myocytes, intravenous iron supplementation has emerged as a promising therapeutic target in patients with HF and may complement beta blockade and the inhibition of the renin-angiotensin-aldosterone system. For the use of intravenous iron supplementation in these patients, current recommendations place a Class IIA recommendation [(6)].

Serum ferritin levels of less than 100 g/L or between 100 and 299 g/L combined with a TSAT level of less than 20% are now recognised and advised criterion for identifying ID in HF patients [(7,8)].

There is no need to evaluate iron status after a few weeks if ID is diagnosed and intravenous iron supplementation is given. Serum ferritin and TSAT are often artificially elevated after intravenous iron supplementation for 2-3 weeks (sometimes for a little longer), and the interpretation of these findings is unclear. Following intravenous iron delivery, it is advised to reevaluate the patient's iron status using serum ferritin and TSAT at 3 and 6 months. A second estimated dosage of intravenous iron is advised if ID continues. It is advised to regularly assess your iron status annually if your iron reserves are full (based on normal blood ferritin and TSAT levels) [(9)].

This study aimed to evaluate the relationship of serum biomarkers of iron stores with CHF in nondialysis chronic kidney disease (CKD) patients, with or without anemia and to evaluate the therapeutic effect of iron sucrose administration for HF patients with iron deficiency.

PATIENTS AND METHODS

This was a prospective study of 3 months duration. 143 patients were consecutively recruited from the general population attended to the outpatient clinic (Internal Medicine and Cardiology section) at Benha University Hospital, Benha, Egypt.

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Patients with CHF who also have non-dialysis CKD (eGFR < 60 ml/min/1.73 m^2) with anemia (Hb concentration <12.5 g/dL in men and <11.5 g/dL in women) or without anemia were enrolled in our first part of study for evaluation and studying the association of iron stores biomarkers (s. ferretin and transferrin saturation) with CHF.

Also, for the analysis of TSAT and s. ferretin level categories with all clinical, biochemical characteristics, NYHA functional class and echocardiographic findings. Then only 100 of our studied patients, who were iron deficient (serum ferritin <100 μg/L, or 100–299 μg/L with transferrin saturation <20%) with or without anemia, were selected to continue in our interventional part of study. Patients who satisfied the criteria for inclusion and gave written informed consent at their initial medical review were randomly assigned to the intervention or control groups using a table of random numbers.

Inclusion criteria: Patients older than or equal to 18 years old, of both gender, CHF patients with nondialysis CKD (eGFR <60 ml/min per 1.73 m^2) with iron deficiency (serum ferritin <100 μg/L, or 100–299 μg/L with transferrin saturation <20%) with anemia (Hb concentration <12.5 g/dL in men and <11.5 g/dL in women) or without anemia.

Exclusion criteria: Patients with a prior kidney transplant or receiving maintenance dialysis, use of erythropoietin or iron (oral or IV), blood transfusion within the previous 30 days, history of hemochromatosis or acquired iron overload, prior hypersensitivity to parental iron preparations, or a history of allergic disorders, active infection, bleeding, malignancy, hemolytic anemia, musculoskeletal disease, unstable angina pectoris, obstructive cardiomyopathy, severe uncorrected valvular disease, or uncontrolled, immunosuppressive medications.

From each patient the following data were collected.

