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Cardiac evaluation in children with malnutrition

Malnütrisyonlu çocuklarda kardiyak değerlendirme

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Abstract

Aim: The main purpose of this study was to identify myocardial changes in malnourished children.

Material and Methods: This prospective study included 47 patients with malnutrition and 44 healthy controls. The subjects who had malnutrition were classified according to the method of Gomez and Waterlow. Electrocardiographic and echocardiographic examinations, 24-h Holter monitoring, and biochemical assessments were performed in all subjects.

Results: The malnutrition group included 20 (42.5%) males, and the control group included 19 (43.1%) males (p<0.05). There was no difference between the malnutrition and control groups with regard to mean age (69.4±57.3 months and 68.9±48.2 months, respectively, p=0.5). Although the left ventricular mass was lower in the patient group compared with the control group, the left ventricular mass index was not different (42.3±24.5 g, 53.4±23.9 g, p=0.049 and 60.7±13.3 g/m^2, 61.9±12.1 g/m^2, p=0.67, respectively). The left ventricular ejection fraction and fractional shortening were lower in the patient group compared with the control group (66.2±5.3%, 69.2±4.07%, p=0.04 and 35.4±4.2%, 37.9±3.4%, p=0.03, respectively). The myocardial performance index was higher in the patient group (0.45±0.09, 0.36±0.05, respectively, p=0.001). The deterioration of cardiac functions was associated with the severity and duration of malnutrition. Troponin concentrations were not elevated in any patients. The corrected QT dispersion was significantly higher in patients with malnutrition (47.9±16.8, 32.9±10.6, respectively, p=0.001). Complex ventricular arrhythmias were not noted in any patients.

Conclusion: The malnourished children in this study exhibited impairment in the functions of cardiac contraction including mainly systolic functions and in cardiac conduction system. Cardiac morbidity and mortality can be prevented by early detection and treatment of malnutrition in these patients.

Keywords: Cardiac function, children, malnutrition

Öz

Amaç: Bu çalışmanın temel amacı malnütrisyonlu çocuklardaki miyokardiyal değişiklikleri ortaya koymaktır.

Gereç ve Yöntemler: Bu prospektif çalışma 47 malnütrisyonlu hasta ve 44 sağlıklı kontrol hastasını içermektedir. Malnütrisyonlu olgular Gomez ve Waterlov sınıflamasına göre gruplara ayrıldı. Tüm olgulara elektrokardiyografik ve ekokardiyografik inceleme, 24 saatlik Holter monitörizasyonu ve biyokimyasal değerlendirme yapıldı.

Bulgular: Malnütrisyonlu grubun 20'si (%42,5), kontrol grubunun ise 19'u (%43,1) erkek idi (p<0,05). Yaş ortalaması bakımından malnütrisyonlu grup ve kontrol grubu arasında fark yoktu (sırasıyla, 69,4±57,3 ay, 68,9±48,2 ay p=0,5). Hasta grupta sol ventrikül kitle ölçümü kontrol grubuna göre düşükken, sol ventrikül kitle indeksi farklı bulunmadı (sırasıyla, 42,3±24,5gr, 53,4±23,9 gr, p=0,049 ve 60,7±13,3 gr/m^2, 61,9±12,1 gr/m^2, p=0,67). Sol ventrikül ejeksiyon fraksiyonu ve kısalma fraksiyonu oranı hasta grubunda kontrol gruba göre düşüktü (sırasıyla, %66,2±5,3, %69,2±4,07, p=0,04 ve %35,4±4,2, %37,9±3,4, p=0,03). Miyokardiyal performans indeksi malnütrisyonlu hastalarda yüksekti (sırasıyla, 0,45±0,09, 0,36±0,05, p=0,001). Kardiyak fonksiyonlardaki bozulmalar malnütrisyonun şiddeti ve süresiyle ilişkiliydi. Troponin düzeyleri hiçbir hastada yüksek değildi. Düzeltilmiş QT dispersiyonu malnütrisyonlu hastalarda anlamlı olarak yüksekti (sırasıyla, 47,9±16,8, 32,9±10,6, p=0,001). Hiçbir hastamızda kompleks ventriküler aritmi bulunmadı.

