Cardiac functions in children with iron deficiency anemia

Demir eksikliği anemili çocuklarda kardiyak fonksiyonlar

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Abstract
In this study, we aimed to assess the effects of iron deficiency (ID) on cardiac functions with M-mode, pulse Doppler, and tissue Doppler and to compare the findings with healthy individuals. Twenty one children with only ID (prelatent ID + latent ID + manifest ID/anemia) and completely healthy 23 children were included in the study. Of all patients, complete blood counts were studied, and serum iron (Fe), iron binding capacity (FeBC), and ferritin levels were measured. Echocardiographic assessment was performed for those who were diagnosed with Fe deficiency at the beginning of the study and after there covery of anemia and once at the beginning of the study for those who were healthy. There were significant differences between control group values and pre-treatment values of patients with manifest ID in terms of hemoglobin (Hgb), hematocrit (Hct), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red blood cell distribution width (RDW), ferritin, Fe, FeBC, aortic diameter (AD), and left atrial diameter (LAD). Between the pre- and post-treatment values of manifest ID (anemia) patients, there were significant differences in terms Hgb, Hct, MCV, MCH, MCHC, ferritin, Fe, FeBC, AD, LAD, left ventricular end systolic diameter, left ventricular end diastolic diameter, septal thickness, posterior wall thickness, ejection fraction, fractional short time, mitral, tricuspid, and septal isovolumetric relaxation time and septal isovolumetric contraction time. Diastolic and systolic cardiac dysfunction occurs at the stage of manifest ID where the value of Hgb decreased and these changes recover with treatment.
Keywords: Anemia; cardiac; children; iron

Özet
Biz bu çalışmada; demir eksikliğinin (ID) kardiyak fonksiyonlar üzerine olan etkilerini, M-mode, pulse Doppler ve doku Doppler ile değerlendirip sonuçlarını sağlıklı bireylerle karşılaştırmayı amaçladık. Çalışmaya sadece ID'sı (prelatent ID + latent ID + belirgin ID/anemi) olan 21 ve tamamen sağlıklı olan 23 çocuk alındı. Bütün hastaların tam kan sayımları yapıldı, serum demir (Fe), demir bağlama kapasitesi (FeBC) ve ferritin düzeyleri ölçüldü. Demir eksikliği saptananlara çalışmanın başında ve anemisi düzeldikten sonra, sağlıklı olanlara ise çalışmanın başında bir defa ekokardiyografik değerlendirme yapıldı. Kontrol grubu ile belirgin ID'si olanların tedavi öncesi değerleri karşılaştırıldığında; hemoglobin (Hgb), hematokrit (Hct), ortalama eritrosit hacmi (MCV), ortalama eritrosit hemoglobini (MCH), ortalama eritrosit hemoglobin konsantrasyonu (MCHC), kırmızı küre dağılım aralığı (RDW), ferritin, Fe, FeBC, aort çapı (AD) ve sol atrium çapı (LAD) değerleri arasında anlamlı farklılık saptandı. Belirgin ID'si (anemi) olan hastaların tedavi öncesi ve sonrası değerleri karşılaştırıldığında ise; Hgb, Hct, MCV, MCH, MCHC, ferritin, Fe, FeBC, AD, LAD, sol vent sistol sonu çapı, sol ventrikül diastol sonu çapı, septum kalınlığı, arka duvar kalınlığı, ejeksiyon fraksiyonu, fraksiyonel kısa zaman, mitral, triküspit ve septal izovolümetrik gevşeme zamanı ile septal izovolümetrik kasılma zamanı değerleri açısından anlamlı farklılıklar vardı. Hgb miktarının düştüğü belirgin ID evresinde diastolik ve sistolik kardiyak disfonksiyon gelişmekte ve bu değişiklikler tedavi ile düzelmektedir.
Anahtar kelimeler: Anemi; kardiyak; çocuklar; demir

Introduction

Anemia is described as a more than -2 SD reduction in hemoglobin (Hgb) concentration, in hematocrit (Hct), or in the number of erythrocytes in a cubic millimeter according to age and gender in proportion to normal population (1). Anemias are divided into three groups as microcytic, normocytic, and macrocytic according to mean corpuscular volume (MCV). The most common microcytic anemia is the iron deficiency anemia (IDA) (2).

