The Relationship Between the Frontal QRST Angle and Thrombus Burden in ST-Elevated Myocardial Infarction

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ABSTRACT

Introduction: Despite the relevance of the frontal QRST angle to the distribution, grade, and complexity of coronary artery disease in ST-elevated myocardial infarction (STEMI), until now no studies have particularly addressed the association of frontal QRST angle with thrombus burden in STEMI. The goal of this study was to assess whether a relevance exists between the frontal QRST angle and the thrombus burden in STEMI.

Patients and Methods: In this retrospective cross-sectional analysis, 192 STEMI patients who underwent primary percutaneous coronary intervention were separated according to TIMI thrombus grade; those with a high thrombus burden (46 patients) and those with a low thrombus burden (146 patients) included. Thrombus burden was categorized according to thrombolysis in myocardial infarction (TIMI) thrombus grades. Frontal QRST angle calculation was done as the absolute value of the difference of the QRS and T axes.

Results: In multivariable analysis, frontal QRST angle (OR= 1.270, 95% CI= 1.140-1.410, p= 0.001), C-reactive protein (OR= 1.185, 95% CI= 1.015-1.383, p= 0.032), and troponin I (OR= 1.295, 95% CI= 1.091-1.536, p= 0.003) were independently associated with high thrombus burden. In ROC analysis, the value of 76.5 for frontal QRST angle had 76% sensitivity and 74% specificity [area under curve (AUC)= 0.76, p< 0.001] for the estimation of high thrombus burden in STEMI.

Conclusion: Frontal QRST angle is a useful tool to detect high thrombus burden in STEMI.

Key Words: Frontal QRST angle; thrombus burden; ST-elevated myocardial infarction.

ST-Yükselmeli Miyokart Enfarktüsünde Frontal QRST Açısı ile Trombüs Yükü Arasındaki İlişki

ÖZ

Giriş: ST-yükselmeli miyokart enfarktüsü (STEMI) gelişen hastalarda frontal QRST açısı ile koroner arter hastalığının yaygınlığı, şiddeti ve karmaşıklığı arasındaki ilişkisine rağmen bugüne kadar özellikle frontal QRST açısının trombüs yükü ile olan ilişkisine değinen hiçbir çalışma yoktur. Bu çalışmanın amacı, STEMI gelişen hastalarda frontal QRST açısı ile trombüs yükü arasında bir ilişki olup olmadığını değerlendirmektir.

Hastalar ve Yöntem: Bu retrospektif kesitsel analizde, primer perkütan koroner girişim uygulanan 192 STEMI hastası TIMI trombüs derecesine göre ayrılmıştır. Çalışmaya, yüksek trombüs yükü olanlar (46 hasta) ve düşük trombüs yükü olanlar (146 hasta) dahil edilmiştir. Trombüs yükü, miyokard infarktüsü trombüs derecelerinde trombolize göre kategorize edilmiştir. Frontal QRST açısı, ECG'ye göre frontal düzlem QRS ve T eksenleri arasındaki farkın mutlak değeri olarak hesaplanmıştır.

Bulgular: Çok değişkenli analizde frontal QRST açısı (OR= 1.270, %95 CI= 1.140-1.410, p= 0.001), C-reaktif protein (OR= 1.185, %95 CI= 1.015-1.383, p= 0.032) ve troponin I seviyesi (OR= 1.295, %95 CI= 1.091-1.536, p= 0.003) yüksek trombüs yükünün bağımsız öngördürücüleriydi. ROC analizinde yüksek trombüs yükünü tahmin etmek için frontal QRST açısının sınır değeri 76.5, duyarlılığı %76, özgüllüğü %74 [eğri altında kalan alan (AUC)= 0.76, p< 0.001] bulunmuştur.

Sonuç: Frontal QRST açısı STEMI'de yüksek trombüs yükünü tahmin etmede yararlı bir araçtır.

Anahtar Kelimeler: Frontal QRST açısı; trombüs yükü; ST-yükselmeli miyokart enfarktüsü.