Çıkarımlar: Çalışmamızda malnütrisyonlu çocuklarda sistolik fonksiyonlar başta olmak üzere kalbin kasılma fonksiyonlarında ve ileti sisteminde bozulma olduğu görüldü. Bu hastalarda malnütrisyonun erken tanı ve tedavisiyle kardiyak nedenli oluşabilecek hastalık ve ölüm önlenebilir.

Anahtar sözcükler: Çocuk, kardiyak fonksiyonlar, malnütrisyon

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Introduction

Malnutrition is a clinical condition that occurs when inadequate or unbalanced intake of one or multiple nutrients, which are essential for growth and development, is present such that the body's balance is disrupted, and this condition needs to be monitored (1–3). This condition mostly affects children and contributes to mortality in a significant portion of pediatric deaths. The etiology is multifactorial (economic, psychosocial, cultural, geographic) and is one of the most important health problems worldwide, including mostly developing countries (1, 3–6).

In malnutrition, anatomic and functional changes develop in the cardiovascular system as in all systems (2, 7–9). The effect of the degree and type of malnutrition on cardiac functions has not been investigated adequately (8, 9). Cardiovascular disorders including hypotension, cardiac arrhythmia, cardiomyopathy, heart failure, and in some cases, sudden death, have been reported in malnutrition. However, it has not been elucidated if these disorders are primary disorders related to malnutrition or if they are related to accompanying sepsis, dehydratation or severe anemia. Although most investigators agree that atrophy in the heart is present in patients with severe malnutriton, the question as to whether left ventricular functions are preserved in atrophic heart is controversial (6, 8, 9).

In malnutrition, impairment in cardiac electrical activity occurs in addition to myocardial tissue loss and morphologic changes at a cellular level (10–12). Studies have reported that the cardiac repolarization time prolongs and more importantly, repolarization is irregular in children with malnutrition (10, 11, 13, 14). It has been reported that prolongation in the ventricular repolarization time and/or heterogeneous repolarizaton increase the risk of ventricular arrhythmia and may lead to sudden cardiac death (15, 16). Some studies reported that this prolongation in the repolarization time was corrected with treatment (17).

In this study, it was aimed to investigate cardiac structural and functional changes that develop in relation to malnutriton and influence mortality and morbidity in children with malnutrition.

Material and Methods

The study was conducted with two groups including a patient and control group. Patients who presented to the outpatient clinics in Department of Pediatrics aged between 1 month and 18 years and were investigated and treated as outpatients or inpatients were included in this study.

Ethics committee aprroval was obtained from Fırat University Faculty of Medicine Local Ethics Committee for this study (02.06.2011/0909). The study was conducted in accordance with the principles of the Decleration of Helsinki. The study received financial support from Fırat University Scientific Research Project Support Fund. Written consent was obtained from the parents of the patients who participated in the study.

The children whose weights for age were below 90% according to the percentile values published by Olcay Neyzi in 2008 in a study conducted with Turkish children, were included in the patient group (18). This group was divided into subgroups as mild, moderate, and severe malnutrition according to the Gomez classification and as acute malnutrition, chronic malnutrition and acute malnutrition with chronic background according to the Waterlow classification (19, 20).

Age- and sex-matched healthy children who presented to the Pediatric Cardiology Outpatient Clinic because of cardiac murmur, had no other problems on physical examination, were diagnosed as having innocent murmur with normal complete blood count, biochemical tests, echocardiographic and electrocardiographic findings were included in the control group.

Children who were preterm (<35 gestational weeks, for <2 years) and who had intrauterine growth retardation (for <2 years), severe anemia (hemoglobin <6 g/dL), chronic disease, and primary cardiac pathology were excluded from the study.

