

Association between Pentraxin-3 and Cardiac Troponin-I with Left Ventricular Systolic Function in Patients with Anterior ST-Segment Elevation Myocardial Infarction: Evaluation by Speckle Tracking Echocardiography

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Abstract

Background: Pentraxin 3 is an inflammatory biomarker whose serum level is increased during acute myocardial infarction (AMI). The aim of this study was to measure the serum pentraxin-3 level in anterior ST-segment elevation myocardial infarction (STEMI), to investigate correlation with the left ventricular (LV) systolic function assessed by speckle tracking echocardiography, and compare with the well-established cardiac biomarkers of myocardial injury such as troponin-I. **Methods:** Serum pentraxin-3 level was measured by ELISA in 50 patients with anterior STEMI. LV strain was measured by speckle tracking echocardiography. The results were compared to twenty sex- and age-matched persons who had history of stable angina and normal LV ejection fraction (LVEF). **Results:** Serum level of pentraxin-3 was significantly higher in STEMI patients in comparison to the control group (8.3 ± 3.1 versus 3.4 ± 1.2 ng/ml, $p < 0.001$). Average LV global longitudinal strain (GLS) was reduced in STEMI patients in comparison to control subjects (11.2 ± 2.4 versus 20.2 ± 2.1 , $p < 0.001$). In addition, there was a significant negative correlation between serum pentraxin-3 level and LVEF ($r = -0.557$, $p < 0.001$) and the average LVGLS ($r = -0.529$, $p < 0.001$). Serum pentraxin-3 cutoff value > 8.3 ng/ml had sensitivity of 81.8% and specificity of 86.4% to detect LVEF less than 50% ($p < 0.001$). While, peak cardiac troponin-I level > 34 ng/ml was 86% sensitive to detect LVEF less than 50% with specificity of

90.9%. **Conclusions:** The elevated serum level of pentraxin-3 and cardiac troponin-I in STEMI patients and their association with both LVEF and GLS support the concept that the magnitude of these biomarkers correlates to the severity of myocardium injury.

Keywords

Anterior MI, Pentraxin-3, Speckle Tracking Echocardiography

1. Introduction

Pentraxin-3 is an inflammatory marker, which is overexpressed with high levels in atheroma and cardiac muscle cells. A role for pentraxin-3 in initiation and progression of cardiovascular diseases was proposed [1] [2].

Although some studies reported adverse effects of pentraxin-3 in thrombotic events, others indicated a protective role. Pentraxin-3 is engaged with different molecular mechanisms leading to vascular injury. During inflammatory process, pentraxin-3 is produced in a large amount by blood vessels that alter the cardiovascular system [3] [4]. Pentraxin-3 increases expression of the tissue factor, and binds to fibroblast growth factor 2, which could initiate plaque instability and thrombus formation [5] [6] [7].

After an acute event, serum level of pentraxin-3 surges over time, reaches its peak level within about 7 hours and returns to basal level after 3 days [8]. We sought to measure serum pentraxin-3 level in anterior STEMI to correlate with systolic function of left ventricle, which is assessed by speckle tracking echocardiography. Additionally, the diagnostic sensitivity of pentraxin-3 will be compared with the well-established cardiac biomarkers of myocardial injury (troponin-I, in particular).

2. Methods

2.1. Study Population

The number of patients included in the current study was 50 (55.6 ± 10 years). Patients were admitted by anterior STEMI, in King Fahad hospital of the university, Saudi Arabia in the period between January 2017 and December 2017. Acute STEMI was diagnosed by rise and fall of cardiac enzymes together with at least one of signs of myocardial ischaemia (chest pain, new ST-elevation at the J-point in 2 contiguous leads with the cut-point ≥ 1 mm in leads other than leads V2 - V3 where the following cut points apply: ≥ 2 mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age, and development of pathological Q waves) [9].