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INTRODUCTION

Acute coronary syndrome (ACS) remains the major reason of cardiovascular mortality and urgent diagnosis and prompt treatment is needed for all ACS patients. Plaque rupture in the coronary arteries and following thrombus formation and cessation of blood flow is the main pathophysiology mechanism for ACS. High thrombus burden is a major determinant of outcomes in ST-elevation myocardial infarction (STEMI) [1]. Primary percutaneous coronary intervention (PCI) to the artery causing acute STEMI limits the myocardial damage and subsequently improves the prognosis and left ventricular functions. However, increased thrombus formation in the coronary arteries affects the procedural success adversely[2]. During PCI, embolization of the thrombus to the distal territory may increase the size of the infarction and depress myocardial functions due to microvascular obstruction[3]. It has been also shown that increased thrombus burden is also associated with other periprocedural complications like no-reflow phenomenon and acute stent thrombosis[4]. Management of thrombus in the coronary arteries is still a problematic issue waiting for a solution Intracoronary and intravenous treatment with glycoprotein IIb/IIIa and mechanical thrombectomy are now used without a clear net benefit for the treatment of intracoronary thrombus[5,6]. Based on the aforementioned information, the estimation of increased thrombus burden by a non-invasive method promptly after admission to the hospital may contribute to improving outcomes of STEMI patients by selecting the optimal medical and interventional strategy. Admission monocyte-HDL ratio (MHR) and chemerin levels were main predictors of high thrombus formation in STEMI patients undergoing primary PCI[7,8]. In addition to these biochemical parameters, epicardial adipose tissue (EAT) as an inflammatory organ measured by echocardiography have been reported to be associated with high thrombus burden in STEMI patients who undergo primary PCI[9]. Frontal QRS-T (QRST) angle, one of many electrocardiogram parameters as a determinant of myocardial depolarization and repolarization heterogeneity, is reported automatically and easily derived from most of the 12-lead electrocardiography (ECG) devices[10,11]. A wider frontal QRST angle has been claimed to be firmly associated with higher cardiovascular mortality and poor prognosis in ACS patients[12,13]. Nonetheless, it was also demonstrated that patients with increased frontal QRST angle had more severe coronary artery disease in STEMI[14,15]. To date, no study has evaluated the relevance of frontal QRST angle to thrombus burden in STEMI. Therefore, in the current study, the goal was to find out a relationship between admission frontal QRST angle and angiographic intracoronary thrombus burden in STEMI patients receiving primary PCI.

PATIENTS and METHODS

Study Population

The present research was conducted as an observational retrospective one center analysis of relevance of frontal QRST angle to thrombus burden in STEMI patients undergoing primary PCI. Consecutive 192 cases accepted to the our cardiology clinic with the identification of STEMI between June 2020 and November 2020, who received the primary PCI, were registered in this study. The identification of STEMI was done in the presence of prolonged typical angina (> 20 minutes) with ST line elevation of ≥ 1 mV in two or more adjacent extremity or precordial ECG leads. Major exclusion criteria included thrombolytic treatment within 24 hours, decompensated heart failure in the past, prior percutaneous coronary angioplasty, bypass surgery, ongoing infectious processes, inflammatory or immunologic syndromes, liver disorders, peripheral vascular obstruction, chronic obstructive lung disorders, chronic kidney failure, oncological diseases, transfusion of blood products in the past 3 months, cardiogenic shock. The medical database of 207 patients were scanned and utilized. Consequently, after the gathering of suitable patients, the final cohort comprised of 192 STEMI cases. The study was administered as required with the ethical rules explicited in the Declaration of Helsinki. Informed assent was signed by overall participants. The local research ethics committee approved the study protocol (approval number 2021/1/5). The diagnosis of hypertension was made as systolic blood pressure 140 mmHg and/or diastolic blood pressure 90 mmHg on regular measurements, or use of any antihypertensive drug. The diagnosis of diabetes mellitus was confirmed when a fasting plasma glucose level of > 126 or > 200 mg/dL at any measurement or use of any antidiabetic drug. Family history of premature CAD was called as reported proof of CAD in a close relative (men < 55 and women < 65 years of age). Active smoking was noted as used to smoking within the past year. Systolic arterial pressure below 80 mmHg with hints of end-organ hypoperfusion was described as cardiogenic shock[16].

Laboratory Measurements

Antecubital vein was used for obtaining peripheral blood samples after the patients were hospitalized in order to the assessment of complete blood count, troponin values, liver and kidney function tests and coagulation tests. Lipid panel and remaining biochemical biomarkers were determined via standard measurements in the next morning within at least 8 hours of fasting state. Anticoagulated EDTA tubes were used for the complete blood count (CBC). Complete blood count tested hemogram parameters and C-reactive protein values as part of the standard protocol.

