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ORIGINAL ARTICLE

Myocardial dysfunction in malnourished Children

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Abstract:

Background: Malnourished children suffer several alterations in body composition that could produce cardiac abnormalities. Measurement of cardiac troponin T (cTnT) in blood is considered one of the gold standers for detecting heart damage and not only used as an indicator of myocardial damage but also for prognostic information. The aim of this study was to detect the frequency of myocardial damage as shown by echocardiography and cTnT level in malnourished children.

Patients and methods: The current study included 45 malnourished infants and young children (25 male and 20 female) with a mean age 11.24 ± 7.88 months, as well as 25 apparently healthy age and sex matched children (11 male and 13 female) with a mean age 10.78± 6.29 months as a control group. Complete blood picture, serum albumin, liver and kidney function tests, serum sodium, potassium and calcium level, cardiac troponin T and echocardiographic evaluation were done for cases and controls.

Results: The results of the present study revealed a significantly lower left ventricular (LV) mass in patients than the control group, and the left ventricular mass index (LVMI) was correlated positively with the body mass index (BMI). In patients with third degree marasmus (M III), kwashiorkor (KWO) marasmic-kwashiorkor (M-KWO) the LV systolic functions were significantly impaired in comparison to controls. cTnT levels were higher than the upper reference limits in 11 (24.44 %) of the studied children with PEM, all of them had severe degree of malnutrition, 6 (54.5%) of them had marasmus third degree, 2 (18%) of them had kwashiorkor (KWO) and 3 (27.7%) had marasmic-kwashiorkor (M-KWO). cTnT level was significantly higher in patients with anemia, sepsis, electrolyte deficiency and it correlated negatively to LVEF. Six (54.5%) of studied children with high cTnT levels died within 21 days of treatment compared to one (2.9%) of those with baseline levels.

Conclusion: LV mass are reduced in malnourished children in proportion to the decrease in body size. Children with M III, KWO and M-KWO not only have cardiac muscle wasting, but also have a significant decrease in LV systolic functions. Elevated cardiac troponin level in malnourished children has both diagnostic and prognostic significance for cardiomyocyte damage.

Key words: Protein Energy Malnutrition, echocardiography, cTnT

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Introduction

Protein–energy malnutrition (PEM) can be defined as the state of decreased body pools of protein with or without fat depletion that is caused at least in part by inadequate nutrient intake relative to nutrient demand that is needed to ensure growth and maintenance [1,2]. Protein energy malnutrition affects approximately one third of children worldwide and is frequently located in less-developed countries due to inadequate food intake, socioeconomic factors or, at times, due to natural disasters [3,4]. Thus, PEM is the concern of many researches[5]. Malnourished

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children suffer several alterations in body composition, with loss of heart and skeletal muscle mass, complicated by electrolyte disorders and mineral or vitamin deficiencies that could produce cardiac abnormalities including hypotension, cardiac arrhythmias, cardiomyopathy, cardiac failure, and even sudden death [3,6]. Cardiac troponins are regulatory proteins of the thin actin filaments of the cardiac muscle [7]. Myocardial cell injury results in the release of cardiac troponin, which differs from troponin isoforms of the skeletal muscle, and thus are highly sensitive and specific biomarkers of myocardial damage [8]. It is the best known molecular marker of myocardial injury [9]. Mortality among troponin-positive patients was reported to be higher compared to that among troponin-negative patients, irrespective of the cause of troponin positivity[10]. A number of studies have suggested a possible association between cardiac troponin and myocardial injury in patients with non cardiac diseases[11-14]. Several authors reported a relationship between elevated cardiac troponin levels and left ventricular dysfunction assessed by echocardiography [15-17].

The aim of this study was to detect the frequency of myocardial damage among malnourished children as shown by echocardiography and cTnT level.

