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Original Research Article

Left ventricular function by speckle-tracking echocardiography in patients with low-T3 syndrome and acute myocardial infarction

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ABSTRACT

Background and objective: Low-T3 syndrome is common in patients with acute myocardial infarction (AMI). Recent experimental and clinical data have suggested a potential negative impact of low-T3 syndrome on myocardial function in patients with AMI. The aim of this study was to assess left ventricular (LV) myocardial function in patients with low-T3 syndrome and to investigate the association between hormonal profile and the severity of LV dysfunction using speckle-tracking echocardiography (STE).

Materials and methods: In 130 patients with first-onset ST-segment elevation acute myocardial infarction (STEMI), conventional 2D and speckle-tracking echocardiography within 48–72 h after the hospitalization was performed, and blood samples for TSH, fT4, fT3, and anti-TPO levels were obtained to investigate thyroid hormone production within 24 h and on the fourth day after the onset of STEMI symptoms.

Results: The patients were divided into two groups according to their serum level of fT3: group 1 with fT3 concentration below 3.2 pmol/L ($n = 34$) and group 2 with normal fT3 (>3.2 pmol/L) level ($n = 96$). LV ejection fraction (EF) tended to be lower in the low fT3 group. The systolic longitudinal strain did not differ between the groups, but the late diastolic

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longitudinal strain rate was lower in group 1 ($P = 0.011$). The systolic basal LV rotation positively correlated with the level of fT3 ($r = 0.4$; $P < 0.001$), while a negative correlation was detected between myocardial rotational parameters – systolic apical rotation ($r = -0.2$; $P < 0.05$), torsion ($r = -0.3$; $P < 0.001$), and diastolic apical rotation rate ($r = -0.3$; $P < 0.01$) – and fT3 levels.

Conclusions: The late diastolic longitudinal LV strain rate and LV rotation evaluated by speckle-tracking echocardiography were impaired in patients with low-T3 syndrome after AMI.

1. Introduction

There is a close relationship between thyroid hormone levels and cardiovascular diseases [1]. Thyroid hormones (TH) regulate cardiac function through the genes encoding important structural and functional proteins in the myocardium [2]. Low triiodothyronine (T3) level is common in patients with acute myocardial infarction (AMI) and is a strong predictor of poor short-term and long-term prognosis [3–5]. Low-T3 syndrome can be described as low serum levels of free T3 (fT3) and elevated reverse T3 (rT3) levels, while thyroid-stimulating hormone (TSH) and free thyroxine (fT4) could also be affected in variable degrees based on the severity and duration of the non-thyroidal illness [6]. Recent experimental and clinical data have suggested a potential negative impact of low-T3 syndrome on myocardial function in patients with AMI, causing a significant decrease in left ventricle (LV) ejection fraction (EF) [7,8]. A new echocardiography technique – 2D speckle-tracking echocardiography (STE) – has been introduced to evaluate myocardial mechanics; this technique provides subtle information about global and regional LV myocardial function. The aim of this study was to evaluate the relationship between low fT3 levels and subtle LV function parameters such as strain and strain rate, rotation, and twisting motion.

2. Materials and methods

2.1. Study population

The study included 130 patients (106 [82%] men and 24 [18%] women) admitted to the emergency cardiac care unit with first-onset ST-segment elevation acute myocardial infarction (STEMI); the patients were recruited between November 2012 and September 2013. The diagnosis of STEMI was made on the basis of typical ECG changes and/or chest pain that was suggestive of myocardial ischemia and associated with the elevation of cardiac biomarkers. All patients with STEMI underwent coronary angiography and primary percutaneous coronary intervention (PCI). The infarct-related artery was identified during coronary angiography.

The exclusion criteria were as follows: thyroid dysfunction (based on medical history and treatment); amiodarone treatment; prior myocardial infarction; arrhythmias; valvular heart diseases (moderate and severe aortic stenosis, moderate and severe aortic regurgitation, prior known mitral valvular disease, or rheumatic affections); 2-dimensional (2D) echocardiography images that were inadequate for analysis; and coronary artery bypass grafting (CABG) undergone within the last 6 months.

A total of 25 patients were excluded from this study because of thyroiditis (anti-TPO > 3.2 kU/L; $n = 13$), and inadequate quality of echocardiography images ($n = 12$).

Written informed consent was obtained from all patients, and the study protocol was approved by the ethics committee of the hospital.