ECG and Echocardiography

An admission 12-lead surface ECG was obtained in supine position promptly prior to coronary angiography and second ECG was carried out 48 hour after PCI. All first ECGs were reviewed and loaded to a personal computer to make the measurement errors minimal and after that utilized to 400% amplification by Adobe Photoshop software. Frontal QRST angle calculation was done as the absolute value of the difference between the QRS and T axes. If the difference was above 180°, frontal QRST angle was modified to the minimal angle as 360° minus the absolute value of the difference between the QRS and T axes[10]. Automatic calculation of ECG machine has minimized the error rate of the personal evaluations. Major ECG exclusion criteria include bundle branch block or Q wave on a 12 lead ECG. Echocardiographical visualization was realized by two expert cardiologists who were unaware of the remaining data. Every measurement was made as per the latest guidelines[17]. Left ventricular ejection fraction (LVEF) was quantified via the Modified Simpson's method.

PCI Procedure

All study patients were treated depending on the latest evidence-based ESC guidelines[18]. Once STEMI was confirmed, patients were given a priming dose of 600 mg clopidogrel or 60 mg prasugrel or 180 mg ticagrelor with 300 mg aspirin. Emergent coronary angiography was performed in eligible STEMI patients via standard judkins catheter. The determination of infarct-related artery was made in multiple planes and angles by cine angiography visualization. TIMI 3 flow of the culprit stenosis was provided with PCI via 7F guiding catheter. After 5000 IU of heparin (70 U/kg) was given intravenously, direct stent was implanted in possible cases, and in the other lesions, predilatation with small size balloons was attempted first.

Visual thrombus burden of culprit lesion was categorized as formerly determined by the thrombolysis in myocardial infarction (TIMI) study group[19]: grade 0: no thrombus, grade 1: possible thrombus, grade 2: the thrombus' maximum size is < 1/2 vessel diameter, grade 3: maximum size > 1/2 to < 2 vessel diameters, grade 4: maximum size > 2 vessel diameters, and grade 5: total vessel occlusion due to the thrombus[20]. Thrombus grade was assessed as soon as the restoration of antegrade flow by guidewire crossing or small balloon predilatation. The participants were categorized as low-thrombus burden (grades 1-3) and high-thrombus burden groups (grades 4 and 5) based on ultimate thrombus grade[21].

Statistical Analysis

Kolmogorov-Smirnov test was applied to differentiate the dispersion of continuous variables. If the continuous variables exhibited a normal distribution Student T-test was carried out. Matching of categorical variables was concluded using Chi-square or Fisher's exact tests. Continuous variables were submitted as mean ± SD while categorical variables as count and percentages. ROC analysis was made to calculate the sensitivity and specificity with 95% confidence interval (CIs) for the frontal QRST angle at cutoff values. Because thrombus burden count has not normal distribution in the Kolmogorov-Smirnov test, correlation analysis was performed using Spearman correlation coefficient. Independent predictors of for high thrombus burden were clarified in univariable and multivariable logistic analysis. The variables with a p value < 0.25 in two groups analysis were selected for univariable analysis. A p value < 0.05 was accepted as statistically significant in multivariable analysis. Model explanatory, such as R 2 value above 0.150, was considered sufficient for the power of the model. R 4.01 software (Austria, Vienna) was used with "rms" "Hmisc" packages.

RESULTS

Baseline characteristics of the low thrombus load and high thrombus load groups are summarized in Table 1. According to the thrombus burden, 192 enrolled patients were examined in two groups: 146 (76%) low thrombus burden [mean age= 59.2 ± 11.4 years, 94 (64.4%) men] and 46 (24%) high thrombus burden [mean age= 61.9 ± 10.7 years, 34 (73.9%) men]. There was no statistically significant difference between the groups in term of gender, hypertension, diabetes mellitus and smoking. The following were higher among patients with high thrombus burden; neutrophil (10.7 ± 3.9 vs. 8.5 ± 3.6; p= 0.016), C-reactive protein [CRP (13.2 ± 6.6 vs. 8.1 ± 3.7; p< 0.001)], baseline troponin I (8.4 ± 4.1 vs. 5.3 ± 3.2; p< 0.001), frontal QRSTa (99.1 ± 44.2 vs. 56.3 ± 39.8; p< 0.001).