1. Complete full history taking, and according to the NYHA functional categorization[(10)], symptoms were evaluated.
2. Clinical examination involving SBP, DBP and BMI.
3. Laboratory investigations
4. Transthoracic Speckle Tracking Echocardiography: All echocardiographic tests were done and recorded for offline analysis using a Philips EPIQ 7C, Release 1.7 equipment with Q Lab 10.4, an S5-1 probe, and simultaneous electrocardiogram signal (Philips Healthcare, Andover, MA, USA).
5. Treatment: In our intervention part of study, according to current criteria, all 100 patients received the best possible therapy for CHF. In addition to conventional therapy, the control group (n=50) got a placebo, whereas the intervention group (n=50) received 200 mg/100 mL of intravenous iron sucrose every week for five weeks in addition to usual therapy. Patients were assessed at baseline then monthly for 3 months by means of physical examination, NYHA functional classification, echocardiography and laboratory determinations:
   - Hemoglobin (Hb) (g/dl), serum ferritin (ng/ml), transferrin saturation (%).
   - Complete blood count (CBC): Platelets (cell/L), white blood cell (WBCs) (cell/L), red blood cell (RBCs) (cell/ L).
   - Blood urea (mg/dl), serum creatinine (mg/dl), albumin /creatinine ratio (ACR) (mg/g).
   - Thyroid function: TSH (uIU /ml), fT3 (pg/ml), Ft4 (ng/dl).
   - Fasting blood glucose (FBG) (mg/dl), Glycosylated hemoglobin (HbA1c) (%).
   - Lipid profile: total cholesterol (mg/dl), LDL-cholesterol (mg/dl), HDL- cholesterol (mg/dl), s.triglycerides (mg/dl).
   - Serum albumin (g/dl).
   - Estimated glomerular filtiration rate (eGFR) by CKD - EPI Equation (ml/min per 1.73 m^2).

Ethical consent:

After receiving the approval from Benha University's Institutional Ethics Committee, the participants provided signed consent after being fully briefed. Each participant was given a secret code number and was given a description of the study's goals. The Helsinki Declaration was followed throughout the study's conduct.

Statistical analysis:

A report form was used to capture the clinical data. To generate descriptive data and analytical statistics, these data were tabulated and examined using the computer programme SPSS V. 26. For presenting quantitative data, the mean ± SD, and for qualitative data, the frequency and distribution were used. P value < 0.05 was regarded as significant.

RESULTS

There was a statistically significant direct relationship between serum ferritin levels and blood pressure. This means that as serum ferritin levels increase, blood pressure increases. Although, inverse relationship was observed between serum ferritin levels and BMI. The same result was observed when TSAT levels were studied in relation to blood pressure and BMI (Table 1).

Table (1): Demographic and clinical characteristics in patients with CHF and CKD in relation to s. ferretin level categories

Variables		<50 ng/ ml.		50–99 ng/ml.		100–299 ng/ml.		>300 ng/ml.		Statistical (F)	P value
		Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD		
Age (year)		57.05	8.11	56.9	8.99	58.31	8.70	58.79	8.24		
		NO	%	NO	%	NO	%	NO	%		
Number of patients (total 143)		**35**	**24.5**	**30**	**21.0**	**35**	**24.5**	**43**	**30.0**		
Sex	**Male**	23	66.0	19	63.0	27	77.0	25	58.0		
	Female	12	34.0	11	37.0	8	23.0	18	42.0		
Etiology	**Ischemic**	19	54.0	17	57.0	21	60.0	29	67.0		
	Dilated	16	46.0	13	43.0	14	40.0	14	33.0		
Risk factors	**Diabetes mellitus**	15	43.0	12	40.0	17	49.0	20	47.0		
	Hypertension	11	31.0	13	43.0	12	34.0	18	42.0		
	Dyslipidemia	13	37.0	11	37.0	13	37.0	17	40.0		
	Smoking	10	29.0	9	30.0	11	31.0	11	26.0		
SBP (mmHg)		133.16	2.24	140.39	4.42	140.39	4.38	140.61	4.72	34.153	<0.001**
DBP (mmHg)		69.37	3.18	72.39	4.40	72.81	4.48	73.06	4.89	6.586	0.001**
BMI (kg/m^2)		28.75	1.84	28.67	1.98	28.35	1.70	25.37	1.53	34.125	<0.001**

TSAT: transferrin saturation, SBP: systolic blood pressure, DBP: diastolic blood pressure, BMI: body mass index

There was a significant decrease in s. creatinine and blood urea, but eGFR increased as the serum ferritin level and transferrin saturation (TSAT) level increased. The other parameters such as thyroid function tests, HDL-C, LDL-C, triglycerides, total cholesterol, fasting blood sugar, hemoglobin A1c, and s. albumin, did not show any significant difference between the different s. ferritin or TSAT level categories (Table 2).