Post-operative MI, preexisting cardiomyopathies, significant valvular heart disease, chronic renal and liver conditions were all set as exclusion criteria. 20 sex- and age-matched persons who had history of stable angina and normal

LVEF were assigned as controls. The study was approved by local ethics committee, and informed consent was obtained from all patients before participation in the study.

2.2. Standard 12-Lead Electrocardiography

A twelve lead ECG was done for all patients using MAC 3500 resting ECG analysis system (GE healthcare, Milwaukee, WI, USA) with a 25 mm/s paper speed and standardization adjusted to 1 mm/mv. An anterior STEMI was diagnosed by the presence of an elevated ST segment in leads representing the anterior wall (V1 - V6).

2.3. Two Dimensional (2D) Resting Echocardiography

A 2D transthoracic echocardiography was carried out (VIVID E9 machine; GE Healthcare, Chicago, IL, USA). The standard parasternal and apical imaging views were used, and the average of three consecutive heartbeats was then studied. The standard 2D and M-mode measurements were recorded. The left ventricle ejection fraction (LVEF) was evaluated by the method of biplane disks (the modified Simpson's rule). The recommendations of the American Society of Echocardiography were applied when registering all of the 2D and conventional Doppler variables [10]. Tissue Doppler Imaging (TDI), Pulsed Doppler Echocardiography, and Left Atrial Volume Index (LAVI), and tricuspid regurgitation (TR) velocity were employed to assess the diastolic function the left ventricle. Recommendations of the American Society of Echocardiography were followed in this regard [11].

2.4. Speckle-Tracking Echocardiography

All of the data were stored digitally in a cine loop arrangement, and the speckle-tracking imaging of the LV was done offline using an EchoPAC workstation (GE Healthcare, Chicago, IL, USA). The frame rate was adjusted from 60 to 100 frames per second to display grayscale 2D images. The time of the aortic valve closure was estimated at the beginning of the study through the apical long axis view. This was then followed by views of the apical four and the apical two chambers. Automated functional imaging (AFI) [12] was used to record the LV GLS. The software allows automated tracing of the regions of interest to identify the whole myocardial wall. To achieve a better tracking score, the endocardial trace line was adopted in those segments with poor traceability. Each LV wall was subdivided into six segments, and the intra-segmental peak systolic strains were then calculated by computer software. GLS-Average represents the summative peak systolic longitudinal strains of the entire LV segments.

2.5. Measurement of Serum Pentraxin-3

Protein concentration was estimated in serum samples that were collected at 24 hours of chest pain onset using the BCA protein estimation kit (ThermoFisher

Scientific, USA). Appropriate sample dilution was done prior to enzyme-linked immunosorbent assay (ELISA) of pentraxin-3. Standard stock was serially diluted and all samples were loaded into the pre-coated ELISA plate. The procedure was followed according to the instructions in the datasheet of human pentraxin 3 ELISA kit (#SEK411Hu, Cloud-Clone Corp., Katy, Texas, USA). The concentration of pentraxin-3 in control and patient samples was derived from the standard curve equation. Other laboratory investigations were done including creatine Kinase-MB (CK-MB), troponin-I, lipid profile, complete blood count and serum creatinine.

2.6. Statistical Analysis

Data analysis was done using the IBM SPSS software package version 20.0 (IBM Corp, Armonk, NY, USA). Normal distribution of data was investigated using the Kolmogorov-Smirnov, Shapiro-Wilk, and D'agostino tests. Group statistical comparisons were done using chi-squared test (Fisher or Monte Carlo) for the categorical variables. The Student's t-test was used to compare the statistical difference between quantitative variables that follow the Gaussian distribution. The Mann-Whitney U test was used to compare the statistical differences between quantitative variables that do not meet the criteria of Gaussian distribution. Spearman's and Pearson's coefficient tests were used to study correlation between the variables. P values ≤ 0.05 indicate statistically significant difference.