Detailed history was taken and a complete physical examination was performed in all patients who constituted the study group. Body weight, height and blood pressure (measured in the right upper extremity) values were recorded. Presence of reduction in the subcutaneous adipose tissue and edema was recorded. In all subjects in the study and control groups, complete blood count, serum sodium, potassium, calcium, magnesium, urea, creatinine, glucose, total protein, albumin, aspartate transaminase (AST), alanine transaminase (ALT), total creatinine kinase (CK), creatinine kinase myocardial band (CK-MB) and troponin I concentrations were measured.

A 12-lead electrocardiogram (ECG) was performed in all subjects in the study group. The QT interval was measured in at least 9 derivations on electrocardiogram. The QTc value was calculated by correcting the QT interval by heart rate using the Bazett formula (QTc= QT value measured/√R-R). The QT dispersion (QDT) value was found by calculating the difference between the minimum and

maximum QT values and the QTc dispersion (QTcD) value was found by calculating the difference between the minimum and maximum QTc values in all patients. All measurements were performed manually.

Twenty-four-hour ECG recording was obtained in 40 subjects in the patient group and in 35 subjects in the control group using a DMS 300–7 three-channel Holter recording device (DMS, Nevada, USA). The recordings were analyzed using a DMS Cardioscan 10 (model 21) Holter analyzer system (DMS, Nevada, USA).

Echocardiographic images were obtained with the patient in the supine position or in the 45-degree left lateral decubitus position using GE Vivid 7 ultrasound system (Version Pro 1.4.0) and 3S and 7S probes. Echocardiographic examinations were performed in accordance with the standard imaging techniques recommeded by the American Society of Echocardiyoraphy (ASE). Ventricular systolic and diastolic functions were calculated by using the mean values of at least 3 measurements in the positions recommeded by the ASE.

The intraventricular septum diastolic diameter (IVSDd) and intraventricular septum systolic diameter (IVSDs), left ventricular end-diastolic internal diameter (LVIDd), left ventricular end-systolic internal diameter (LVIDs), left ventricular posterior wall thickness in end-diastole (LVPWd), and left ventricular posterior wall thickness in end-systole (LVPWs) were calculated using an M-mode echocardiogram. Left ventricular mass (LMV), left ventricular end-diastolic volume (EDV), left ventricular end-systolic volume (ESV), ejection fraction (EF), fractional shortening (FS), and stroke volume (SV) were calculated using the Teichholz formula. In addition, cardiac output (CO) was calculated by multiplying SV with heart rate per minute. The values found were divided into the body surface area value and IVSDdI, IVSDsI, LVIDdI, LVIDsI, LVPWdI, LVPWsI, LVMI, EDVI, ESVI, CI and SI were calculated.

When assessing left ventricular diastolic functions, left ventricular entrance flows were recorded in the mitral valve ends using pulse wave (PW) Doppler in the apical four-chamber position. Among the recordings obtained, the average of at least 3 measurements was found and early diastolic flow velocity (E), late diastolic flow velocity (A), E acceleration time (EAT), and E deceleration time (DT) were measured and the E/A ratio was calculated. The left ventricular output flow and enterance flow were recorded together in the appropriate positions and the interval between closure of the aortic valve and opening of the mitral valve (IVRT), the interval between closure of the mitral valve and opening of the aortic valve (IVCT) and the systole time between opening and closure of the aortic valve (LVET) were measured. The myocardial performance index (MPI) was calculated by dividing the sum of IVRT and IVCT to LVET.

Statistical Analysis

The SPSS version 12.0 computer package program for Windows was used for statistical analysis. Continuous variables are expressed as mean ± standard deviation (SD). The Chi-square test was used for the evaluation of categorical variables. The Mann-Whitney U test was used for analyses between groups that were not compatible with normal distribution. The median values were considered. Student's t-test (for independent samples) was used for measurements that showed normal distribution. A p value of <0.05 was considered significant.