Patients and Methods:

The current study included 45 infants and young children diagnosed as having PEM according to Waterlow classification (1972) [18]. They were recruited from the inpatients Departments of Assiut University Children Hospital, Egypt. The patients were 25 males and 20 females with an age range 2 to 30 months. 3 children had first degree marasmus (M I), 16 had second degree marasmus (M II), 19 had third degree marasmus (M III), 4 had kwashiorkor (KWO) and 3 had marasmic-kwashiorkor (M-KWO). They were compared to 25 apparently healthy age and sex matched children (11male and 14 female) with an age range 4 to 24 months, as a control group. Patients were excluded if they were preterm infants or they showed intrauterine growth retardation at birth, if they had any documented cardiothoracic event (congenital heart disease, pericarditis, cardiomyopathy, acute severe lower respiratory tract infection, etc) and severe anemia (blood hemoglobin level $\leq$ 6 g/dl). After obtaining the approval of ethical committee of Faculty of Medicine, Assuit University, an informed written consent was taken from the parents or legal guardians. Before nutritional rehabilitation, all studied malnourished infants were subjected to full history taking including dietetic history, thorough clinical examination with special emphasis on the anthropometric measurements and signs of malnutrition. Height and weight were marked on Cairo university growth charts [19]. For all children included in this study the followings were done, complete blood picture, serum albumin, liver and kidney function tests, serum sodium, potassium and calcium levels. Nutritional rehabilitation was done to all patients according to WHO (1999) recommendation [20].

Assessment of cardiac troponin T was done by electrochemiluminescence immunoassay (ECLIM) I intended for use on the Roche Elecsys 1010 immunoassay analyzer. 0.010 ng/ml defined by the lower detection limit [21]. Values below the detection limit are reported as < 0.010 ng/ml.

Echocardiographic Evaluation:

All patients had transthoracic Echocardiographic examination using Hewlett Packard (HP) sonos 4500 using phased array transducers with frequency 8 MHZ. Imaging was done while the patient in recumbent or lateral decubitus position.

M-mode, 2-Dimensional echocardiography, pulsed and continuous wave Doppler and color flow mapping were performed for every patient using the standard views as parasternal long axis, short axis, apical four, five chamber and

Table 1. Some clinical and laboratory data of the studied patients with PEM compared to controls.

	Age (ms)	Weight (Kg)	Height (Cm)	BMI ($Wt/ht^{2)}$	Na (m eq/l)	K (m eq/l)	Ca (mg/dl)	Total Protein (g/dl)	albumin (g/dl)	Hg (g/dl)	WBCs ($\times 10^3$/ul)	cTnT (ng/ml)
Patients (n=45)	11.24 ± 7.88	5.4 ± 1.6	63.4 ± 8.93	13.33 ± 2.19	137.4 ± 5.23	3.82 ± 0.54	8.64 ± 1.25	61.43 ± 9.68	34.38 ± 8.4	9.39 ± 1.6	10.52 ± 4.23	0.01 ± 0.01
Control (n=25)	10.78 ± 6.29	8.97 ± 2.26	74.8 ± 9.54	15.81 ± 2.28	139.64 ± 3.1	3.88 ± 0.454	9.15 ± 1.0	70.61 ± 7.34	40.14 ± 11.91	12.53 ± 1.1	8.83 ± 3.6	0.009 ± 0.001
p	NS	0.000*	0.000*	0.000*	NS	NS	0.000*	0.000*	0.025*	0.000*	0.000*	0.007*

BMI = body mass index. Na = serum sodium. K = serum potassium. Ca = serum calcium. Hg = hemoglobin level. Wbcs = total leucocytic count. cTnT = cardiac topinin T. Quantitative variables are expressed as mean ± standard deviation
*= statistically significant result. NS = non significant result

subcostal views to assess the following parameters:

(1) left ventricular function:

1. Left ventricular dimensions.

Measured from the derived M-mode echocardiography in the parasternal long axis. All the tracings were recorded using the leading edge technique [22].

2. Percentage of fractional shortening (FS).

Left ventricular FS was calculated using the following formula:
FS=EDD-ESD/EDD X 100 [23].

EDD is end diastolic diameter of left ventricle.

ESD is end systolic diameter of left ventricle.