3. Results

3.1. Characteristics of Patients Included in the Study

The current study cohort consists of 50 anterior STEMI patients. Male patients represent 62% (31 M) of the study population, whereas female subjects constitute 38% of the study population (19 F) (55.6 ± 10 years). Amongst patients, sixteen (32%) of cases were hypertensive, and 25 (50%) were diabetic and 12 (24%) had family history of CAD. Twenty sex- and age-matched subjects with a history of stable coronary artery disease were assigned as controls (**Table 1** and **Table 2**).

3.2. 2D, Doppler and TDI Echocardiography

2D Doppler and TDI echocardiography showed that LVEF was significantly decreased in patient with STEMI when compared to the control. A significant reduction in the peak systolic velocity at mitral annulus (Sa) velocity was observed in STEMI patients than in control group. LV diastolic filling parameters varied significantly between both groups reflecting impaired LV diastolic filling in STEMI group. The mitral E velocity, E/A ratio and TDI mitral annulus Ea wave were significantly reduced ($p < 0.001$), while the A velocity, E/Ea ratio and LAVI were increased in STEMI group as compared to the control group; this increase was statistically significant, $p < 0.001$ for A velocity and E/Ea, and 0.002 for LAVI (**Table 3**).

Table 1. Study population characteristics.

	Control (n = 20)	STEMI (n = 50)	P
Age (years)	52.8 ± 9.5	55.6 ± 10.0	0.276
Sex (male, %)	14 (70%)	31 (62%)	0.528
BSA	1.8 ± 0.1	1.9 ± 0.1	0.163
BMI (kg/m ²)	27.8 ± 2.9	28.3 ± 3.1	0.520
HTN (n, %)	5 (25%)	16 (32%)	0.774
DM (n, %)	8 (40%)	25 (50%)	0.597
Dyslipidemia (n, %)	9 (45%)	27 (54%)	0.793
Family history of CAD (n, %)	2 (10%)	12 (24%)	0.321
Smoking (n, %)	8 (40%)	22 (44%)	0.760
Systolic BP (mmHg)	128 ± 13.6	129.5 ± 12.4	0.637
Diastolic BP (mmHg)	78.4 ± 7.3	80 ± 7.5	0.362
Serum creatinine (mg/dl)	1.1 ± 0.19	1 ± 0.24	0.772
Total Cholesterol (mg/dl)	191 ± 26	194.4 ± 33.9	0.659
Triglycerides (mg/dl)	138.4 ± 42.7	142.2 ± 58.6	0.792
LDL (mg/dl)	125 ± 22	127 ± 32	0.842
HDL (mg/dl)	37.5 ± 7	39 ± 6.8	0.394
HbA1C	6.5 ± 1.1	6.9 ± 1.8	0.382

BP: blood pressure, DM: diabetes mellitus, BSA: body surface area, BMI: body mass index, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, CAD: Coronary artery disease.

Table 2. Coronary angiography data and medications of STEMI patients.

Variable	STEMI group (N = 50)
Primary PCI (n, %)	47 (94%)
Medications	
Aspirin (n, %)	50 (100%)
Clopidogrel (n, %)	50 (100%)
ACEI (n, %)	44 (88%)
Statins (n, %)	50 (100%)
Beta blockers (n, %)	43 (86%)
Peak Troponin-I, ng/ml, median (Min. - Max.)	35.5 (14 - 134)
Peak CK- MB, U/l, median (Min. - Max.)	307 (145 - 550)
Coronary angiography	
Culprit artery, %	LAD (100%)
Single vessel disease (n, %)	11 (22%)
Two vessel disease (n, %)	21 (42%)
Three or more vessel disease (n, %)	18 (36%)

PCI: Percutaneous coronary intervention, LAD: left anterior descending artery, ACEI: angiotensin Converting enzyme inhibitors, CK-MB: creatine Kinase-MB.

Table 3. Comparison between the two studied groups according to 2D and Doppler echocardiographic findings.