In severe anemia, the amount of oxygen provided to the body is reduced with the decrease in Hgb levels (3). This situation activates some compensatory hemodynamic (diminish in vascular resistance and viscosity, vasodilatation due to hypoxia, increase in cardiac output and heart rate, increase in nitric oxide activity, stimulation of angiogenesis) and non-hemodynamic (increase in erythropoietin production, increase in oxygen release to tissues with a shift of hemoglobin-oxygen curve to the right)

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mechanisms (4). If these compensatory mechanisms are inadequate, anemia may lead to cardiac problems. When anemia develops in those with anatomical cardiac problems, cardiac problem may precipitate and become decompensated. On the other hand, heart failure may occur even in those who are cardiologically normal, depending on erythrocyte mass (5) such that anemia has been shown to be an independent risk factor for development of congestive failure (6) and cardiomyopathy (7).

The study presented was planned in order to assess the effects of iron deficiency (ID) on cardiac functions with M-mode, pulse Doppler, and tissue Doppler and to compare the findings with healthy individuals.

Materials and Methods

Ethical Declaration

The study was approved by the Scientific Researches Ethical Committee Medical Faculty of our university (decision no: B.30.2.SEL.0.28.00.00/130-176). Informed consent and signed voluntary participation form was received from the families of all patients.

Patients and Study Design

Twenty one patients, who applied to our clinic for any reason and in whom any problem other than ID was not encountered in physical and laboratory examinations, and age and gender matched 23 totally healthy children were included into this prospective study. Complete blood counts of all patients were perfomed; their serum iron (Fe), iron binding capacity (FeBC), and ferritin levels were measured. In order to diagnose ID, hematological parameters of each patient were assessed one by one according to reference values corresponding with their own age group and gender and the values below -2 SD were accepted as abnormal (1). Later all anemic cases were grouped according to the grade (prelatent, latent, manifest/anemia) of anemia (8). Among patients in whom Fe deficiency was identified, echocardiography (M-mode, pulse Doppler, and tissue Doppler) was performed at the onset of the study; subsequently Fe therapy (3-6 mg/kg/day, Fe^{+2}) for anemia has been started. Approximately 3 months later (with more frequently in those with manifest anemia), hematologic parameters were re-evaluated. Within this period, cardiac re-evaluation was performed in patients whose ID was recovered. In patients who did not recover, therapy was continued, evaluation was repeated 1 to 2 months later according to degree of deficiency, and cardiac evaluation was performed after the recovery of anemia. While hematologic values were obtained from examinations carried out during routine screening in healthy group, cardiac evaluation was performed once at the onset of the study. Patients with an additional problem during cardiac evaluation in both groups were excluded from the study. Data obtained by the aid of a statistics software (SPSS, version 16.0) was compared in the manner of control group-all patient groups (prelatent ID + latent ID + manifest ID), control group- pre-treatment values of patient group with manifest anemia only, control group-post-treatment values of patient group with manifest anemia only, and pre- and post-treatment values of patients with manifest anemia.

Laboratory Studies

All laboratory parameters were studied on the day when blood was obtained from patient. For complete blood count, Abbott Diagnostics, Cell-Dyn 3700, for Fe and FeBC, Abbott, Architect C 16000, and for ferritin, Siemens, Advia Centaur XP devices were used.

Cardiac Evaluation

All patients were examined echocardiographically by the same pediatric cardiologist. Cardiologist was blind to clinical information of patient during measurements. All echocardiographic evaluations were carried out by means of Toshiba, Aplio50, Japan device equipped with 3-5-6.5 MHz transducers. Systolic functions were assessed by M-mode method, diastolic functions were by pulse and tissue Doppler method. Records of patients were obtained while lying in the supine and left lateral position. Each parameter was measured three times and the averages of these measurements were obtained. Measurement technique was accordant with standard imaging technique (9) suggested by American Society of Echocardiography. Left atrial size and aortic size were measured from the long parasternal axis window by M-mode echocardiography. Images were recorded in the apical 4-spaces (left ventricle, lateral mitral, lateral tricuspid annulus, and basal segment of interventricular septum) position.