Table (2): Laboratory parameters and TSAT level categories

Variables	<15%		16%–20%		21%–25%		26%–35%		36%–45%		> 46 %		Statistical (F)	P value
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD		
S. Creatinine (mg/dl)	1.89	0.26	1.89	0.25	1.47	.07	1.47	.07	1.45	0.07	1.44	.05	23.23	<0.001**
eGFR (ml/min per 1.73 m2)	33.00	3.11	34.33	4.06	38.23	4.28	39.14	3.46	42.58	7.28	43.71	7.49	6.89	<0.001**
Blood urea (mg/dl)	69.00	0.00	68.07	0.62	65.33	1.00	62.38	1.56	59.25	0.59	57.27	0.98	558.56	<0.001**
FT3 (pg/mL)	2.81	0.49	2.89	0.53	3.02	0.59	2.40	0.37	2.89	0.64	2.67	0.48	1.82	0.113
FT4 (ng/dL)	1.22	0.21	1.21	0.22	1.22	0.26	1.33	0.15	1.24	0.24	1.34	0.21	.91	0.476
TSH (uIU/ml)	1.79	0.31	1.90	0.1	2.29	0.16	1.53	0.17	1.97	1.07	2.06	0.38	.93	0.464
FBG (mg/dl)	92.75	3.38	92.90	3.65	93.23	4.02	90.67	1.80	92.79	3.96	93.00	3.56	.70	0.626
HbA1c (%)	5.45	0.26	5.43	0.25	5.44	0.30	5.50	0.27	5.38	0.22	5.49	0.34	.33	0.895
Total cholesterol (mg/dl)	166.96	29.18	162.33	31.55	162.08	36.60	177.00	13.28	156.93	32.27	172.71	39.02	.69	0.634
LDL–C (mg/dl)	86.10	8.39	85.60	8.41	88.15	8.70	88.56	7.95	84.71	7.69	91.43	8.24	.97	0.439
HDL–C (mg/dl	37.52	4.18	37.17	4.18	37.69	3.99	39.33	3.46	37.79	4.06	39.57	2.99	.78	0.568
S. Triglycerides(mg/dl)	150.79	6.21	150.40	6.13	150.08	4.94	151.11	4.94	150.79	5.19	150.86	4.38	.06	0.997
Serum albumin(g/dl)	3.74	0.17	3.74	0.17	3.71	0.18	3.69	0.08	3.71	0.17	3.76	0.15	.30	0.914

There were statistically significant differences in NYHA functional class between s. ferretin level categories as there was significant improvement in NYHA functional class while ferretin level was increasing from less than 50 ng/ml to more than 300 ng/ml. The same result was observed with TSAT level categories (Table 3).

Table (3): NYHA functional class and ferretin level categories

Variables		<50 ng/ ml.		50–99 ng/ml.		100–299 ng/ml.		>300 ng/ml.		Statistical (F)	P value
		Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD		
NYHA	Class I	1.87	0.05	1.77	0.14	1.62	0.11	1.19	0.13	269.687	<0.001**
	Class II	2.90	0.02	2.86	0.07	2.76	0.04	2.38	0.18	195.114	<0.001**
	Class III	3.92	0.05	3.82	0.11	3.72	0.05	3.32	0.16	230.652	<0.001**
	Class IV	4.90	0.06	4.82	0.11	4.70	0.05	4.36	0.16	192.286	<0.001**
	Class V	5.89	0.06	5.79	0.11	5.68	0.07	5.34	0.16	181.403	<0.001**

There was a significant inverse correlation between s. ferretin level and (IVS thickness, GLPSS%, LVDd diameter, LVSd diameter, LVPW thickness) respectively. The same inverse correlation was found between these echocardiogenic parameters and TSAT level. However, there was a significant positive correlation between s. ferretin level and LVEF %. The same positive correlation was found between LVEF% and TSAT level (Table 4).