	Control (n = 20)	STEMI (n = 50)	p
LV septal wall thickness (cm)	1 ± 0.2	1.1 ± 0.2	0.257
LV end diastolic volume (ml)	91.8 ± 10.5	106.3 ± 20.8	<0.001
LV end systolic volume (ml)	36.8 ± 4.4	54.5 ± 10.7	<0.001
LV ejection fraction (%)	59.3 ± 5.3	47.7 ± 5.5	<0.001
Left atrium dimension (cm)	3.5 ± 0.4	3.8 ± 0.4	0.015
LAVI (ml/m ²)	26.6 ± 2.8	29.6 ± 5.1	0.002
E (cm/s)	76.8 ± 13.1	61.1 ± 9.4	<0.001
A (cm/s)	51.6 ± 7.4	63.6 ± 8.3	<0.001
E/A ratio	1.5 ± 0.3	1 ± 0.2	<0.001
DT (ms)	178.8 ± 26.2	197.2 ± 31.1	0.022
TR Velocity	2.63 ± 0.49	2.88 ± 0.4	0.025
Sa (cm/s)	10.4 ± 1.9	7.7 ± 1.4	<0.001
Ea (cm/s)	12.3 ± 1.9	7.7 ± 1.4	<0.001
E/Ea	6.4 ± 1.4	8.2 ± 1.8	<0.001
Aa (cm/s)	9.1 ± 2	8.9 ± 2	0.703

LAVI: left atrial volume index, E: early peak pulsed Doppler velocity on mitral valve, A: late pulsed Doppler velocity on mitral valve, DT: deceleration time, TR: tricuspid regurgitation, Sa: peak systolic velocity at mitral annulus, Ea: early tissue velocity of the mitral annulus, Aa: late tissue velocity of the mitral annulus.

3.3. LV Speckle-Tracking Longitudinal Strain

LV speckle tracking longitudinal strain results are demonstrated in **Table 4**. Average GLS was reduced in cases in comparison to control subjects (-11.2 ± 2.4 versus -20.2 ± 2.1 , $p < 0.001$) (**Table 4** and **Figure 1**).

3.4. Serum Pentraxin-3 in Studied Group and Correlation Analysis

Cases group revealed significant higher levels of pentraxin-3 in comparison to control group (8.3 ± 3.1 versus 3.4 ± 1.2 ng/ml, $p \leq 0.001$) (**Table 5** and **Figure 2**).

The association between serum pentraxin-3 level and LVEF was statistically significant as confirmed by correlation studies ($r = -0.572$, $p < 0.001$) and average LVGLS ($r = -0.475$, $p < 0.001$) (**Figure 3**). There was also a significant association between peak troponin-I level and LVEF ($r = -0.557$, $p < 0.001$) and LVGLS ($r = -0.529$, $p < 0.001$) (**Table 6**).

28 (56%) STEMI patients had LVEF less than 50%. Serum pentraxin-3 cutoff value > 8.3 ng/ml showed an estimated sensitivity of 81.8% and specificity of 86.4% to detect LVEF less than 50% ($p < 0.001$). While, peak cardiac troponin-I level > 34 ng/ml was 86% sensitive to detect LVEF less than 50% with specificity of 90.9% (**Table 7** and **Figure 4**).

Table 4. Comparison between the two studied groups according to LV global longitudinal strain.

	Controls (N = 20)	STEMI group (N = 50)	P
Average GLS %, Mean \pm SD	-20.2 \pm 2.1	-11.2 \pm 2.4	<0.001

GLS: global longitudinal strain.

Table 5. Serum pentraxin-3 levels in control and study groups.