3. Ejection fraction (EF).

Measured from the "cubed equation".
EF= (EDD) - (ESD)/ (EDD) X 100

4. Left ventricular diastolic function.

Using E/A ratio of the mitral flow by pulsed wave Doppler across mitral valve.

(2) <u>left ventricular mass and mass index:</u>

It is done using left ventricular mass index calculator.

Statistical analysis:

Analysis was done using SPSS (version 16). The numerical data were represented as mean ± SD. Student's *t* test was used for parametric data. The difference was considered significant if probability (*p*) values were less than 0.05. Linear correlations were performed by Spearman's or Pearson's test.

Results:

Some clinical and laboratory data of the studied patients compared to control are listed in table 1. No statistically significant difference between patients and controls regarding the heart rate, the mean diastolic and systolic blood pressure, liver and kidney functions could be detected (data are not tabulated).

Table 2 shows echocardiographic findings of patients with PEM compared to controls.

Cardiac troponin T level and echocardiographic findings of different groups of malnutrition and controls are shown in table 3.

Table 2. Echocardiographic findings of the patients with PEM compared to controls

Echo	LVSD	EDD	ESD	PWD	LVM	LVMI	FS	EF	E/A
Patients (45)	0.40 ± 0.05	2.56 ± 0.63	1.55 ± 0.42	0.4 ± 0.08	18.96 ± 7.59	67.78 ± 23.04	0.46 ± 0.13	40.31 ± 9.12	72.6 ± 10.76
Controls (25)	0.46 ± 0.05	2.84 ± 0.52	1.72 ± 0.36	0.45 ± 0.07	29.89 ± 12.09	64.46 ± 12.59	0.57 ± 0.12	55.46 ± 22.04	75.09 ± 8.54
P value	0.000*	NS	NS	0.013*	0.000*	NS	0.001*	0.000*	NS

LVSD = left ventricular septal diameter. EDD = end diastolic diameter of left ventricle. ESD = end systolic diameter of left ventricle. PWD = posterior wall diameter. FS = fractional shortening. EF = Ejection fraction. LVM = left ventricular mass. LVMI = left ventricular mass index. Quantitative variables are expressed as mean ± standard deviation
*= statistically significant result. NS = non significant result

Table 3. Cardiac troponin T and echocardiographic data of different groups of malnutrition and controls.

	cTnT (ng/ml)	LVSD	EDD	ESD	PWD	LVM	LVMI	FS	EF	E/A
(1)= MI+ MII 19/45	0.093 ± 0.004	0.41 ± 0.02	2.8 ± 0.71	1.68 ± 0.45	0.39 ± 0.51	22.73 ± 8.86	72.39 ± 1.66	0.52 ± 0.13	45.63 ± 8.05	77.15 ± 8.85
(2)= MIII 19/45	0.138 ± 0.01	0.39 ± 0.06	2.41 ± 0.53	1.46 ± 0.37	0.39 ± 0.07	17.3 ± 4.96	69.93 ± 2.59	0.46 ± 0.13	38.73 ± 6.83	71 ± 7.44
(3)= KWA+ M-KWA 7/45	0.028 ± 0.018	0.35 ± 0.02	2.27 ± 0.55	1.4 ± 0.43	0.35 ± 0.018	13.19 ± 4.09	45.24 ± 1.93	0.45 ± 0.97	30.14 ± 7.53	64.57 ± 1.72
(4)= Controls 25	0.093 ± 0.006	0.46 ± 0.05	2.84 ± 0.52	1.72 ± 0.36	0.45 ± 0.07	29.89 ± 12.09	64.46 ± 1.25	0.57 ± 0.12	55.46 ± 2.204	75.09 ± 8.54
***P value* 1vs 2**	NS	NS	NS	NS	NS	0.026*	NS	NS	0.007*	0.026*
1vs 3	0.000*	0.000*	0.07*	NS	NS	0.012*	0.002*	NS	0.000*	0.021*
1vs4	NS	0.000*	NS	NS	0.003*	0.023*	0.041*	NS	NS	NS
2vs3	0.017*	NS	NS	NS	NS	NS	0.031*	NS	0.011*	NS
2vs4	0.006*	0.000*	0.006*	0.013*	0.01*	0.000*	NS	0.002*	0.002*	NS
3vs4	0.000*	0.000*	0.008*	0.028*	0.001*	0.001*	0.001*	0.024*	0.004*	0.013*