Table (4): Correlation between echocardiographic parameters and s. ferretin level

Echocardiographic parameters			Ferretin ng/ml. Mean	Ferretin ng/ml. ±SD	Statistical (R)
IVS (mm)	Mean	11.00	146.06	17.62	- 0.831**
	±SD	0.259			
LVPW (mm)	Mean	10.78			- .859**
	±SD	0.22			
LVDd (mm)	Mean	68.25			- .878**
	±SD	3.82			
LVSd (mm)	Mean	59.06			- .962**
	±SD	6.75			
LVEF (%)	Mean	31.91			.859**
	±SD	5.44			
AO (mm)	Mean	3.47			.114
	±SD	0.198			
LAD (mm)	Mean	4.51			-.047
	±SD	0.194			
GLPSS%	Mean	-12.08			- .898**
	±SD	1.34			

**Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).

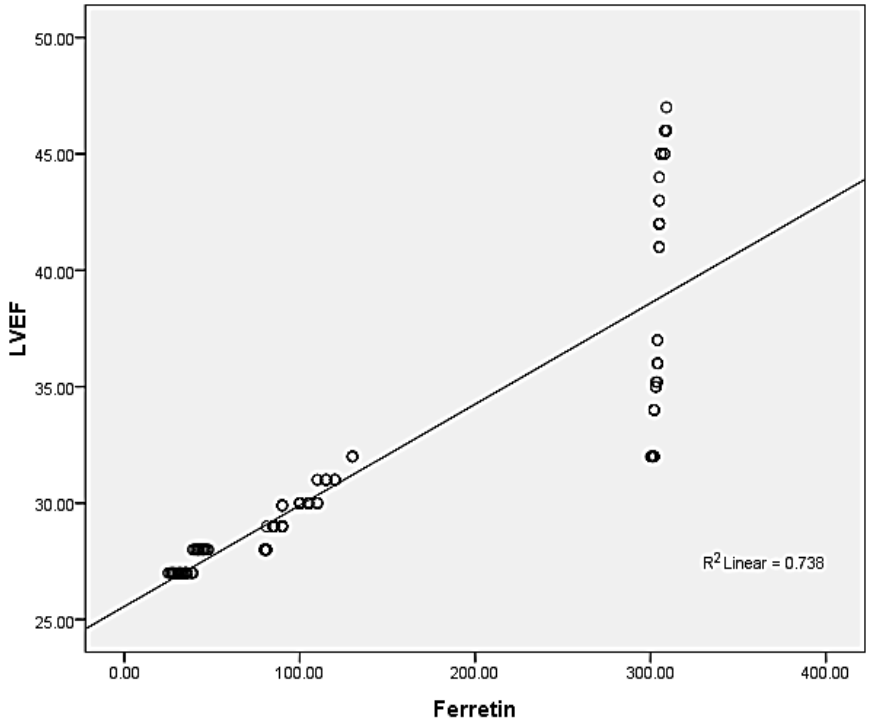

Figure (1): A direct correlation between LVEF% and S. ferretin level

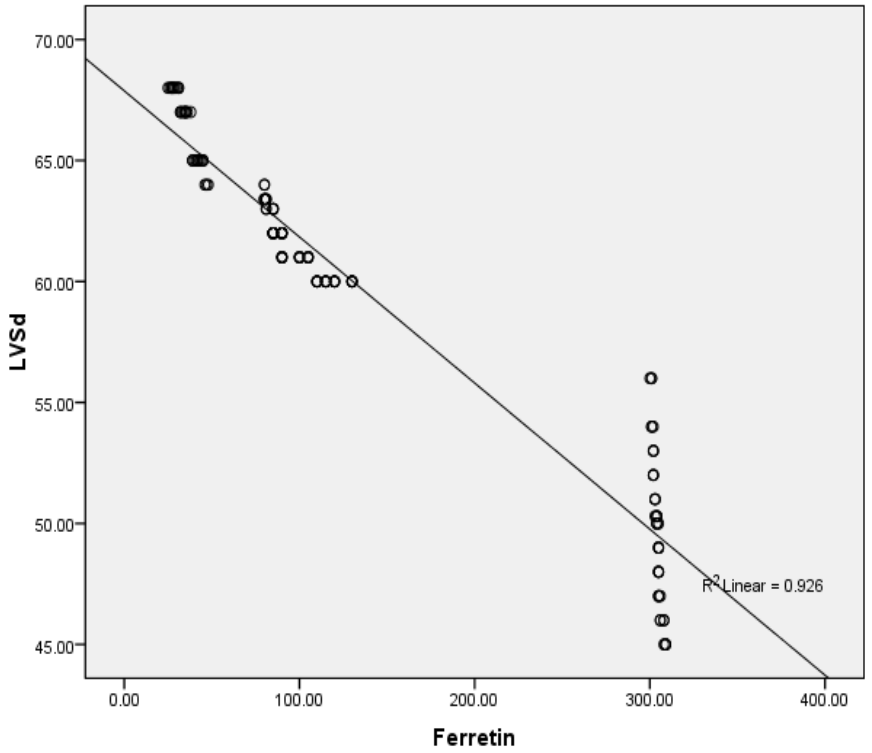

Figure (2): Inverse correlation between LVSD% and S. ferretin level

LVEF was statistically substantially higher in patients taking iron sucrose at the conclusion of the trial compared to their own baseline, according to echocardiographic examination. Over the course of the three-month investigation, the interventricular septum (IVS), LVEF%, LV systolic diameter (LVSd), LV diastolic diameter (LVDd), LV posterior wall (LVPW), and all other parameters were unaltered in the control group. The iron sucrose group, on the other hand, had a substantial reduction in LVSd and LVDd throughout the course of the three-month trial. Notably, LVPW and IVS thickness in the iron sucrose group were significantly reduced. There was favourable and significant decrease in global longitudinal peak systolic strain over the 3-month study period in iron sucrose group relative to their own baseline and relative to control group (Table 5).