Statistical Analysis

Statistical analyses of data obtained with the study were performed with SPSS, version 16.0 software. In comparison of groups; Mann-Whitney Test from non-parametric tests was used for parameters of which normal distribution approach was unable to be achieved and Independent T Test from parametric tests was used for parameters of which normal distribution approach was achieved. In the comparison of two dependent groups Two Related Samples Test from non-parametric tests was used for parameters of which normal distribution approach was unable to be achieved and Paired Samples T Test from parametric tests was used for parameters of which normal distribution approach was achieved. Furthermore, several characteristic properties presented in the study were assessed by means of descriptive statistical tests. The $P<0.005$ values were accepted as statistically significant.

Results

There were 23 children in control group and 21 children in all patient groups (prelatent ID + latent ID + manifest ID/anemia). While the female patient

ratios in control and patient groups were 47.8% and 47.6%, respectively, the male patient ratios were 52.2% and 52.4%, respectively. The mean age of controls was 9.04±4.65 years with a range of 2 to 18 years; while the mean age of patient group was 9.04±6.64 years with a range of 1 to 17 years. Both groups were compatible in terms of age (P>0.05) and gender (P>0.05). When the values of control group and all patient groups (prelatent ID + latent ID + manifest ID/anemia) were compared, significant differences existing in terms of Hgb, Hct, MCV, mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red blood cell distribution width (RDW), ferritin, Fe, and FeBC were not present in any of the cardiac parameters (Table 1). Twenty three child (63.9%) were present in control group, and 13 child (36.1%) were present in impressive ID group. While the female patient ratios from both groups were 47.8% and 46.2%, respectively, the male patient ratios were 52.2% and 53.8%, respectively. The mean age of controls was 9.04±4.65 years with a range of 2 to 18 years; while the mean age of patient group was 11.30±6.22 years with a range of 1 to 17 years. Both groups were compatible in terms of age (P>0.05) and gender (P>0.05). When the control group values and the pre-treatment values of patients with manifest anemia were compared, there were significant differences in

Table 1. The parameters with significant difference in comparison of control and all patient groups (prelatent ID + latent ID + manifest ID/anemia)

	Control group (n=23)		Patient group (n=21) (prelatent ID + latent ID + manifest ID/anemia)		P value
	Mean	Interval	Mean	Interval	
Age (Year)	9.04±4.65	2-18	9.04±6.64	1-17	0.906
Gender (F/M)	47.8% / 52.2%	11-12	47.6% / 52.4%	10-11	0.613
Hgb (g/dl)	13.75±1.19	11.70-17	10.04±1.94	6.20-13.6	**0.0001**
Hct (%)	40.73±3.5	34.50-51.0	33.88±4.76	25.50-42.0	**0.0001**
MCV (fl)	78.40±3.60	70.20-85.90	68.87±7.71	52-82	**0.0001**
MCH (pg)	26.13±1.78	22.60-30.10	21.73±3.65	13.6-28.40	**0.0001**
MCHC (g/dl)	33.35±1.56	28-35.20	31.26±2.61	23.4-35	**0.02**
RDW (%)	13.16±0.71	12-14.90	17.02±2.34	13.40-23.3	**0.0001**
Ferritin (ng/ml)	37.30±23.34	14.10-100	5.28±2.23	1.29-12	**0.0001**
Fe (µg/dl)	85.26±28.68	45-139	15.23±8.80	3-38	**0.0001**
FeBK (µg/dl)	267.47±57.77	170-375	403.31±72.83	239-500	**0.0001**

F, Female; Fe, Iron; FeBC, Iron Binding Capacity; Hct, Haematocrit; Hgb, Haemoglobin; M, Male, MCH, Mean Corpuscular Haemoglobin; MCHC, Mean Corpuscular Haemoglobin Concentration; MCV, Mean Corpuscular Volume; RDW, Red blood cell Distribution Width.

Table 2. The parameters with significant difference in comparison of control group values and pre-treatment values of manifest ID group