cTnT = cardiac topinin T. LVSD = left ventricular septal diameter. EDD = end diastolic diameter of left ventricle. ESD = end systolic diameter of left ventricle. PWD = posterior wall diameter. FS = fractional shortening. EF = Ejection fraction. LVM = left ventricular mass. LVMI = left ventricular mass index. Quantitative variables are expressed as mean ± standard deviation. *= statistically significant result. NS = non significant result.

Table 4. Some laboratory and echocardiographic parameters of the studied patients with normal cTnT level compared to those with high cTnT level.

	Na (m eq/l)	K (m eq/l)	Ca (mg/dl)	Total Protein (g/dl)	albumin (g/dl)	Hg (g/dl)	Wbcs (×10³/ul)	FS	EF	E/A	Mortality %
Patients with normal cTnT (n=34)	138.3 ± 4.95	3.9 ± 0.48	8.8 ± 1.14	63.32 ± 7.7	36.1 ± 7.7	9.85 ± 1.12	9.97 ± 4.19	48.24 ± 12.17	42.52 ± 8.27	73.88 ± 8.96	1/34 (2.94%)
Patients with high cTnT (n=11)	134.6 ± 5.32	3.5 ± 0.63	8.1 ± 1.45	55.59 ± 8.7	29.1 ± 8.6	7.96 ± 2.01	15.21 ± 4.04	50.91 ± 14.46	33.45 ± 8.45	68.63 ± 1.49	6/11 (54.54%)
p	0.043*	0.043*	NS	0.024*	0.015*	0.000*	0.001*	NS	0.003*	NS	0.000*

Na = serum sodium. K = serum potassium. Ca = serum calcium. Hg = hemoglobin level. Wbcs = total leucocytic count. FS = fractional shortening. EF = Ejection fraction. Quantitative variables are expressed as mean ± standard deviation

*= statistically significant result. NS = non significant result.

The present study revealed that left ventricular mass index (LVMI) correlated positively with the body mass index (BMI) r= 0.126, P= 0.411 (Figure 1).In the present study cTnT levels were higher than the upper reference limits in 11 (24.44 %) of the studied children with PEM, all of them had severe degree of mal nutrition, 6 (54.5%) had M III, 2 (18%) had KWO and 3 (27.7%) had M-KWO. Table 4 shows some laboratory and echocardiographic parameters of PEM children with normal cTnT level compared to those with high cTnT level.

Table 5. Shows some laboratory and echocardiographic parameters in studied patients with PEM according to the outcome.

	Na (m eq/l)	K (m eq/l)	Ca (mg/dl)	Total Protein (g/dl)	albumin (g/dl)	Hg (g/dl)	WBCs (×10³/ul)	cTnT (ng/ml)	FS	EF	E/A
Survivors (n=38)	137.97 ± 4.79	3.94 ± 0.47	8.74 ± 1.2	62.7 ± 9.6	35.55 ± 7.7	9.5 ± 1.64	10.3 ± 4.2	0.01 ± 0.00	49.2 ± 13.2	42.31 ± 8.1	74.3 ± 9.1
Non survivors (n=7)	134.4 ± 6.8	3.18 ± 0.44	8.08 ± 1.31	54.57 ± 9.7	28.01 ± 9.7	8.7 ± 1.17	11.6 ± 4.4	0.03 ± 0.01	47.1 ± 0.1	29.4 ± 6.8	63.4 ± 1.5
p	NS	0.000*	NS	0.046*	0.027*	NS	NS	0.000*	NS	0.000*	0.012*

Na = serum sodium. K = serum potassium. Ca = serum calcium. Hg = hemoglobin level. WBCs = total leucocytic count. cTnT = cardiac topinin T. FS = fractional shortening. EF = Ejection fraction. Quantitative variables are expressed as mean ± standard deviation

*= statistically significant result.