Table (5): Serial changes and comparison of echocardiographic parameters in patients with placebo (control group) and patients with iron sucrose treatment over the 3 months period of the study

Variables	Control Group								Iron Sucrose Group								Statistical test (T)			P value		
	Baseline		4w		8w		12w		Baseline		4w		8w		12w							
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	4w	8w	12w	P1	P2	P3
IVS (mm)	11.10	.24	11.10	.24	11.09	0.25	11.09	.23	11.11	.24	11.09	.24	10.85	.43	10.78	.13	.042	3.44	8.45	0.967	0.001**	0.000**
LVPW(mm)	10.87	.16	10.88	.16	10.91	.19	10.88	.17	10.87	.16	10.85	.14	10.71	.36	10.54	.15	.809	3.56	11.13	0.420	0.001**	0.0001**
LVDd (mm)	69.20	3.07	69.76	3.23	69.04	3.10	69.62	3.12	69.64	3.15	67.27	2.82	65.97	3.34	64.52	2.85	4.11	4.78	8.54	0.0001**	0.0001**	0.0001**
LVSd (mm)	62.99	2.48	62.96	2.68	63.22	2.64	63.14	2.67	63.10	2.70	58.84	2.41	53.78	5.93	49.49	3.07	8.09	10.30	23.75	0.0001**	0.0001**	0.0001**
LVEF (%)	28.99	1.64	28.95	1.59	29.03	1.59	28.97	1.63	28.99	1.61	29.78	1.41	31.18	1.18	33.76	2.16	2.75	7.67	12.51	0.007**	0.0001**	0.0001**
AO(mm)	3.41	.254	3.39	.244	3.43	.260	3.44	.235	3.42	.26	3.43	.32	3.44	.311	3.45	0.30	.231	.716	.477	0.818	0.476	0.634
LAD(mm)	4.53	.259	4.50	.132	4.49	.168	4.6	.26	4.53	.25	4.49	.128	4.50	.142	4.50	.16	.308	.449	.241	0.759	0.655	0.810
GLPSS%	-10.63	.188	-10.58	.232	-10.66	.192	-10.60	.183	-10.62	..19	-12.38	.258	-13.21	.16	-14.05	.164	40.87	78.79	103.81	0.0001**	0.0001**	0.0001**