	Control group (n=23)		Patient group (n=13) (Manifest ID- pre-treatment)		P value
	Mean	Interval	Mean	Interval	
Age (Year)	9.04±4.65	2-18	11.30±6.22	1-17	0.224
Gender (F/M)	47.8% / 52.2%	11-12	46.2% / 53.8%	7-6	0.599
Hgb (g/dl)	13.75±1.19	11.70-17	9.34±1.49	6.20-11.7	**0.0001**
Hct (%)	40.73±3.5	34.50-51.0	32.06±4.61	25.50-42.0	**0.0001**
MCV (fl)	78.40±3.60	70.20-85.90	67.37±7.94	52-78	**0.0001**
MCH (pg)	26.13±1.78	22.60-30.10	20.47±3.63	13.6-26.0	**0.0001**
MCHC (g/dl)	33.35±1.56	28-35.20	30.03±2.49	23.4-33	**0.0001**
RDW (%)	13.16±0.71	12-14.90	17.62±2.53	13.40-23.3	**0.0001**
Ferritin (ng/ml)	37.30±23.34	14.10-100	4.85±2.65	1.29-12	**0.0001**
Fe (µg/dl)	85.26±28.68	45-139	10.76±4.04	3-16	**0.0001**
FeBC (µg/dl)	267.47±57.77	170-375	417.18±77.47	257-500	**0.0001**
AD (mm)	20.61±3.55	16-27	23.07±3.09	16-27	**0.048**
LAD (mm)	23.95±4.05	18-32	27.38±3.06	20-34	**0.018**

AD, Aortic diameter; F, Female; Fe, Iron; FeBC, Iron Binding Capacity; Hct, Haematocrit; Hgb, Haemoglobin; LAD, Left Atrial Diameter; M, Male; MCV, Mean Corpuscular Volume; MCH, Mean Corpuscular Haemoglobin; MCHC, Mean Corpuscular Haemoglobin Concentration; RDW, Red blood cell Distribution Width.

Table 3. The parameters with significant difference in comparison of control group values and post-treatment values of manifest ID group

	Control group (n=23)		Patient group (n=13) (Manifest ID-post-treatment)		P value
	Mean	Interval	Mean	Interval	
Age (Year)	9.04±4.65	2-18	11.30±6.22	1-17	0.224
Gender (F/M)	47.8% / 52.2%	11-12	46.2% / 53.8%	7-6	0.599
Hgb (g/dl)	13.75±1.19	11.70-17	12.76±0.95	11.50-14.2	**0.015**
RDW (%)	13.16±0.71	12-14.90	17.41±3.08	13.80-23.70	**0.0001**

F, Female; Hgb, Haemoglobin; M, Male; RDW, Red blood cell Distribution Width

Table 4. The parameters with significant difference in comparison of pretreatment and posttreatment values of manifest ID group

	Patient group (n=13), (Manifest ID- pre-treatment)		Patient group (n=13) (Manifest ID- post-treatment)		P value
	Mean	Interval	Mean	Interval	
Age (Year)	11.3±6.22	1-17	11.3±6.22	1-17	1.000
Gender (F/M)	53.8% / 46.2%	7-6	53.8% / 46.2%	7-6	1.000
Hgb (g/dl)	9.34±1.49	6.20-11.7	12.76±0.95	11.50-14.2	**0.0001**
Hct (%)	32.06±4.61	25.50-42.0	38.62±3.13	33.90-45.30	**0.003**
MCV (fl)	67.05±8.21	52.0-78.0	75.66±5.83	69.2-89	**0.014**
MCH (pg)	20.47±3.63	13.60-26.0	25.38±2.74	22.4-30.80	**0.002**
MCHC (g/dl)	30.03±2.49	23.4-33.0	33.21±1.30	30.9-34.8	**0.004**
Ferritin (ng/ml)	4.85±2.65	1.29-12	35.88±18.87	13-84	**0.0001**
Fe (µg/dl)	10.76±4.04	3-16	87.23±46.55	30-193	**0.0001**
FeBC (µg/dl)	417.18±77.47	257-500	249.72±64.02	136-328	**0.001**
AD (mm)	23.07±3.09	16-27	20.00±4.58	16-30	**0.03**
LAD (mm)	27.38±3.66	20-34	22.84±4.93	16-33	**0.007**
LVESD (mm)	24.15±4.96	15-32	19.38±5.47	11-31	**0.024**
LVEDD (mm)	40.69±6.40	28-48	33.46±8.33	21-48	**0.007**
ST (mm)	7.46±1.03	6-9	6.5±1.39	5-9.5	**0.027**
PWT (mm)	7.46±1.06	6-9	6.23±1.54	5-9	**0.004**
EF (%)	70.76±5.93	60-84	74.92±5.02	62-80	**0.026**
FS (%)	37.92±4.19	30-44	42.84±4.07	32-47	**0.003**
M-IVRT (msn)	47.61±6.31	32-56	42.07±5.13	35-53	**0.027**
T-IVRT (msn)	47.91±8.10	35.0-67.0	40.75±6.56	32-53	**0.025**
S-IVCT (msn)	49.23±9.86	32.0-67.0	42.46±10.46	32-67	**0.034**
S-IVRT (msn)	51.07±11.35	35.0-80.0	41.84±8.66	32-61	**0.014**