Results

Forty-seven patients with malnutrition and 44 control patients were included in the study. According to the Gomez classification, seven (14.8%) patients had severe malnutrition, 20 (42.5%) had moderate malnutrition, and 20 (42.5%) had mild malnutrition. According to the Waterlow classification, 15 (31.9%) patients had chronic malnutrition, 21 (44.6%) had acute malnutrition, and 8 (17%) had acute malnutrition with chronic background. There was no difference between the malnoureshed patients and the control patients in terms of age and heart rate. Among the malnutrition groups, the heart rate was found to be higher only in the severe malnutrition group compared with the control group ($p<0.05$). Hemoglobin, potassium, and calcium concenrations, among biochemical measurements, were found to be significantly lower compared with the control group. The troponin I concentration was not found to be high in any patient. Thirty (38.2%) of the children with malnutrition and one (2.2%) child in the control group had illiterate mothers. The difference was statistically significant ($p<0.001$). The general characteristics of the patients are shown in Table 1.

Electrocargiogram revealed that the QTD and QTcD times were prolonged in the malnutrition group compared with the control group ($p<0.01$). An intergroup examination of the subjects' ECG data is shown in Table 2.

Twenty-four-hour Holter monitoring was performed in 40 of 47 patients in the patient group and in 35 of 44 patients in the control group. In the malnutrition group, frequent supraventricular beats were observed in three patients, rare supraventricular beats were found in seven patients, first-degree AV block was observed in one patient, and unifocal ventricular preterm beats were ob-

Table 1. General characteristics of the patients with malnutrition

	Malnutrisyonlu grup (n=47)	Kontrol grubu (n=44)	p
Sex (M/F)	20/27	19/25	0.95[c]
Age, mean±SD/ median (minimum–maximum), months	69.4±57.3/ 65 (3–204)	68.9±48.2/ 60 (3–180)	0.50[a]
Weight, mean±SD/ median (minimum–maximum), kg	17.2±11.2/ 15 (3.3–60)	23.2±13.9/ 19 (5.8–57.5)	**0.02[a]**
Body surface area, mean±SD/ median (minimum–maximum), m^2	0.67±0.32/ 0.63 (0.21–1.6)	0.84±0.35/ 0.76 (0.31–1.6)	**0.02[a]**
Height, mean±SD/ median (minimum–maximum), cm	102.6±30.4/ 103.5 (56–168)	110.5±28.4/ 109 (61–165)	0.2[a]
Heart rate/minutes, mean±SD/ median (minimum–maximum)	107±26.8/ 104 (68–180)	100.6±18.4/ 100 (55–140)	0.19[a]
Systolic blood presusre, mean±SD/ median (minimum–maximum), mm Hg	90 (70–115)	97.5 (80–130)	**0.001[b]**
Diastolic blood pressure, mean±SD/ median (minimum–maximum), mm Hg	55 (21–80)	60 (40–95)	**0.01[b]**
Hemoglobin, mean±SD/ median (minimum–maximum), g/dL	12.1±1.8/ 12.6 (6.6–16.7)	12.7±1.08/ 12.9 (10.4–14.9)	**0.039[a]**
Potassium, mean±SD/ median (minimum–maximum), meq/L	4.2±0.4/ 4.2 (2.8–5.2)	4.4±0.2/ 44 (3.8–5.1)	**0.028[a]**
Calcium, mean±SD/ median (minimum–maximum), mg/dL	9.7±0.6/ 9.7 (8.3–10.8)	10±0.5/ 10 (9–11.2)	**0.022[a]**
Illiterate mother, n	30 (63.8%)	1 (2.2%)	**0.001[c]**

M: Male; F: Female; SD: Standard deviation; [a]Student's t-test; [b]Mann-Whitney U test; [c]Chi-square test