2.2. Clinical data

The patients' clinical records were reviewed for cardiac risk factors, including cigarette smoking, hyperlipidemia, hypertension, and obesity. A patient was deemed to be a current smoker when he or she had smoked any number of cigarettes on a regular basis within 3 weeks before cardiac catheterization, or was a past smoker. Hyperlipidemia was defined as total serum cholesterol level of more than 5.0 mmol/L; low density cholesterol, more than 3.0 mmol/L, and triglyceride level, more than 1.7 mmol/L; or the use of statin medications. Hypertension was defined as systolic pressure ≥140 mm Hg and/or diastolic pressure ≥90 mm Hg, or the use of antihypertensive medication; obesity was defined as BMI ≥ 30 kg/m^2 [9].

2.3. Echocardiography

All patients with STEMI were examined using 2D echocardiography within 48–72 h after hospitalization. Both conventional echocardiography and STE were performed using the Vivid 7 ultrasound line (GE VingMed Ultrasound AS; Horten, Norway) in the left lateral decubitus position. Standard images were obtained using a 3.5-MHz transducer, in the parasternal (long- and short-axis images) and apical (2- and 4-chamber images) views. Standard 2D and Doppler data, triggered to the QRS complex, were saved in a sine-loop format. Measurements were averaged from at least three consecutive beats. The mean frame rate of the obtained images was 80–90 fps.

At baseline, 2D echocardiography was used to assess conventional parameters: LV end-systolic diameters (LVESD), LV end-diastolic diameter (LVEDD), LV end-systolic volume

(LVESV), LV end-diastolic volume (LVEDV), LV ejection fraction (EF), left atrial (LA) diameter, LA volume, and the ratio of peak early mitral inflow velocity (E) and peak early mitral annular velocity (E′), or E/E′ ratio. The LV volumes and LVEF were calculated from the conventional apical two-and four-chamber images, using the biplane Simpson's technique. The LA dimension was measured at end-systole. LA volume was measured using Simpson's method from the apical two-and four-chamber images. All morphometric parameters were indexed by body surface area.

Myocardial tissue deformation (strain) was calculated using STE. LV strain and strain rate analysis was performed using the available software (EchoPac 6.1, GE Medical Systems, Horten, Norway).

Before speckle-tracking analysis, the timings of aortic and mitral valve opening and closure were assessed using pulsed-wave Doppler recordings of aortic and transmitral flows. The interval between two subsequent closures of the mitral valve was used for regional strain and rotation analysis.

Apical 4-chamber and 2-chamber views were used for the longitudinal strain (Ls) and strain rate analysis. Parasternal short-axis views at the base (at the tips of the mitral valve leaflets), at the level of papillary muscles, and at the apex (with the minimal circular LV cavity at the end-systole) were used for radial strain (Rs), circumferential strain (Cs), and rotation (R) analysis.

Endocardial borders were manually traced in the end-systole. The myocardium was automatically divided into six segments. The apical-axis view was manually divided into four segments. The deformation and rotation values of each segment were collected from the result window. Peak systolic strain, strain rate, early and late diastolic strain rate, and rotation were used for the analysis. All global deformation indices were calculated as an average of the observed segmental values.

The LV twist was calculated as the absolute apex-to-base difference in LV rotation. LV torsion was calculated as the LV twist normalized with respect to ventricular diastolic longitudinal length between the LV apex and the mitral plane ([apical LV rotation − basal LV rotation]/LV diastolic longitudinal length).

2.4. *Thyroid hormone analysis*

Within 24 h and on the fourth day after the onset of STEMI symptoms, concentration of the thyroid hormones (TH) in serum was measured. Blood samples for TSH, fT4, fT3, and anti-TPO were collected by venipuncture in vacuum tubes for serum with gel separator (5 mL). Blood samples were stored at 18–25 °C until a clot formed (after about 15–45 min), and serums were separated by centrifugation (1200 × *g*, for 15 min), and were analyzed using the automated enzyme immunoassay analyzer AIA-2000 (Tosoh corporation, Japan). The reference intervals were as follows: 3.2–5.9 pmol/L for fT3, 9–21.07 pmol/L for fT4, 0.38–4.31 mU/L for TSH, and <3.2 kU/L for anti-TPO.