AD, Aortic diameter; EF, Ejection Fraction; F, Female; Fe, Iron; FeBC, Iron Binding Capacity; FS, Fractional Short Time; Hct, Haematocrit; Hgb, Haemoglobin; LAD, Left Atrial Diameter; LVEDD, Left Ventricular End-Diastolic Diameter; LVESD, Left Ventricular End-Systolic Diameter; M, Male; MCH, Mean Corpuscular Haemoglobin; MCHC, Mean Corpuscular Haemoglobin Concentration; MCV, Mean Corpuscular Volume; M-IVRT, Mitral Isovolumic Relaxation Time; PWT, Posterior Wall Thickness; S-IVCT, Septal Isovolumic Contraction Time; S-IVRT, Septal Isovolumic Relaxation Time; ST, Septal Thickness; T-IVRT, Tricuspid Isovolumic Relaxation Time.

between the values of Hgb, Hct, MCV, MCH, MCHC, RDW, ferritin, Fe, FeBC, aortic diameter (AD), left atrial diameter (LAD) (Table 2). When the control group values and the post-treatment values of patients with manifest ID were compared, there was significant difference between Hgb and RDW values only. No significant difference was present in any of cardiac parameters (Table 3).

The number of patients with manifest ID was 13. Of those 13 patients, 46.2% were male, 53.8% were female. The mean age of those patients was 11.3±6.22 years. When the pre- and post-treatment values of these patients were compared, there were significant differences in between the values of Hgb, Hct, MCH, MCH, MCHC, ferritin, Fe, FeBC, AD, LAD, Left Ventricular End-Systolic Diameter (LVESD), Left Ventricular End-Diastolic Diameter (LVEDD), Septal Thickness (ST), Posterior Wall Thickness (PWT), Ejection Fraction (EF), Fractional Short Time (FS), Mitral Isovolumic Relaxation Time (M-IVRT), Tricuspid Isovolumic Relaxation Time (T-IVRT), Septal Isovolumic Contraction Time (S-IVCT), and Septal Isovolumic Relaxation Time (S-IVRT) (Table 4).

Discussion

While IDA is the most frequent blood disease (10) of childhood, elementary Fe deficiency is the most seen nutritional deficiency in the World (11). Iron defficiency occurs in three stages: In the prelatent stage, Hct and serum Fe levels are normal; however, ferritin level is low. In the latent stage, serum Fe level decreases, FeBC increases; however, there is no change in Hct. In the manifest (anemia) stage, microcytosis and hypochromia owing to the long-lasting Fe deficiency occur (8).

Iron is a basic element for some functions such as electron transfer reactions, gene regulations, immune functions, cell growth and differentiation, and oxygen binding and transport (11). In IDA, adequate energy cannot be produced in heart, due to inadequate oxygen (3) presentation and disturbed mitochondrial enzymes (12). In this case, first relaxation phase is affected; since only relaxation phase depends on energy but not the contraction phase. The reason of this is the necessity of a large quantity of energy for actin cross-bridges to be separated from myosin in relaxation phase (13). As relaxation also represents diastolic features, diastolic dysfunction may be predicted to occur in IDA.

Diastolic dysfunction is a manifestation characterized by failure of left ventricle filling, where normal end-systolic pressure is present, but filling pressure does not increase appropriately (14). A ventricle with a thick wall, concentric hypertrophy, small cavity, and restricted filling is present (15). Pathophysiologically it is examined in three phases: early dysfunction due to abnormalities in left ventricle relaxation and elastic structure, intermediate dysfunction as a result of left ventricle passive stiffness and compliance, and late dysfunction owing to left atrial contraction (14). The reasons are compression (pericardial, tumoral, or pleural) and impairment of wall elasticity (edema, fibrosis, amyloidosis, hemosiderosis etc.) (16).