Table 2. Inter-group examinations of the subjects' echocardiographic data

Parameter	Malnutrition group (n=47)	Control group (n=44)	p
QT, mean±SD Median (minimum–maximum), s	0.31±0.032 0.32 (0.24–0.4)	0.31±0.02 0.32 (0.26–0.38)	0.8[b]
QTc, mean±SD Median (minimum–maximum), sn	0.42±0.02 0.42 (0.35–0.47)	0.41±0.02 0.42 (0.37–0.45)	0.4[a]
QTD, mean±SD Median (minimum–maximum), msn	28.2±13.6 20 (9–60)	21.5±9.2 20 (5–40)	**0.008[b]**
QTcD, mean±SD Median (minimum–maximum), msn	47.9±16.8 44 (23–101)	32.9±10.6 32 (13–68)	**0.001[a]**

QTc: Corrected QT; QTcD: Corrected QT dispersion; QTD: QT dispersion; SD: Standard deviation; [a]Student's t-test; [b]Mann-Whitney U test

served in one patient. In the control group, abnormal Holter monitoring findings were not observed except for rare supraventricular beats in six patients. Complex ventricular arrhythmia was not observed in any patients.

Left ventricular EF and FS were found to be significantly lower in the malnutrition group compared with the control group ($p<0.05$). Intergroup examinations of the subjects' M-mode values are shown in Table 3. LV systolic functions were additionaly examined in the acute and chronic malnutrition groups in order to investigate the effect of the duration of malnutrition on left ventricular systolic functions. In this examination, the EF and FS values were found to be significantly lower in the chronic malnutrition groups compared with the acute malnutrition group. Change in cardiac functions according to the time and severity of malnutrition is shown in Table 4.

In the PW Doppler examination, the IVRT, IVCT, and MPI values were found to be significantly high in the patient group ($p<0.05$). An intergroup examination of the subjects' PW Doppler measurements is shown in Table 5.

Table 3. Inter-group examinations of the subjects' M-mode values

	Malnutrition group (n=47)	Control group (n=44)	p
IVSsI, mean±SD Median (minimum–maximum), (mm/m^2)	15.05±5.8 13.5 (6.7–36.1)	13.2±4.07 12.6 (7.3–24.5)	0.1[a]
IVSdI, mean±SD Median (minimum–maximum), (mm/m^2)	10.5±4.4 9.4 (5.3–23.3)	9.03±3.3 8.2 (4.7–18)	0.06[a]
LVIDsI, mean±SD Median (minimum–maximum), (mm/m^2)	33.9±11 31.7 (15.8–67)	27.2±8.6 26.5 (14.6–49.9)	**0.002**[a]
LVIDdI, mean±SD Median (minimum–maximum), (mm/m^2)	52.6±16.5 50.2 (25.8–100)	43.6±12.4 42.2 (25.4–73.9)	**0.005**[a]
LVPWsI, mean±SD Median (minimum–maximum), (mm/m^2)	14.3±4.9 12.4 (7.5–27)	12.5±3.8 11.3 (6.9–22.2)	0.054[a]
LVPWdI, mean±SD Median (minimum–maximum), (mm/m^2)	9.2±4.06 8.1 (4.9–23.3)	7.7±2.9 7.1 (4.2–15.9)	0.051[a]
LVM, mean±SD Median (minimum–maximum), (g)	42.3±24.5 38 (8.5–122)	53.4±23.9 47.8 (18.3–149)	0.049[a]
LVMI, mean±SD Median (minimum–maximum), (g/m^2)	60.7±13.3 60 (31–97)	61.9±12.1 61 (43–99)	0.67[a]
CI, mean±SD Median (minimum–maximum), (L/min/m^2)	4.2±1.3 4 (2–7.8)	3.8±1.01 3.9 (1.9–6.2)	0.16[a]
SI, mean±SD Median (minimum–maximum), (mL/m^2)	39.6±10.5 40 (6.5–57)	38.3±7.4 37 (21–60)	0.51[a]
EDVI, mean±SD Median (minimum–maximum), (mL/m^2)	59±16.4 61 (5–93)	55.5±11 53 (33–96)	0.24[a]
ESVI, mean±SD Median (minimum–maximum), (mL/m^2)	20.2±5.8 20 (8–35)	17.1±4.6 16.3 (9.1–35.9)	**0.007**[a]
EF, mean±SD (%)	66.2±5.3	69.2±4.07	**0.04**[a]
FS, mean±SD, (%)	35.4±4.2	37.9±3.4	**0.03**[a]