2.5. *Statistical analysis*

Continuous data are presented as mean ± standard error of mean or median and quartiles (Q1; Q3). Dichotomous variables are presented as percentages. The normality assumption of continuous variables was tested using the Kolmogorov–Smirnov test. The Mann–Whitney *U* test was used in samples less than 20 and the exact Fisher test, for differences between proportions.

The correlation between strain, strain rate, diastolic strain rate or rotation and TH (fT3, fT4, TSH) was evaluated using Spearman correlation. A two-sided *P* value of less than 0.05 was considered to indicate statistical significance. All analyses were performed using SPSS 22 software.

Interobserver variability was assessed for the measurements of systolic Ls in the septal and lateral walls; systolic Rs and Cs, at the basal and apical levels; and systolic rotation, at the basal and apical levels in 10 randomly selected subjects using the Bland–Altman method.

3. Results

3.1. *Clinical characteristics of the study population*

A total of 130 patients with STEMI were enrolled in our study (mean age, 58.7 ± 10.2 years). The study population was divided into two groups according to their serum fT3 level: group 1 with low fT3 (<3.2 pmol/L) (*n* = 34; 26%), and group 2 with normal fT3 (>3.2 pmol/L) (*n* = 96; 74%). In group 1, the fT3 value was lower. We found that in the low fT3 group, the fT3 value on day 4 was lower ($P < 0.001$) than on day 1 ($P < 0.01$), while TSH levels were higher on day 1 ($P < 0.002$). No significant differences in TSH and fT4 levels were found between the groups on day 4 (Table 1).

The comparison of fT3 levels in the study population showed that fT3 levels were lower on day 4 than on day 1 (3.64 ± 0.05 pmol/L and 3.96 ± 0.08 pmol/L, respectively; $P < 0.001$) (Fig. 1). TSH levels on day 4 were significantly higher (2.3 ± 0.25 mU/L) than on day 1 (1.13 ± 0.08 mU/L) ($P < 0.001$) (Fig. 2). FT4 values were similar on day 1 and day 4 (14.4 ± 0.29 pmol/L vs. 14.5 ± 0.26 pmol/L; $P > 0.05$) (Fig. 3).

There were a higher percentage of patients with obesity ($P < 0.03$) and hyperlipidemia ($P < 0.014$) in the group with low fT3 levels. There were no significant differences in age, sex, serum troponin I, white blood cells, hemoglobin, creatinine, CRP levels, blood pressure on admission, a history of

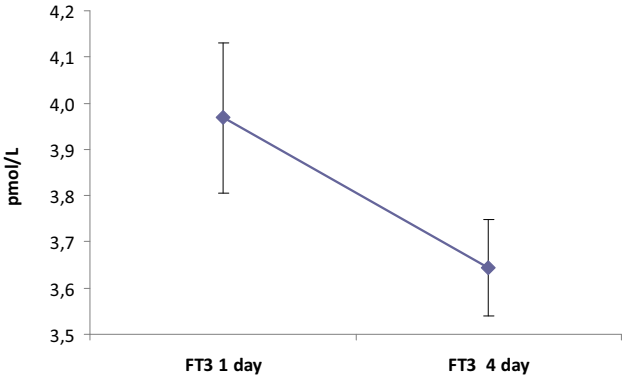

Fig. 1 – fT3 levels on days 1 and 4. Values are means and error bars represent 95% confidence intervals.

Table 1 – Characteristics of study population.