Univariable analysis calculated that neutrophil, CRP, troponin I levels, frontal QRST angle were significantly associated with high thrombus burden (Table 2). Multivariable analysis revealed that CRP (OR= 1.185, 95% CI= 1.015-1.383, p= 0.032), admission troponin I (OR= 1.295, 95% CI= 1.091-1.536, p= 0.003), and admission frontal QRSTa (OR= 1.270, 95% CI= 1.140-1.410, p= 0.001) were the independent predictors of high thrombus burden (Table 2).

In ROC analysis, the cut-off value for frontal QRST angle to identify high thrombus burden with a sensitivity of 76% and

Table 1. Baseline characteristics of the patients[a,*]

Variable	Low thrombus burden (n= 146)	High thrombus burden (n= 46)	p value
Age (year)	59.2 ± 11.4	61.9 ± 10.7	0.359
Gender (male) [n (%)]	94 (64.4)	34 (73.9)	0.398
Hypertension [n (%)]	52 (35.6)	18 (39.1)	0.762
Diabetes mellitus [n (%)]	42 (28.8)	16 (34.8)	0.584
Smoking [n (%)]	60 (41.1)	22 (47.8)	0.569
Heart rate (per minute)	79 ± 18	84 ± 23	0.285
Creatinine (mg/dL)	0.91 ± 0.22	0.98 ± 0.25	0.275
WBC (μL)	11.8 ± 3.5	13.2 ± 3.8	0.131
Hemoglobin (g/dL)	13.7 ± 1.6	13.2 ± 1.2	0.194
Platelet count (10^3/μL)	227 ± 59	246 ± 65	0.183
Neutrophil count (10^9/L)	8.5 ± 3.6	10.7 ± 3.9	0.016
Lymphocyte count (10^9/L)	2.2 ± 1.2	2.4 ± 1.4	0.743
Total cholesterol (mg/dL)	192.4 ± 45.1	200 ± 51.6	0.511
LDL-C (mg/dL)	130.2 ± 33.5	138.1 ± 35.2	0.332
HDL-C (mg/dL)	42.8 ± 13.6	39.8 ± 10.6	0.426
Triglyceride (mg/dL)	156.8 ± 80.2	172.0 ± 89.1	0.482
CRP (mg/L)	8.1 ± 3.7	13.2 ± 6.6	**0.001**
Admission fQRST angle	56.3 ± 39.8	99.1 ± 44.2	**0.001**
Infarct related artery [n (%)]			0.487
LAD	64 (43.8)	24 (52.2)	
CX	28 (19.2)	4 (8.7)	
RCA	54 (37.0)	18 (39.1)	
Multivessel disease [n (%)]			0.568
1st vessel disease	82 (56.2)	20 (43.5)	
2nd vessel disease	44 (30.1)	18 (39.1)	
3rd vessel disease	20 (13.7)	8 (17.4)	
Syntax score	11.6 ± 7.3	13.5 ± 6.5	0.253
LVEF (%)	44.7 ± 6.8	42.5 ± 5.7	0.154
Baseline troponin I (ng/dL)	5.3 ± 3.2	8.4 ± 4.1	**0.001**

CRP: C-reactive protein, CX: Circumflex coronary artery, fQRST: Frontal QRS-T, HDL-C: High density lipoprotein-cholesterol, LAD: Left coronary descending artery, LDL-C: Low density lipoprotein-cholesterol, LVEF: Left ventricular ejection fraction, RCA: Right coronary artery, WBC: White blood cells.

[a] Data are presented as mean + SD or n (%).

* Statistically significant p values shown in boldface.

specificity of 74% was 76.5 in STEMI patients (Figure 2). The area under the curve was 0.76 (CI= 0.644-0.876, p< 0.001).

The correlations between thrombus burden and neutrophil, CRP, baseline troponin I and frontal QRST angle are shown in Table 3. Spearman nonparametric analysis demonstrated a moderate positive correlation of frontal QRST angle and high thrombus burden (r= 0.458, p< 0.001) and mild positive correlation of neutrophil and high thrombus burden (r= 0.260, p= 0.01) and mild-moderate correlation of CRP and high thrombus burden (r= 0.327, p< 0.001) and mild-moderate correlation of troponin I and high thrombus burden (r= 0.334, p< 0.001).