	Controls (N = 20)	STEMI group (N = 50)	P
Pentraxin-3 serum (ng/ml) Mean \pm SD	3.4 \pm 1.2	8.3 \pm 3.1	<0.001

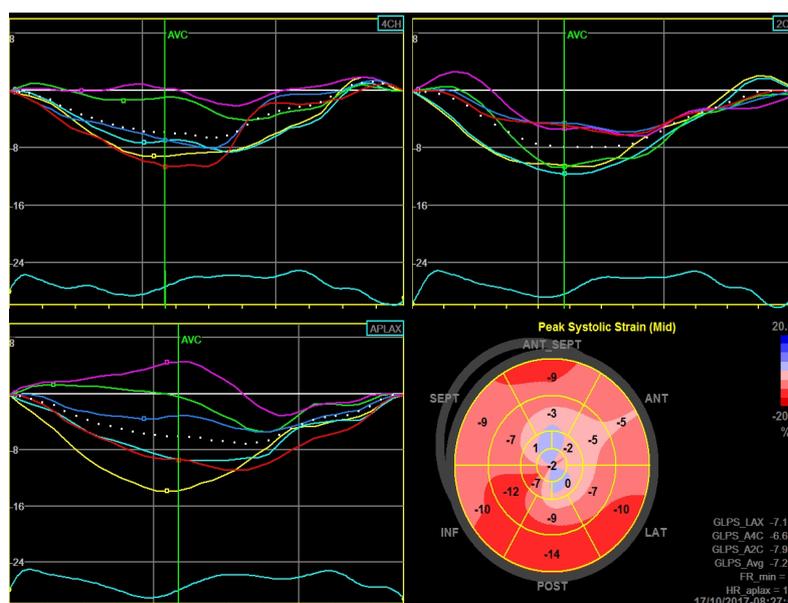
Table 6. Correlation of pentraxin-3 and troponin-I with LV systolic function in STEMI group.

	Pentraxin-3		Troponin I	
	r	p	r	p
LVEF%	-0.572	<0.001	-0.557	<0.001
Average GLS%	-0.475	<0.001	-0.529	<0.001

r: Pearson coefficient.

Table 7. Agreement for pentraxin-3 and troponin-I to diagnose LVEF < 50% in cases group.

	Cut off value	Sensitivity	Specificity	PPV	NPV
Pentraxin-3 (ng/ml)	>8.3	81.82	86.36	85.7	82.6
Troponin I (ng/ml)	>34	86.36	90.91	90.5	87.0

**Figure 1.** Bull's-eye view shows longitudinal strain of all individual LV segments and a value for global LV longitudinal strain in patient with anterior infarction.

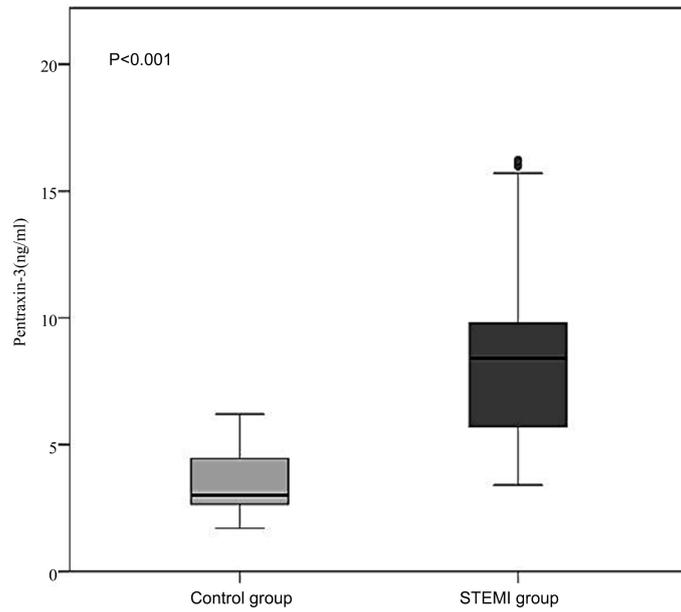


Figure 2. Box plot showing the median and inter quartile rang of pentraxin 3 among the studied groups.