Characteristics	Group 1 (low fT3)	Group 2 (normal fT3)	P value
Gender, *n* (%)	28 (82.4)	78 (81.3)	0.85
Age (years)	59.3 ± 2.2	58.5 ± 1.2	0.09
History of hypertension, *n* (%)	21 (61.8)	58 (60.4)	0.87
History of smoking, *n* (%)	22 (64.7)	51 (53.1)	0.06
Obesity, *n* (%)	26 (76.5)	53 (55.2)	<0.03
Hyperlipidemia, *n* (%)	21 (61.8)	36 (37.5)	<0.014
Troponin I, median (Q1; Q3) (μg/L)	6.4 (0.3; 56)	12.7 (4.6; 80.4)	0.07
WBC, $\times 10^9$/L	10.4 ± 0.6	10.8 ± 0.4	0.87
Hemoglobin (g/L)	142.8 ± 1.9	141.1 ± 1.5	0.75
Creatinine (μmol/L)	84.5 ± 3.1	85.5 ± 2.3	0.98
CRP, median (Q1;Q3) (mg/L)	1.9 (1; 5.4)	3.4 (1.6; 8.4)	0.12
Systolic pressure (mm Hg)	140.9 ± 4.0	138.6 ± 2.5	0.21
Diastolic pressure (mm Hg)	85.3 ± 2.4	81.8 ± 1.2	0.09
Heart rate, beats per min	69.9 ± 2.1	74 ± 1.3	0.087
Killip class on admission, *n* (%)			
I	8 (23.5)	29 (30.5)	0.08
II	22 (64.7)	60 (63.2)	0.27
III	0 (0)	2 (2.1)	–
IV	4 (11.8)	4 (4.2)	–
Single vessel, *n* (%)	11 (32.4)	38 (39.6)	0.87
Multivessel, *n* (%)	23 (67.6)	58 (60.4)	0.12
Anterior MI location, *n* (%)	16 (47.1)	42 (44.2)	0.89
Inferior MI location, *n* (%)	18 (52.9)	53 (55.8)	0.068
Q-wave MI	26 (76.5)	66 (68.8)	0.06
fT3 on day 1 (pmol/L)	3.2 ± 0.1	4.2 ± 0.1	<0.01
fT3 on day 4 (pmol/L)	3.1 ± 0.1	3.9 ± 0.1	<0.001
fT4 on day 1 (pmol/L)	13.9 ± 0.6	14.7 ± 0.3	0.08
fT4 on day 4 (pmol/L)	13.9 ± 0.5	14.8 ± 0.3	0.09
TSH on day 1, median (Q1; Q3) (mU/L)	1.0 (0.5; 1.6)	0.9 (0.5; 1.5)	<0.002
TSH on day 4, median (Q1; Q3) (mU/L)	1.7 (1.3; 2.8)	1.7 (1.1; 2.6)	–

Values are mean ± standard error unless otherwise indicated.
fT3, free T3; fT4, free T4; TSH, thyroid-stimulating hormone; WBC, white blood cell; CRP, C-reactive protein.

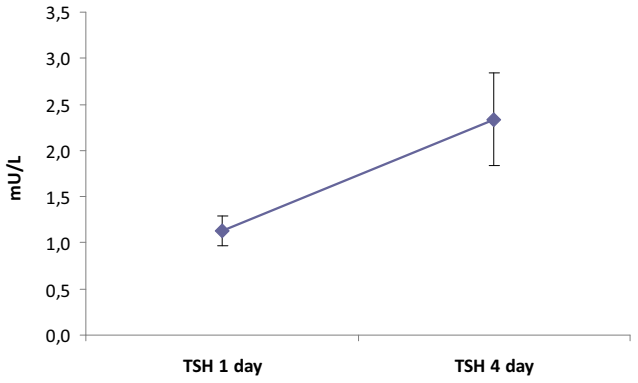

Fig. 2 – TSH levels on days 1 and 4. Values are means and error bars represent 95% confidence intervals.

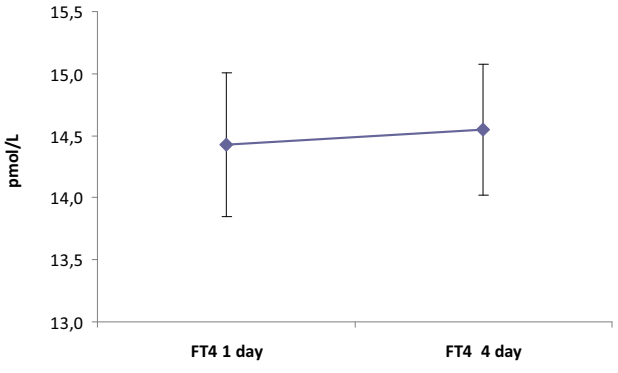

Fig. 3 – fT4 levels on days 1 and 4. Values are means and error bars represent 95% confidence intervals.

hypertension, Killip class, or the location of myocardial infarction between the two groups.

3.2. *Assessment of echocardiography data*

Left ventricular diameters, volume and volume indices were similar between the groups, except the LVESD index was higher in group 2, compared to group 1. LVEF had a tendency to be lower in group 1. The LA diameter was significantly higher in group 1 than in group 2 ($P = 0.04$). We also found a negative correlation between LA and fT3 on day 4 ($r = -0.24$; $P = 0.011$).