CI: Cardiac index; EF: Ejection fraction; EDVI: Left ventricular end-diastolic volume index; ESVI: Left ventricular end-systolic volüme index; FS: Fractional shortening; IVSDdI: Interventricular septum thickness in diastole index; IVSDsI: Interventricular septum thickness in systole index; LVIDd: Left ventricular end-diastolic diameter index; LVIDsI: Left ventricular end-systolic diameter index; LVMI: Left ventricular mass index; LVPWdI: End-diastolic left ventricular posterior wall thickness index; LVPWsI: End-systolic left ventricular posterior wall thickness index; SD: Standard deviation; SI: Stroke index; [a]Student's t-test

Table 4. Change in cardiac functions according to time and severity of malnutrition

	Acute malnutrition (n=21)	Chronic malnutrition (n=23)	p	Mild malnutrition (n=20)	Moderate–severe malnutrition (n=27)	p
Age, median (minimum–maximum), months	27 (3–204)	70 (4–186)	0.4[b]	64.5 (7.5–204)	70 (3–204)	0.9[b]
Heart rate/minutes Median (minimum–maximum)	102 (68–180)	104 (75–176)	0.8[b]	103 (68–180)	104 (75–176)	0.57[b]
EF, median (minimum–maximum), %	68 (57.3–81.2)	63.9 (58.2–75.3)	**0.007**[b]	66 (60.7–75.3)	64 (57.3–81.2)	0.08[b]
FS, median (minimum–maximum), %	37 (27.7–47.2)	33.3 (28.5–43)	**0.005**[b]	35.8 (31.3–43)	34.2 (27.7–47.2)	0.07[b]
IVRT, median (minimum–maximum) msn	59 (33–73)	57 (38–81)	0.9[b]	57 (33–81)	59 (33–81)	0.71[b]
MPI, median (minimum–maximum)	0.43 (0.3–0.7)	0.43 (0.27–0.67)	0.4[b]	0.4 (0.3–0.64)	0.45 (0.27–0.7)	0.06[b]

EF: Ejection fraction; FS: Fractional shortening; IVRT: Isovolumetric relaxation time; MPI: Myocardial performance index; [b]Mann-Whitney U test

Table 5. Intergroup examinations of the subjects' PW Doppler measurements

Parameter	Malnutrition group (n=47)	Control group (n=44)	p
E, mean±SD Median (minimum–maximum), (m/s)	1.08±0.17 1.1 (0.62–1.44)	1.02±0.17 1 (0.6–1.56)	0.15[a]
A, mean±SD Median (minimum–maximum), (m/s)	0.71±0.13 0.67 (0.49–1.1)	0.69±0.13 0.68 (0.44–1.02)	0.47[a]
E/A, mean±SD Median (minimum–maximum)	1.5±0.3 1.5 (0.93–2.36)	1.5±0.2 1.52 (1–2)	0.5[a]
EAT, mean±SD Median (minimum–maximum), (ms)	71.6±18.6 66 (36–133)	71.1±15.1 73 (36–103)	0.88[a]
DT, mean±SD Median (minimum–maximum), (ms)	145.3±35.2 140 (14–210)	152.2±31.1 152 (86–228)	0.32[a]
IVRT, mean±SD Median (minimum–maximum), (ms)	57.8±12.3 59 (33–81)	49±10.7 50 (29–66)	**0.002**[b]
IVCT, mean±SD Median (minimum–maximum), (ms)	50.9±15.5 51 (22–110)	43.9±10.5 44 (29–66)	**0.02**[b]
LVET, mean±SD Median (minimum–maximum), (ms)	242.6±43.4 258 (136–310)	261.6±37.3 269 (166–343)	**0.028**[a]
MPI, mean±SD Median (minimum–maximum)	0.45±0.09 0.43 (0.27–0.7)	0.36±0.05 0.35 (0.25–0.5)	**0.001**[a]