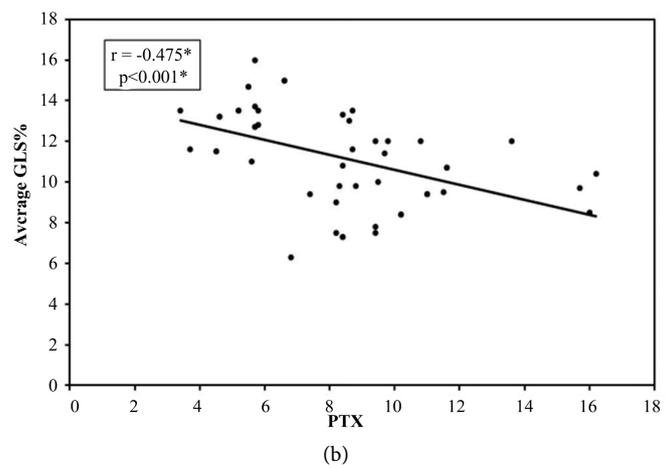
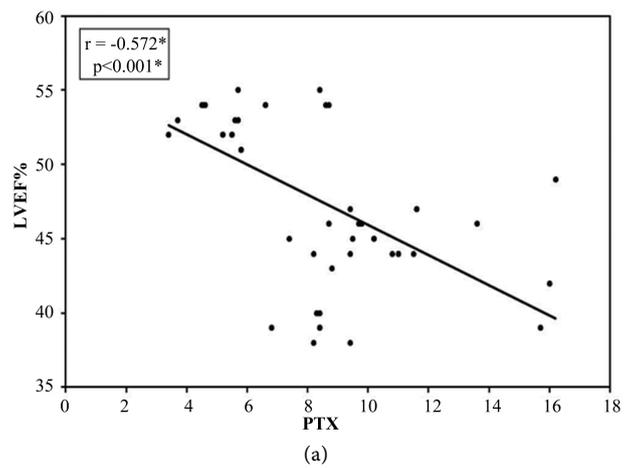


Figure 3. (a) Correlation between pentraxin-3 and LVEF in cases group; (b) Correlation between pentraxin-3 and average GLS% in cases group.

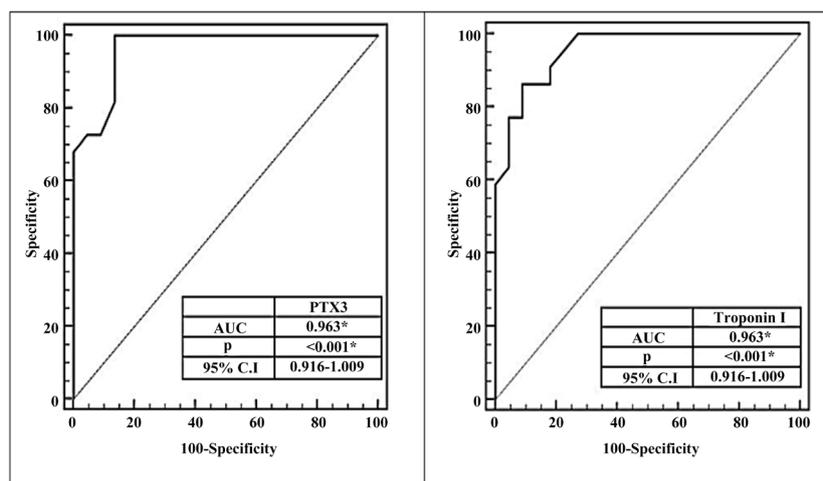


Figure 4. ROC curve for pentraxin-3 (right panel) and cardiac troponin-I (left panel) to predict LVEF < 50%. AUC: Area Under a Curve, P value: Probability value, CI: Confidence Intervals.