Doppler mitral E- and A-waves had a tendency to be lower and deceleration time (DT) to be longer in the low fT3 group. The E/E′ ratio was lower, and the diastolic mitral annular velocity of the septum was higher in group 1 than group 2

Table 2 – 2D, transvalvular, and tissue Doppler parameters.

Parameter	Group 1 (low fT3)	Group 2 (normal fT3)	P value
LVEDD (mm)	48.4 ± 0.9	48.6 ± 0.5	0.48
LVEDD index (mm/m^2)	24.0 ± 0.5	24.6 ± 0.3	0.87
LVESD (mm)	38.1 ± 0.9	39.8 ± 0.5	0.27
LVESD index (mm/m^2)	18.9 ± 0.5	20.1 ± 0.3	0.01
LVEDV (mL)	104.8 ± 2.6	106.7 ± 1.6	0.12
LVEDV index (mL/m^2)	51.7 ± 1.1	53.9 ± 0.7	0.09
LVESV (mL)	39.2 ± 1.0	42.5 ± 1.3	0.06
LVESV index (mL/m^2)	19.4 ± 0.5	21.4 ± 0.6	0.78
LVEF (%)	40.9 ± 1.5	41.5 ± 0.9	0.57
LA (mm)	40.4 ± 0.6	39.9 ± 0.4	0.04
LA volume (mL)	38.1 ± 2.0	38.2 ± 1.0	0.89
LA volume index (mL/m^2)	19.0 ± 1.1	19.2 ± 0.5	0.31
E/A ratio	0.98 ± 0.07	0.95 ± 0.1	0.06
E peak rate (m/s)	67.1 ± 2.6	70.3 ± 2.1	0.53
A peak rate (m/s)	73.6 ± 2.9	78.3 ± 1.8	0.10
E′ lateral (m/s)	10.1 ± 0.5	9.0 ± 0.3	0.87
E′ septal (m/s)	8.6 ± 0.4	7.6 ± 0.2	0.04
E/E′	7.2 ± 0.4	8.9 ± 0.4	0.006
DT (ms)	268.0 ± 19.8	237.5 ± 10.7	0.46

Values are mean ± standard error.
LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; LA, left atrium; E/A, early diastolic transmitral flow velocity (E) and atrial systolic velocity (A) ratio; E, early diastolic transmitral flow velocity; A, atrial systolic velocity; E′, early diastolic mitral annular velocity; E/E′, early diastolic transmitral flow velocity and early diastolic mitral annular velocity; DT, deceleration time.

Table 3 – Differences of left ventricular rotation and systolic and diastolic longitudinal, radial, and circumferential strain and strain rate between low fT3 and normal fT3 groups.

Parameter	Group 1 (low fT3)	Group 2 (normal fT3)	P value
Rotation			
Systolic basal R (°)	−3.2 ± 0.8	−2.6 ± 0.5	0.04
Systolic basal R rate (°/s)	−77.1 ± 4.6	−77.2 ± 3.4	0.781
Early diastolic basal R rate (°/s)	62.3 ± 4.3	71.2 ± 3.8	0.02
Systolic apical R (°)	8.51 ± 0.97	5.97 ± 0.53	0.018
Systolic apical R rate (°/s)	78.0 ± 6.8	77.0 ± 3.8	0.54
Early diastolic apical R rate (°/s)	−74.1 ± 9.5	−79.9 ± 4.2	0.21
LV twist (°)	11.7 ± 1.1	8.6 ± 0.7	0.002
LV torsion (°/cm)	1.7 ± 0.2	1.3 ± 0.1	0.002
Longitudinal strain			
Systolic Ls (%)	−13.3 ± 0.82	−13.0 ± 0.4	0.07
Systolic Ls rate (1/s)	−1.09 ± 0.05	−1.03 ± 0.04	0.127
Late diastolic Ls rate (1/s)	0.84 ± 0.05	1.01 ± 0.03	0.011
Radial strain			
Systolic Rs (%)	25.3 ± 1.9	25.1 ± 1.1	0.09
Early diastolic Rs rate (1/s)	−2.2 ± 0.1	−1.9 ± 0.5	0.09
Circumferential strain			
Systolic Cs (%)	−14.8 ± 0.8	−13.8 ± 0.5	0.06
Early diastolic Cs rate (1/s)	1.8 ± 0.1	1.7 ± 0.1	0.432

Values are mean ± standard error.
LV, left ventricular; R, rotation; Ls, longitudinal strain; Rs, radial strain; Cs, circumferential strain.