DT: E decceleration time; E: E flow velocity; A: A flow velocity; EAT: E acceleraton time; IVCT: Isovolumetric contraction time; IVRT: Isovolumetric relaxation time; LVET: Left ventricular ejection time; MPI: Myocardial performance index; SD: Standard deviation; [a]Student's t-test; [b]Mann-Whitney U test

Discussion

In malnutrition, anatomic and functional changes occur in the cardiovascular system as in all systems (2, 7–9). In this study, the cardiac effects of malnutrition were evluated echocardiographically and electrocardiographically.

The parents' and especially the mother's level of education has an important role in child nutrition (5). In our study, the mothers' education levels were found to be markedly lower in the malnourished patients compared with the control group. This finding suggests that malnutrition is a social problem as well as being a medical problem.

One of the findings that has been investigated in patients with malnutrition and caused excitement is impairments in the heart's electrical activity (10, 12, 14, 17, 21). Many studes have reported that prolonged and/or heterogeneous ventricular repolarization time increases the risk for ventricular arrhythmia (10–13, 15, 22). It is thought that the areas of ventricular myocardium that show slow conduction cause an increase in QTD, and ventricular tachycardia develop from these areas by way of a 'reentrant' mechanism (12, 13, 15, 22). In studies conducted with adolescents with anorexia nervosa and children with malnutrition, it was reported that the cardiac repolarization time was prolonged and more importantly, repolarization was irregular (10, 11, 13, 23). In patients with anorexia nervosa who died suddenly, it was reported that ECGs performed shortly before the patient died revealed prolongation in the QT interval and ventricular tachycardia (24–26). Occurence of ventricular arrhythmia and sudden death in obese patients who lose weight rapidly suggests that there might be a tendency to ventricular arrhythmias in malnutrition (27–29).

In our study, the QTD and QTcD values were found to be markedly higher in the malnutrition group compared with the control group. This finding was compatible with the literature data (11–14). Some investigators could not demonstrate a correlation between the degree of malnutrition and electrocardiographic measurements (12, 23). However, these studies included a low number of subjects or most subjects had mild malnutrition. On the other hand, a study conducted by Swenne et al. (11) with patients who had anorexia nervosa showed that the reduction rate and velocity in body weight influenced the QTc and QTcD values.

Although QTcD was studied frequently in patients with malnutrition and it was proposed that an increase in QTcD caused ventricular arrhythmias and sudden deaths, Holter monitoring was not performed to strengthen this thesis proposed in these groups as far as we know. In our

study, unifocal premature ventricular beats were observed in only one patient in the patient group in whom 24-hour Holter monitoring was performed, and complex ventricular arrhythmia was not observed in any patients. The fact that we did not observe ventricular arrhythmia may be related to the low number of subjects in our study or other factors influencing occurence of arrhythmias.

Although most investigators agree that there is a reduction in cardiac mass in patients with malnutrition, the question as to whether left ventricular functions are preserved is controversial (6, 8, 9, 30–32). The initial findings related to change of cardiac mass in patients with malnutrition are based on postmortem studies. In an autopsy study performed by Kerpel-Fronius and Varga (31) in 1949, it was shown that a 60% reduction occured in cardiac weight in patients with malnutrition. In another autopsy study, the cardiac weight to body weight ratio was found to be higher in patients with malnutrition compared with the control group, though a reduction in LVM occured (33). Many studies also echocardiographically showed that cardiac weight was reduced (6, 8, 9, 32, 34). In our study, LVM was found to be lower in the malnutrition group compared with the conrol group, whereas LVMI showed bo difference between the two groups in accordance with many other studies (8, 9). In the analysis performed between the malnutrition subgroups, there was no difference in terms of LVMI values. These results showed that the heart was affected in children with malnutrition like the other organs, but reduction in cardiac mass was proportional to the reduction in body mass. In the malnutrition group, ESVI and LVIDsI were found to be higher compared with the control group. This finding may be related to the reduction in left ventricular findings in patients with malnutrition.