4. Discussion

The current case-control study evaluated serum levels of pentraxin-3 in patients presented with anterior STEMI and investigated the relationship between LV longitudinal strain and pentraxin-3 levels. Significant higher pentraxin-3 values were observed in STEMI patients in comparison to the levels in subjects with stable angina (8.3 ± 3.1 versus 3.4 ± 1.2 ng/ml). Several previous studies [13]-[18] have addressed the validity and the prognostic role of pentraxin-3 in ACS. According to these studies, significant elevation in pentraxin-3 levels were reported in ACS patients when compared to controls, variation in serum levels was observed though. Such variations may be related to different timing of blood sampling and/or ELISA kits that were used to estimate pentraxin-3 levels. In this respect, Peri G and colleagues in a similar study had reported that pentraxin-3 levels were peaked at 7.5 hours with a mean peak value of 6.94 ± 11.26 ng/ml. In addition, diagnostic autopsy of myocardial samples from 12 patients who died by STEMI were done.

Results revealed that pentraxin-3 was present in normal and hypertrophied myocytes; meanwhile no pentraxin 3 was detected in necrotic myocytes. Taken these results together, authors concluded that serum level of pentraxin-3 rises in patients with STEMI as a consequence of its release from necrotic myocytes [8].

The current study suggested that the average GLS was reduced in STEMI patients than in control subjects. Additionally, there was a strong negative association between peak troponin-I and pentraxin-3 level and both LVEF and GLS. George M and co-authors have reported similar results in regard to pentraxin-3 level in ACS. They also stated a moderate correlation between LV systolic function and pentraxin-3 levels. In patients who had serum pentraxin-3 level ≥ 6.5 ng/ml, LVEF was significantly reduced [19].

The current study concluded an estimated sensitivity and specificity of 81.8% and 86.4%, respectively for serum pentraxin-3 (cutoff value > 8.3 ng/ml) to iden-

tify LVEF less than 50% ($p < 0.001$). While, peak cardiac troponin-I level > 34 ng/ml was 86% sensitive to detect LVEF less than 50% with specificity of 90%. The cardiac isoform, troponin-I is recognized as a highly reliable cardiac function marker in ACS. Information suggested that cardiac troponin-I is correlated to myocardial necrosis and its level in the blood correlates with the systolic function of the left ventricle and infarct size.

The usefulness of peak troponin-I to predict degree of LV systolic dysfunction evaluated by 2D echocardiography was studied by Shaikh *et al.* They reported peak troponin-I > 63.5 and > 87.5 ng/ml predict impaired LV systolic function in patients received either fibrinolytic therapy or primary PCI respectively for first anterior STEMI [20].

Chia and co-authors used single-photon emission computed tomography to study the association between cardiac biomarkers and LV systolic function. According to the study, a sensitivity of 90% was estimated for the peak troponin-I level cutoff value > 55 ng/ml to detect infarct size equal to, or more than 10% and less than 40% LVEF (specificities were 70% and 52%, respectively). Peak troponin-I level was correlated with LVEF ($r = -0.434$), and infarct size ($r = 0.740$) and this correlation was significant [21].

High serum levels of pentraxin-3 in STEMI patients and its association with both LVEF and GLS support the concept that the magnitude of pentraxin-3 levels correlates to the severity of myocardium injury. This confirms the role of pentraxin-3 as a biomarker of AMI due to its local production and increased blood level after acute event.

An emphasis on the prognostic role of pentraxin-3 in STE-ACS patients was reported by Latini and colleagues, who reported an association between serum pentraxin-3 and 3-months cardiac events after acute STEMI. 724 patients have participated, and after adjustment for major risk factors; median pentraxin-3 value was 7 ng/ml in event-free patients, 9 ng/ml in patients with non-fatal heart failure, and 16 ng/ml in patients who died as a consequence of the disease [22]. In a similar context, George M *et al.* observed greater numbers of deaths in STE-ACS patients having higher levels of pentraxin-3 [19].

5. Conclusion

The elevated serum level of pentraxin-3 and cardiac troponin-I in STEMI patients and their association with both LVEF and GLS support the concept that the magnitude of these biomarkers correlates to the severity of myocardium injury and attract more attention towards the prognostic, as well as, the diagnostic value of pentraxin-3 to monitor acute cardiac events.

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This is no funding authority to declare.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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