(P = 0.006 and P = 0.04, respectively) (Table 2). A positive correlation was observed between the E/E′ ratio and fT3 levels on day 4 (r = 0.22; P = 0.02).

Regarding the strain analysis, the systolic apical rotation was higher in group 1 than in group 2 (P = 0.018). Significant differences between the groups were found in the systolic basal rotation and early diastolic rotation rate. LV twist and torsion were higher (P = 0.002), while the late diastolic longitudinal strain rate – lower in group 1 than in group 2 (P = 0.011). There were no significant differences in the systolic longitudinal, radial, or circumferential strain or strain rate between the groups (Table 3).

A positive correlation was detected between the systolic basal rotation and the level of fT3 on day 4 (r = 0.4; P < 0.001), while the correlation between LV rotation parameters such as systolic apical rotation on day 1 (r = −0.2; P < 0.05), twist, torsion (r = −0.3; P < 0.001), and diastolic apical rotation rate (r = −0.3; P < 0.01) on day 4 and fT3 was negative. The late diastolic longitudinal strain rate indices showed significant correlations with fT3 on day 1 (r = 0.32; P < 0.001) and on day 4 (r = 0.35; P < 0.001) (Fig. 4).

3.3. *Reproducibility*

The Bland–Altman analysis showed 95% limits of interobserver agreement. The mean differences in Ls measurements were −0.24% (−4.14% to 3.66%); in Rs, 1.88% (−5.74% to 9.5%); in Cs, −0.87% (−7.8% to 6.06%); in basal rotation, −0.71% (−3.87% to 2.44%); and in apical rotation measurements, −0.04% (−5.12% to 5.04%).

4. Discussion

The aim of our study was to evaluate the prevalence of low-T3 syndrome among patients with AMI, and its relationship with LV rotation and deformation parameters assessed by STE. The mechanisms underlying the low-T3 syndrome observed in AMI are not fully understood. The secretion of the thyroid hormone is controlled by the pituitary gland and its secretion of TSH, which is stimulated by the hypothalamic thyrotropin-releasing hormone (TRH). The main product of the thyroid gland is T4, which is converted into T3. The conversion of T4 to T3 is catalyzed by deiodinases. Some investigators explained that low deiodinase type 1 (D1) activity could result from an increase in serum interleukin level (particularly, interleukin 6), which occurs after AMI [10]. The TH-degrading enzyme deiodinase type 3 (D3) converts T3 into the inactive metabolite rT3. It has also been found that many cardiac genes involved in the contractile dysfunction following pathological ventricular remodeling are transcriptionally regulated by TH and TH receptors (TRs). A number of factors stimulate D3 activity through transcriptional activation of the DIO3 gene directly or in combination with other factors. These factors include the