IVS: interventricular septum LVDd: left ventricular diastolic diameter LVEF: left ventricular ejection fraction LVPW: left ventricular posterior wall LVSd: left ventricular systolic diameter LAD : left atrial diameter AO: Aorta GLPSS: Global Longitudinal Peak Systolic Strain.

P1=statistically significant difference between control group and IS group at week 4.

P2=statistically significant difference between control group and IS group at week 8.

P3=statistically significant difference between control group and IS group at week 12.

DISCUSSION

There was a statistically significant direct relationship between serum ferritin levels and blood pressure. This means that as serum ferritin levels increase, blood pressure increases. Although, inverse relationship was observed between serum ferritin levels and BMI. The same result was observed when TSAT levels were studied in relation to blood pressure and BMI. This is inconcordant with **Koo *et al.* [11] and Amaechi *et al.* [12]** who found that there was no significant difference in the blood pressure between s. ferretin groups.

Koo *et al.* [11] found that there was mild significant decrease in BMI with the increase of s. ferretin level. While, this is inconcordant with **Amaechi *et al.* [12]** who found that there was no significant difference in BMI with the increase of s. ferretin level.

There was a significant decrease in serum creatinine and blood urea, but eGFR increased as the serum ferritin level and TSAT level increased. This suggests that higher levels of serum ferritin and TSAT are associated with better kidney function. This is inconcordant with **Koo *et al.* (11)** who found that s. creatinine levels were significantly lower in the lowest s. ferretin group compared to the highest s. ferretin group. This may be explained with different cut off values of s. ferretin being higher in his study leading to iron toxicity.

Patients with a s. ferritin level of less than 50 ng/mL compared to those with a level of more than 300 ng/mL had a substantially higher NYHA functional class. This indicates that patients' chances of developing a more severe type of HF increased with decreased blood ferritin levels. This is concordant with **Amaechi *et al.* [12]** who found that there was significant improvement of NYHA functional class with increase of s. ferretin level.

Similarly, patients with a TSAT level of less than 15% had a significantly higher NYHA functional class than patients with a TSAT level of more than 46%. This implies that patients' chances of developing a more severe type of HF were also increased by their lower TSAT levels.

Significant inverse link was found between the levels of s. ferretin and each of IVS thickness, GLPSS%, LVDd diameter, LVSd diameter, LVPW thickness. This means that as serum ferritin level increases, these echocardiogenic parameters decrease. The same inverse correlation was found between these echocardiogenic parameters and TSAT level.

The level of s. ferretin and LVEF% had a strong positive connection. This indicates that when serum ferritin levels rise, so does LVEF. A similar positive connection was discovered between LVEF% and TSAT level.

According to **Das *et al.* [13]**, patients who had greater blood ferritin levels had larger LVDs and worse EF. A few investigations, like those by **Usha *et al.* [14] and Khalilian *et al.* [15]**, found no statistically significant relationship between blood ferritin levels and echocardiographic parameters. This could be explained by different study population number (being lower in their studies) and different cut off values of s. ferretin used in their studies (being higher in their studies), which may lead to iron overload in cardiac muscle and iron toxicity.