A significant negative correlation was found between cTnT level and LVEF, r = -0.213, P= 0.043 (Figure 2). cTnT level was significantly higher in patients with anemia, sepsis, electrolyte deficiency. six (54.5%) of studied children with high cTnT levels died within 21 days of treatment compared to one (2.9%) of those with baseline levels**.** Table 5 shows some laboratory and echocardiographic parameters in studied patients with PEM according to the outcome.

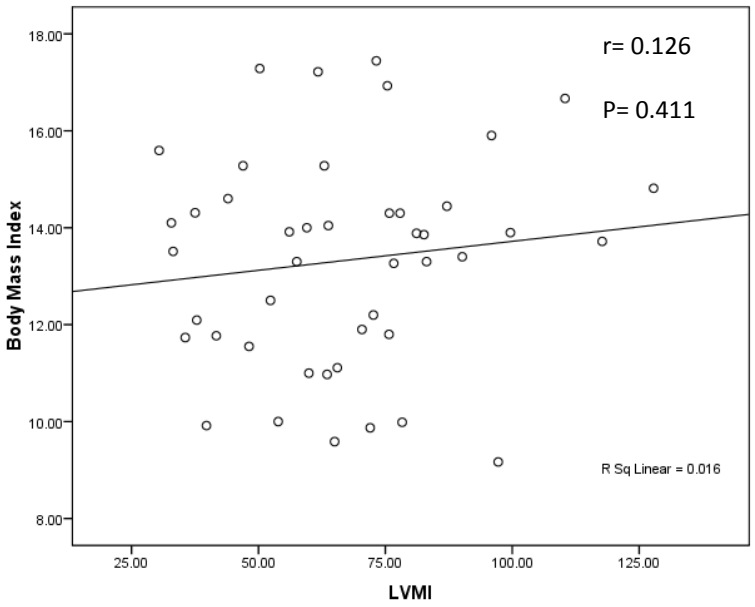

Figure 1. Correlation between BMI and LVMI (BMI = body mass index. LVMI = left ventricular mass index).

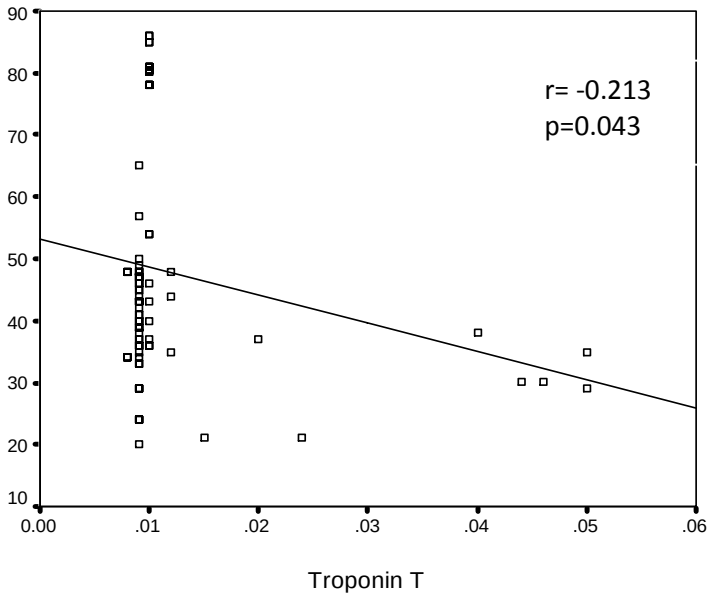

Figure 2. Correlation between cTnT and LVEF (cTnT = cardiac topinin T. EF = Ejection fraction)

Discussion:

PEM is a serious disease responsible for high morbidity and mortality rates among children in developing countries [24]. Children suffering from severe malnutrition frequently exhibit cardiovascular abnormalities [3].