Balloon predilatation was carried out with 2 mm semicompliant balloons in the majority of the cases; 133 (91%) patients in low thrombus group vs. 40 (93%) patients in high thrombus group. Consequently, balloon size was not taken into consideration as also done in the previous studies.

DISCUSSION

The primary end point of the study was the evaluate the predictors of thrombus burden in STEMI. Our results indicate that there is significant correlation between acceptance frontal QRST angle on admission ECG and thrombus burden in STEMI. The other strong predictors of high thrombus burden are CRP and baseline troponin I. As far as we know its the first time in literature to define the usefulness of frontal QRST angle to estimate thrombus burden in STEMI. According to what is believed that the major mechanism for ACS is the tear of unstable atherosclerotic plaque which lead to thrombus formation and obstruction of the coronary artery. During the PCI in patient with ACS the high burden of the thrombus in culprit lesion raise serious difficulties in making a decision of how to manage and deal with lesion which can lead to increase the rate of stent thrombosis later. In STEMI cases with high thrombus burden treated with drug-eluting stents has been linked directly to the success of the procedure and can influence stent thrombosis leading to the increased 30-day mortality[4]. Distal microembolization of atherothrombotic particles causing microvascular obstruction result in no reflow phenomenon which is determinant of larger infarct size, lower ventricular function and increased mortality even after achieving normal epicardial flow. Moreover, STEMI cases with high thrombus burden treated by primary PCI had higher rate of no reflow incidence and distal embolization[20,21]. In the view of these results, the prediction of preprocedural high thrombus burden

Table 2. Univariate and multivariate regression analysis*

Variable	p	OR	95% CI	p	OR	95% CI
CRP	0.001	1.194	1.084-1.315	**0.032**	1.185	1.015-1.383
fQRST angle	0.001	1.340	1.150-1.540	**0.001**	1.270	1.140-1.410
Hemoglobin	0.246	0.834	0.614-1.133	0.966	1.012	0.586-1.746
LVEF	0.099	0.935	0.863-1.013	0.760	0.984	0.890-1.089
Neutrophil	0.02	1.161	1.024-1.316	0.167	1.220	0.920-1617
Platelet	0.182	1.006	0.997-1.014	0.911	1.001	0.988-1.014
Troponin I	0.001	1.251	1.102-1.420	**0.003**	1.295	1.091-1.536
WBC	0.137	1.000	1.000-1.000	0.993	1.000	1.000-1.000

CI: Confidence interval, CRP: C-reactive protein, fQRST: Frontal QRS-T, LVEF: Left ventricular ejection fraction, OR: Odds ratio, WBC: White blood cells.
* Statistically significant p values shown in boldface.