Echocardiographic evaluation revealed that LVSd (62.99 ± 2.48 and 63.14 ± 2.67) for baseline and 12 weeks respectively, LVDd (69.20 ± 3.07 and 69.62 ± 3.12) for baseline and 12 weeks respectively, LVPW (10.87± 0.16 and 10.88 ± 0.17) for baseline and 12 weeks respectively, IVS (11.10± 0.24 and 11.09 ± 0.23) for baseline and 12 weeks respectively and EF% (28.99 ± 1.64 and 28.97± 1.63) for baseline and 12 weeks respectively remained unchanged ($p > 0.05$) in the control group over the three-month study period. This is concordant with **Toblli *et al.* [16]** who found, the iron sucrose group's LVSd and LVDd were statistically substantially higher than baseline ($p<0.01$) whereas their EF% was considerably higher ($p<0.01$).

As opposed to that, both LVSd (63.10± 2.70 and 49.49 ± 3.07) for baseline and 12 weeks respectively and LVDd (69.64± 3.15 and 64.52 ± 2.85) for baseline and 12 weeks respectively, were significantly decreased in the iron sucrose group ($p<0.01$) from baseline to the 3-month investigation. As regard EF% (28.99 ± 1.61 and 33.76 ± 2.16) for baseline and 12 weeks respectively was significantly improved in iron sucrose group ($p<0.01$). This is concordant with **Toblli *et al.* [16]** who found that both LVSd and LVDd were significantly decreased in the iron sucrose group ($p<0.01$) relative to baseline with statistically significant increase in EF% ($p<0.01$).

Notably in our study, LVPW (10.87 ± 0.16 and 10.54 ± 0.15) for baseline and 12 weeks respectively and IVS thickness (11.11± 0.24 and 10.78 ± 0.13) were significantly decreased in the IS group ($p<0.01$). This is in concordant with **Toblli *et al.* [16]** who found that despite being clinically significant, the iron sucrose group's change in LVPW and IVS thickness did not approach statistical significance.

This is the first study to evaluate myocardial function with speckle tracking echocardiographic imaging using GLPSS (global longitudinal peak systolic strain) as a new parameter.

GLPSS was significantly decreased throughout our study period indicating shortening of cardiac muscle fibers and improvement of cardiac muscle function in iron sucrose group (-10.63± 0.19 and -14.05± 0.164) for baseline and 12 weeks respectively ($p<0.01$). In comparison to control group, GLPSS remained unchanged (-10.63± 0.19 and -10.60 ± 0.183) for baseline and 12 weeks respectively ($p>0.05$).

A positive and statistically significant change in NYHA functional class was also observed (3.10± 0.23 and 2.04 ± 0.18) for baseline and 12 weeks respectively in response to iron sucrose at three months' follow-up ($p<0.01$) as a result of improved cardiac muscle

function in iron sucrose group. On the contrary, NYHA functional class (3.10 ± 0.23 and 3.10 ± 0.24) over the course of the trial, baseline and 12 weeks, respectively, remained constant in the control group. This is consistent with the findings of **Toblli *et al.* [16] and Gaber *et al.* [17],** who discovered that only patients receiving intravenous iron improved in NYHA functional class.

These results, according to our theory, result from patients receiving intravenous iron having better hemodynamics because of enhanced Hb and iron metabolism, which in turn leads to higher tissue oxygenation. Additionally, the intervention group required less diuretics during the three-month follow-up, indicating improved heart function.

It is important to note in this context that the statistically significant correlation between echocardiographic parameters and change in TSAT in response to intravenous iron offers further support for a potential link between cardiac structure/function and iron status. These findings are corroborated by the improvement in LVSd, which helped to enhance LVEF and GLPSS. As a result, IS appears to favourably alter the morbid processes that go along with a drop in LVEF.

CONCLUSION

These findings imply that individuals getting the best care for CHF who have iron deficiency and CKD will benefit from intravenous iron on heart dimensions and myocardial functional measures.

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Competing interests: Nil.

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