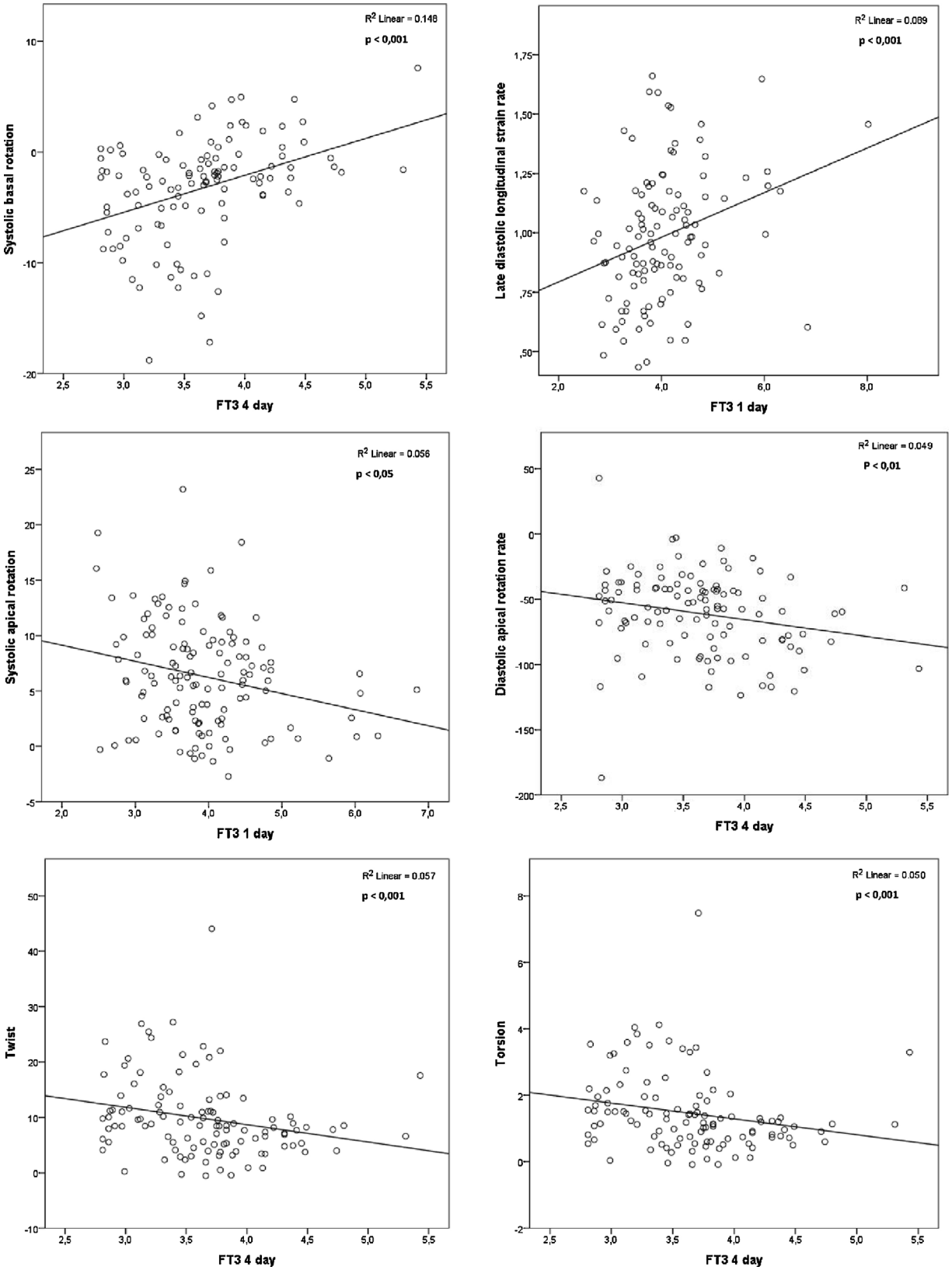

Fig. 4 – Significant correlations between parameters.

transforming growth factor (TGFβ), the mitogen-activated protein kinase (MAPK) system, and the hypoxia-inducible factor 1 (HIF-1). The increased D3 activity converts fT3 into the inactive metabolite rT3, resulting in reduced T3 levels, which in turn affects contractile activity and energy metabolism [11].

Inflammation, hypoxia, and hemodynamic instability might also contribute to the decrease in T3 levels [12,13]. Low thyroid hormone state, particularly, low fT3 levels, is found in ischemic myocardial conditions, and may be a protective response against ischemic stress [14]. T3 down-regulation occurs in the AMI phase, while TSH and fT4 levels do not change significantly. In this study, we found a low-T3 state in 26% of patients with AMI, compared to around 30% indicated in literature [15,16]. We noticed a decrease in fT3 levels on the fourth day after the onset of AMI symptoms, while TSH increased but was not above the standard limit. Due to the fact that we did not find any correlations between echocardiographic/STE parameters and fT4 and TSH, we did not provide any results about this. All the observed correlations were with fT3.

Obesity and dyslipidaemia are really more prevalent in the group with low-T3 state. Thyroid hormones modulate enzyme activity and receptor expression, and can alter cholesterol metabolism through multiple mechanisms, including a decrease in biliary excretion [10]. The reduction in fT3 levels could be a model of abnormal thyroid hormone metabolism acting as a risk factor for coronary heart diseases [5]. The increase in cardiovascular risk associated with thyroid dysfunction is not only related to changes in lipid levels, but also to changes in hemodynamics, the endothelial function, coagulation, metabolism, and C-reactive protein – although inflammation parameters did not differ in our population.