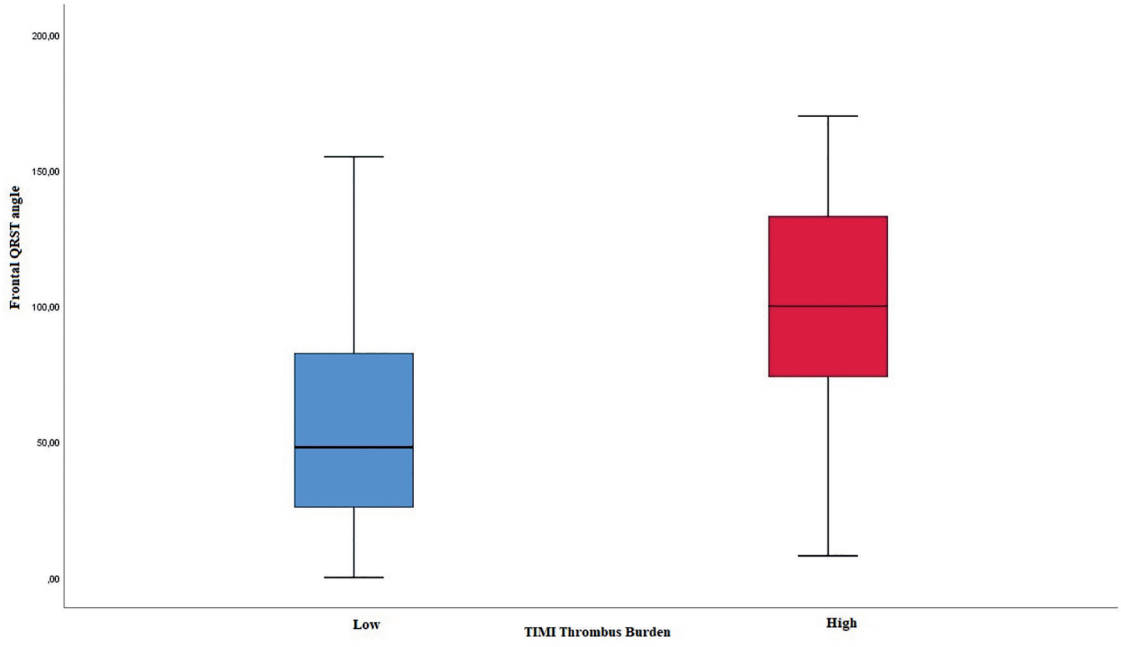

Figure 1. Box-plot for frontal QRST angle values according to thrombolysis in myocardial infarction (TIMI) thrombus burden.

Table 3. Correlations between thrombus burden and neutrophil, CRP, troponin I and fQRST angle

Variable	r	p value
Neutrophil	0.260	0.011
CRP	0.327	< 0.001
Baseline troponin I	0.334	< 0.001
fQRST angle	0.458	< 0.001

CRP: C-reactive protein, fQRST: Frontal QRST.

of culprit lesion in the IRA is especially substantial, for the purpose of better procedural accomplishment and related clinical consequences in patients with ACS. Estimation of intracoronary thrombus burden before the angiography could be used to assist physicians to implement prevention strategies for possible complications and their timely management during PCI. Therefore, several carefully conducted studies to date have tried to identify the predictors of high thrombus burden in patients with ACS.

The independent predictors of high thrombus burden were reported as total bilirubin levels, CRP to albumin ratio

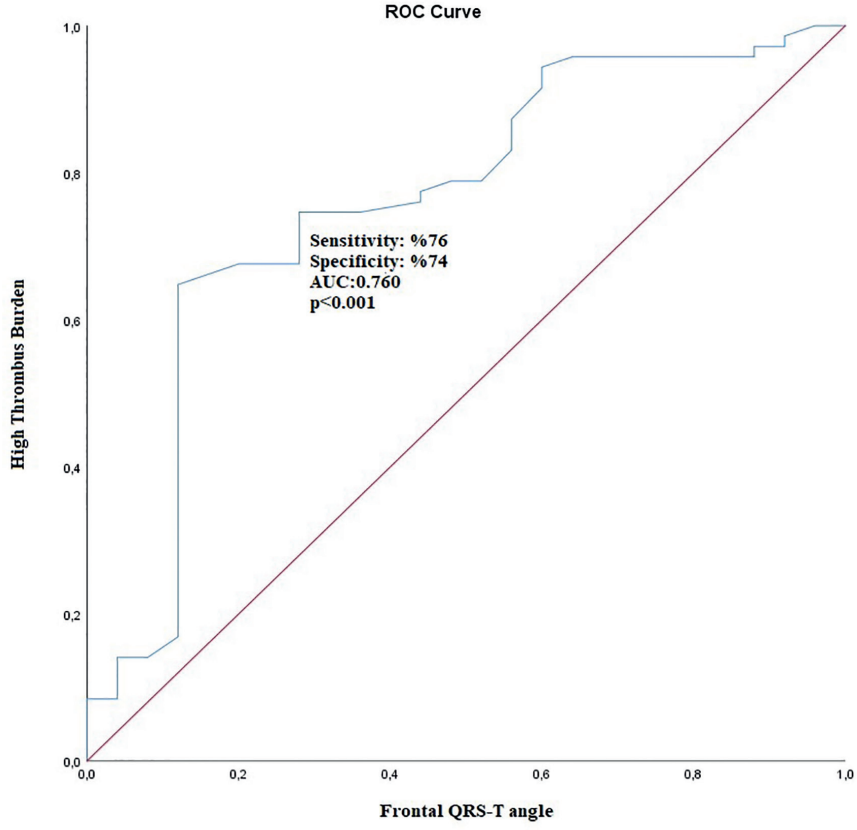

Figure 2. Receiver operating characteristics (ROC) curve analysis indicating the discriminative ability of frontal QRST angle.