Hypertrophy occurring in anemia is the result of both increasing preload due to water and salt retention

and increasing velocity and contractility owing to sympathetic nervous system (17). When the pre- and post-treatment values of patient group with manifest anemia were compared, a statistically significant increase was also present in both ST and PWT values in our study. Emerged hypertrophy had recovered with treatment and that was compatible with the literature (4). Moreover, LAD and left ventricle end-diastolic pressure increase and diastolic filling parameters string out in diastolic dysfunction occurring owing to the pathophysiological mechanisms in IDA (18).

Herein, LAD enlargement is because of both diastolic dysfunction itself and decreasing erythrocyte mass (19). In our study, there was a significant difference in terms of left atrium size between the pre- and post-treatment values of patient group with manifest ID (anemia) and the pre-treatment values of patients with manifest ID and control group values. When the post-treatment values of the same patient group and control group values were compared, this difference was lost. This also indicates that diastolic dysfunction and increased left atrium size occur in manifest ID stage.

Increase in the left ventricle end-diastolic pressure is the result of increases in preload and cardiac output. As the metabolic requirements also increases on the other hand, cardiac dilatation occurs while peripheral resistance and afterload decrease (18). In the study also, we presented, a significant difference was present in terms of LVEDD between the pre- and post-treatment values of patient group with manifest ID. This difference was not present when the post-treatment values of patients with manifest ID and control group or of all patients groups and control group were compared. End-systolic and diastolic volume indexes represent preload of the left ventricle and these indexes increase in anemia (20). When the pre- and post-treatment values of patients with manifest ID were compared in our study, there was significant differences in both LVESD and LVEDD.

We studied peak velocity of the early diastolic filling wave (E) and peak velocity of the atrial filling wave (A) from diastolic filling parameters. Increase in E/A ratio in severe anemia indicates the alterations in left ventricle diastolic function (20). However, no significant difference was present in terms of E and A waves in any groups in statistical comparison performed in our study.

IVRT is related to decrease in ventricular stiffness and viscosity (14). Both IVRT and IVCT change in diastolic dysfunction (21). When the pre- and post-treatment values of patient group with manifest ID were compared in our study, significant differences were identified in M-IVRT, T-IVRT, S-IVRT, and S-IVCT. These differences were not present between post-treatment group and control group.

Systolic dysfunction refers to inappropriate end-systolic pressure resulting in systemic hypoperfusion (14). In systolic dysfunction, ejection is impaired due to a dilated ventricle with eccentric hypertrophy in general and output is reduced (15). When the pre- and post-treatment values of patient group with manifest ID were compared in our study, there were significant differences in both EF and FS.

Mild and moderate anemia has no prominent effects on cardiovascular system (17). When the pretreatment values (pre-latent ID + latent ID + manifest ID/anemia) of all patient groups and our control group were compared, there were significant differences in only hematological parameters (Hgb, Hct, MCV, MCH, MCHC, RDW, ferritin, Fe, FeBC). However, when control group values were compared with the pretreatment values of patient group with manifest anemia, there were significant differences in terms of AD and LAD in addition to the same hematological parameters. This also suggests us that cardiac alterations may be associated with advanced stages of anemia where Hgb also decreases. On the other hand, when control group values were compared with the post-treatment values of patient group with manifest ID, significant differences existing in echocardiographic parameters were determined to be lost while statistically significant difference was still present in the Hgb value. Recovery of echocardiographic values without complete recovery of the Hgb values may be contrary to the opinion above; quite the contrary, however, it supports the same opinion because, when considered, post-treatment Hgb is established to elevate significantly in proportion to the pre-treatment Hgb of patient group with advanced anemia and, based on this, the echocardiographic values are observed to become normal.

When the pre- and post-treatment values of patient group with manifest ID were compared in our study, all impairments occurring in echocardiographic parameters were determined to recover significantly after treatment. This situation may be suggestive of that cardiac problems occurring due to iron deficiency are not persistent; however, it may also be related to the short duration of anemia.

In conclusion, both diastolic and systolic cardiac dysfunction develop possibly under a specific Hgb value and in the stage of manifest iron deficiency (anemia) where the Hgb value also decreases; however, all these changes improve with treatment. Nevertheless, this recovery may also arise from short duration of anemia. Therefore, the studies which will be carried out in a longer period and with a wide range of patient groups are warranted.

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