There is no consensus in the issue of the effect of malnutrition on left ventricular systolic functions (6, 8). In the literature, EF and FS values, which are the most commonly studied values to measure left ventricular functions, were found to be unchanged in some studies (6, 32, 34), whereas other investigators found that these measurements were reduced to an important extent, especially in severe malnutrition (8, 9, 14, 35, 36). In our study, the EF and FS values were found to be lower in the malnutrition group compared with the control group. These values were found to be lower in patients with chronic malnutrition compared with those with acute malnutrition. This finding suggests that the heart cannot preserve its systolic functions despite reduced basal metabolism and decreased requirement in prolonged malnutrition. Therefore, we think that malnutrition should be treated before it becomes chronic.

In many studies, it was observed that CO reduced in parallel to the severity of malnutrition in patients with malnutrition, but CI did not change despite this reduction (6, 9). In our study, no differences were found between the malnutrition group and the control group in terms of EDVI, CI, and SI values. The unchanged CI in the patient and control groups shows that CO reduces proportionally with body mass and basal metabolism, and cardiac functional reserve is adequate for reduced circulatory load. However, some studies reported that the CI is low in patients with malnutrition, and becomes normal in the first week of treatment (35).

Studies have shown that cardiac diastolic functions are generally preserved in patients with malnutrition (9, 32). However, some studies reported that cardiac diastolic functions were also influenced, especially in cases of severe malnutrition (37). In a sudy conducted by Fieretto et al. (38) with young rats, it was advocated that changed ventricular geometry prevented impairment in diastolic functions, though passive stiffness was found in the ventricle in malnutrition. Schocken et al. (37) reported that diastolic functions could not be preserved in cases of severe wieght loss. In our study, the E, A, E/A, EAT, DT values, which are echocardiographic indicators of diastolic dysfunction, were not found to be different. In our study, the IVRT value was found to be significantly higher in patients with malnutrition compared with the control group. Prolongation in IVRT may be a sign of diastolic dysfunction related to reduced LV relaxation and decceleration of reduction of the LV pressure.

Myocardial performance index was discovered as a Doppler index that could evaluate left ventricular systolic and diastolic functions together (39). In our study, the MPI values were found to be higher in all malnutrition groups compared with the control group independent of the severity and acute or chronic characteristics of malnutrition, and no significant difference was found between the groups. We think that the MPI values, which were found to be high in these patients, mostly reflected systolic dysfunction.

Although there are studies that found increased troponin concentrations in patients with malnutrition in the literature, additional factors including sepsis, severe infection, severe anemia, and severe electrolyte imbalance are present in these patients, concurrently with malnutrition (8, 32). In our study, troponin concentrations were found to be within the normal limits in all patients. Normal troponin concentrations show that myonecrosis has not developed in the absence of aggravating additional factors. Absence of myonecrosis suggests that cardiac changes may improve following apropriate nutritional treatment.

There are limitations in the study. The patients could not be divided into age groups because the number of the patients was low, but the data were presented by indexing them to body surface area in order to minimize the effect of age. The study was a cross-sectional study and did not involve long-term follow-up.

In conclusion, it was observed that cardiac contraction functions including mainly systolic functions were impaired in children with malnutrition. In these children, increased QTD and QTcD values showed that there was a tendency to ventricular arrhythmias, and sudden deaths could occur. We think that cardiac morbidity and mortality can be prevented in these patients with early diagnosis and treatment of malnutrition.

Ethics Committee Approval: The study was conducted in accordance with the principles of the Declaration of Helsinki. Approval was obtained from Fırat University Faculty of Medicine local ethics committee (02.06.2011/0909).

Informed Consent: Written consent was obtained from the parents of all patients.

Peer-review: Externally peer-reviewed.

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Hasta Onamı: Yazılı hasta onamı bu çalışmaya katılan hastaların ebeveynlerinden alınmıştır.

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