We evaluated LA and LV systolic and diastolic function using conventional echocardiography. When comparing our two groups, LA proved to be significantly larger in the low T3 group. This data pertain to the conclusions of Ozturk et al. indicating that the mechanical and electromechanical function of LA is affected in subclinical thyroid disorders [17]. Moreover, a significant decrease in the E/E′ ratio, septal E′, and deceleration time in patients with low T3 state was revealed. A lower vs. normal level of triiodothyronine aggravates myocardial function by suppression of the diastolic function. T3 affects the diastolic function and left ventricular relaxation through the activation of the sarcoplasmic reticulum Ca^{2+}-ATPase which, as a result, reduces cytosolic calcium. TH encode the synthesis of proteins such as phospholamban, sarcoplasmic reticulum proteins, and heavy chains of myosin, which play an important role in cardiac functioning, determining diastolic and systolic function [18]. It is noteworthy that LVEF was not markedly decreased, especially in the low fT3 group, which we link with an early stage of myocardial damage when no obvious changes are seen.

Recent studies prove STE to be a superior method for the evaluation of global and regional LV function. STE may reveal earlier changes in the myocardium that may not be seen on conventional echocardiography at that time; furthermore, STE gives a more precise insight into the damaged ischemic area, whether it comprises only subendocardial or further – transmural – necrosis [19,20].

The obtained images of strain and strain rate and data from its analysis have already revealed that longitudinal deformation mainly depicts subendocardial contraction and relaxation (it decreased in patients with subendocardial infarcts when LVEF was preserved), whereas circumferential strain denotes mainly mid-myocardial and subepicardial contraction [19,21]. As shown in previous studies, the systolic longitudinal strain (cut-off value at −11.6%) is an independent determinant of LV remodeling [22]. In our study, the systolic longitudinal strain and strain rate were decreased, but did not differ between the groups, while a lowered late diastolic longitudinal strain rate – which correlated with fT3 – was observed on the first day. These findings support previous reports, where suppression of LV diastolic function in patients with low T3-syndrome was noted. Recent studies have shown an impaired longitudinal myocardial diastolic and systolic function but preserved circumferential function and LV twist in patients with the metabolic syndrome [23]. Obesity and hyperlipidemia were more prevalent in patients with low T3 state. We analyzed differences between groups, and did not find any relationships between obesity, hyperlipidemia and systolic longitudinal strain and late diastolic longitudinal strain rate and rotation parameters. According to our findings, LV twist (11.7°) exceeded the mean value (8.0°) in patients with low fT3 levels, while it remained in a normal range in patients with normal fT3 levels. This increase in LV twist can be explained by a less opposed apical rotation, resulting from a gradual diminution in the subendocardial function [19]. This, in turn, may result from the suppressing effect of the low T3 state.

Low-T3 syndrome reached its peak on the fourth day when it positively correlated with LV systolic basal rotation, and inversely with LV systolic apical rotation, twist, torsion, and diastolic apical rotation. Some authors have detected the second wave of the highest concentration of matrix metalloproteinases, enzymes responsible for collagenolysis, on the fourth day after AMI [24]. This correlates with creatine kinase and possibly reveals the real extent of cardiac injury. Thus, correlations obtained from strain imaging may indicate values of deformation and rotation to be early signs of myocardial damage, and may reveal the true extent of myocardial damage. T3 levels could be a significant determinant of myocardial function – especially under stress conditions such as AMI.

According to our study data, LV dysfunction can be early assessed by using STE in patients with low-T3 state and normal ejection fraction values.

The main limitation of our study was that echocardiography was performed during the acute phase of MI. Furthermore, we have not investigated TH during the late period after AMI, and therefore it remains unclear whether TH decline is transient and whether it is restored following the acute phase. Baseline levels of TH were not available. To conclude with one cannot draw whether low-T3 was induced by AMI or preceded the ischemic event. Total T3 levels were not available. We chose a cross-sectional design of the study, and thus it was not possible to investigate the probable association of TH with mortality after an AMI.

5. Conclusions

Speckle-tracking echocardiography is a technique that can be applied early, and is superior to standard echocardiography in

a detailed evaluation of LV systolic and diastolic function among patients with low-T3 syndrome after AMI. With the use of STE, we proved that impaired thyroid function during AMI is associated with an impaired LV late diastolic longitudinal strain and LV rotation, but not with the radial or circumferential LV function.

Conflict of interest

The authors declare that they have no competing interests.

Authors' contributions

E.J., P.O., J.J.V.: design, data collection, drawing up the manuscript, data analysis and statistics. All authors: design, critical revision of the article, and approval of the article. All authors read and approved the final manuscript.

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