(CAR), EAT and MHR previously[7,9,22,23]. In earlier research, Hamur et al., have suggested that total bilirubin levels were a strong predictor of high thrombus burden in STEMI patients[22]. Also, in another recent study, higher CRP levels, lower serum albumin levels, higher CAR, higher NLR, and baseline troponin I level were found to be independent predictors of high thrombus load in patients with ACS[23]. Increased EAT has been identified as an independent predictor of thrombus formation in patients with STEMI who treated by primary PCI within 24 hour after admission[9]. Arisoy et al. has reported that MHR and baseline troponin I were the independent predictors of high thrombus load in STEMI[7].

The absolute value of the difference between the frontal QRS axis and T axis on 12-lead surface ECG reflecting myocardial repolarization which is regarded as a new risk marker called frontal QRST. In ischemic conditions widening of frontal QRST angle occurs, as the inhomogeneous areas of the myocardium cause depolarization and repolarization heterogeneity. Normally, the directions of the myocardial depolarization axis and repolarization axis are in the intimate orientation. Therefore, frontal QRST angle is most of the time estimated to be a narrow-angle (< 45°)[24]. In former studies, the frontal QRST angle has been shown to predict the risk of cardiovascular mortality, sudden cardiac death, heart failure (either reduced ejection fraction or preserved ejection fraction) [12,25,26]. In a former study, Raposeiras-Roubin et al., 2014, showed that frontal QRST angle > 90° is an independent predictor of long-term mortality in acute STEMI patients[27]. The relationship between frontal QRST angle and the severity of CAD has been studied previously preferentially depending on the number of vessels or localization of lesions. Colluoglu et al., has shown that patients with baseline frontal QRST angle ≥ 95.6° had a sufficiently great higher frequency of three-vessel disease and more frequent proximal vessel disease compared to patients with baseline frontal QRST angle < 95.6° in STEMI patient[14]. It was also found that baseline and postprocedural frontal QRST angle were associated with in-hospital mortality in this study. Dogan et al., has shown that frontal QRST angle is an independent predictor of coronary atherosclerotic burden in STEMI patients[15].

Prothrombotic and pro-inflammatory states are two closely related processes which shares similar biochemical mediators. So, we can propose that increased coronary thrombus burden can be implied as increased pro-inflammatory state[4]. These results can be correlated with the increased content of inflammatory cells in the circulation. Similarly, we found that, there is a correlation between thrombus burden and inflammatory markers including CRP and neutrophil count which may support its role in systemic inflammation. Niccoli et al., has recently shown the relationship between high thrombus burden and CRP levels[28]. In our study, we have also found that increased CRP levels was predictor of high thrombus burden. Baseline frontal QRST angle was also increased in these patients, and after multivariate analysis, this finding still persisted.

The diabetes mellitus risk factor, even expected to have a high thrombus burden, was found to have no significance in our study. In recent similar studies, diabetes mellitus was not observed as significant predictor, in line with our results[7,8,23]. A conceivable explanation is that due to the nature of the regression model, some important variables may not be included. Moreover, even if the probable sampling method was used, it may not be possible for some variables found in other studies to reach the limit of significance in our study because of the insufficient sample size.

LIMITATIONS

First limitation of this study is the relatively small number of patients who were enrolled in a single center. Additionally, the dynamicity of frontal QRST which allow changes in measurements daily may also interfere with the results. As a result, single frontal QRST angle measurement may be unable to show relationship between frontal QRST angle and thrombus burden in a longer period. Besides, measurement of spatial QRST angle may be better than frontal planar QRST angle if we consider protection from cardiac risks and we did not measure spatial QRST angle values. Visual assessment of the thrombus burden may be somewhat subjective and it would have been better to use a quantitative method. Large-scale prospective studies should be carried out to better define the association between frontal QRST angle, thrombus burden, pathophysiological mechanisms, and clinical outcomes.

CONCLUSION

Admission higher frontal QRST angle may predicts high thrombus burden independently from CRP and troponin I in patients with STEMI. In addition, high thrombus burden was also positively correlated with inflammation parameters such as CRP.

Ethics Committee Approval: This study was approved by Samsun Training and Research Hospital Non-Invasive Clinical Research Ethics Committee (2021/1/5, Date: 13.01.2021).

Informed Consent: Informed consent was obtained.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept/Design - GE, UA; Analysis/Interpretation - UA, MY; Data Collection - GE, DK; Writing - GE, UA; Critical Revision - ÖÇ, EA; Final Approval - UA; Statistical Analysis - AK; Overall Responsibility - GE.

Conflict of Interest: The authors have no conflicts of interest to declare.

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