Echocardiographic evaluation of children with PEM in the present study revealed a significantly lower left ventricular septal thickness, posterior wall thickness and left ventricular (LV) mass than the control group (table 2). This decrement was more prominent in patients with marasmus third degree, KWO and marasmic KWO (table 3), and the decrease of left ventricular mass index (LVMI) correlated significantly to the decrease in the body mass index (BMI) (figure 1). These findings are in agreement with previous authors who reported that in patients with PEM the heart is unable to escape from atrophy affecting other organs and this decrement is proportional with the decrement in the total body mass [3,5,6]. In 2002, Cunha et al. [25]. suggested that the cause of diminished cardiac mass in patients with PEM was slow myocardial anabolic rate rather than increased catabolism. Most investigators agree that the heart atrophies during starvation, but controversy persist as to whether atrophic heart with PEM functions normally or demonstrate left ventricular dysfunction. Also it was not clear whether there is a difference in cardiac performance as a function of type and severity of PEM [6]. The present study showed that the parameters of the LV systolic function (the ejection fraction and fraction shortening) were significantly affected in patients than the controls (table 2), and this affection is more prominent in patients with M III, KWO and M-KWO (table 3). This is in agreement with Singh et al. [26] who reported that LV systolic function was reduced in PEM children with more than 40% loss of the expected weight and Shoukry et al. [27] who reported that infants with KWO had a reduction in the fractional shortening compared with the controls. Others did not find any evidence of LV systolic dysfunction in their patients [3,5,6]. This difference possibly could be due to the effect of other factors such as electrolyte imbalance or trace element deficiency that affect LV systolic function in the different studies. Regarding the LV diastolic function (the E/A ratio), the present study showed no significant difference between patients and the control group. These findings are in keeping with that reported by other studies [3,5,6]. Myocardial damage in children may be clinically occult in a variety of stressful settings [28]. Measurement of cTnT in blood is considered one of the gold standers for detecting heart damage. Cardiac cTnT is an abundant cardiac protein which is not detected in significant levels in healthy individuals and released predominantly during cardiac damage [29]. The present study revealed that cTnT levels were higher than the upper reference limits in 11 (24.44 %) of the studied children with PEM, all of them had severe degree of malnutrition, 6 (54.5%) of them had M III, 2 (18%) had KWO and 3 (27.7%) had MKWO. cTnT level was significantly higher in patients with anemia, sepsis and electrolyte deficiency (table 4) and a significant negative correlation was found between cTnT level and LVEF (figure 2). The results of the present study are in line with that reported by Elsayed et al. [5]. who stated that in patients with PEM there is no place for cardiac proteins to be released massively in a detectable way in circulation except in acute severe cases or in the presence of complications. These findings could be explained by the presence of complications such as sepsis, electrolyte imbalance and anemia in cases of severe PEM either alone or in combination may cause myocardial injury and consequently affecting LVEF and causing a significant elevation in cTnT in the blood. Children may have more deleterious consequences of low- level cardiomyocyte loss than adults due to both the length of subsequent survival and an insufficient potential for myocardial growth to compensate for both early

damage and somatic development [28]. The increased cTnT level not only used as an indicator of myocardial damage but also for prognostic information [28,30]. In the present study 6 (54.5%) of PEM children with high cTnT levels died within 21 days of treatment compared to 1 (2.9%) of those with baseline levels (table 5). These results are in keeping with Kontos et al. [30] who reported that the mortality at 30 days was significantly higher among patients with elevated troponin levels at presentation than among patients with no biomarkers detected. These cardiac changes in patients with PEM denote the severity of the disease and its fatal outcome requiring urgent effective measures to overcome this serious disease. Nutritional rehabilitation can reverse these abnormalities to great extent [5].

In conclusion: LV mass is reduced in patients with PEM in proportion to the decrease in body size. Children with M III, KWO and MKWO not only have cardiac muscle wasting, but also have a significant decrease in LV systolic functions. Elevated cardiac troponin level in patients with PEM has both diagnostic and prognostic impact for cardiomyocyte damage.

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