ORIGINAL ARTICLE

Relationship between fragmented QRS complexes in leads V4-V6 and left ventricular apical thrombus formation in patients presenting with first acute anterior myocardial infarction

Birincil ön duvar ST yükselmeli miyokart enfarktüslü hastalarda V4-V6 derivasyonlardaki parçalanmış QRS ile sol ventrikül apeksi trombüsü arasındaki ilişki

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ABSTRACT

Objective: The present study was an investigation of the relationship between fragmented QRS (fQRS) and left ventricular apical thrombus (LVAT) in patients presenting with first acute anterior myocardial infarction (MI).

Methods: Consecutive 148 patients (mean age: 60.1±10.1 years; male: 75.6%) with first acute anterior MI who underwent primary percutaneous coronary intervention (PCI) were included. Study population was divided into 2 groups based on presence of LVAT. fQRS was defined as presence of various RSR' patterns, which included additional R wave or notching of R wave or S wave, and presence of more than 1 R in 2 contiguous leads corresponding to major coronary artery territory on 12-lead electrocardiogram. Patients with bundle branch block were excluded from the study.

Results: Of these, 32 (21.6%) had LVAT. Patients with LVAT had higher prevalence of fQRS (53.1% *vs.* 22.4%; p<0.001) and lower rate of successful PCI (75% *vs.* 94%; p=0.002) compared with patients without LVAT. More patients in LVAT group had left ventricular ejection fraction of <30% (87.5% vs 65.5%; p=0.010). Groups were similar with respect to other baseline characteristics (p>0.05 for all). Presence of fQRS was independent predictor of LVAT (odds ratio [OR], 2.795; 95% confidence interval [CI], 1.058–7.396) in multivariable logistic regression analysis.

Conclusion: Presence of fQRS in leads V4-V6 is independently associated with LVAT in patients presenting with first acute anterior MI.

ÖZET

Amaç: Bu çalışmada, ilk akut ön duvar miyokart enfarktüsü ile başvuran hastalarda sol ventrikül apeksi trombüsü (LVAT) ile V4-V6 derivasyonlarında parçalanmış QRS (fQRS) varlığı arasında ilişki olup olmadığı araştırıldı.

Yöntemler: İlk kez ön duvar miyokart enfarktüsü geçiren ve primer perkütan koroner girişim uygulanan ardışık 148 hasta (ortalama yaş: 60.1±10.1, erkek: %75.6) çalışmaya dahil edildi. Çalışma popülasyonu LVAT olup olmamasına göre iki gruba ayrıldı. FQRS, ek bir R dalgası veya R dalgası veya S dalgasının çentiklenmesi veya 12 derivasyonlu EKG üzerinde büyük koroner arter bölgesine karşılık gelen iki bitişik derivasyonda birden fazla R bulunması gibi çeşitli RSR paternlerinin varlığı olarak tanımlandı. Dal bloğuna sahip hastalar çalışma dışı bırakıldı.

Bulgular: Hastaların 32'sinde (%21.6) LVAT vardı. Sol ventrikül apeksi trombüsü olan hastalar LVAT'siz hastalarla karşılaştırıldığında daha yüksek fQRS prevalansı (%53.1 ve %22.4, p<0.001) ve daha düşük başarılı perkütan koroner girişim (%75 ve %94, p=0.002) oranları ile karşılaşıldı. Sol ventrikül apeksi trombüsü grubunda daha fazla hastada sol ventrikül ejeksiyon fraksiyonu <%30 (%87.5, %65.5, p=0.010) bulundu. Gruplar diğer temel özelliklere göre benzerdi (hepsi için p>0.05). Çok değişkenli lojistik regresyon analizinde fQRS'nin varlığı LVAT'nin bağımsız bir öngördürücüsü olmuştur (odds oranı 2.795, %95 güven aralığı 1.058–7.396).

Sonuç: Bu çalışma ilk kez geçirilen ön duvar ST yükselmeli miyokart enfarktüslü hastalarda V4-V6 derivasyonlarında izlenen fQRS'in LVAT ile bağımsız ilişkili olduğunu göstermiştir.

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Left ventricular apical thrombus (LVAT) formation is a well-known complication of acute anterior myocardial infarction (MI). Incidence of LVAT has decreased in the early revascularization era compared with the prethrombolytic era, but it still occurs in range of 2.9% to 15%.[1] One of the most feared complications is occurrence of embolic events in patients with LVAT.[2,3] Systemic anticoagulation with oral anticoagulant drugs in the presence of documented LVAT reduces risk of systemic embolization3. However, there are limited data supporting routine prophylactic use of oral anticoagulation drugs following first acute anterior MI complicated by left ventricular (LV) dysfunction.

Fragmented QRS (fQRS) complexes are novel electrocardiographic (ECG) signals which reflect altered ventricular conduction delays around regions of myocardial scar.[4,5] fQRS has been shown to be associated with major adverse cardiac events, LV dysfunction, and total mortality in patients with coronary artery disease (CAD).[6–11]

Aim of the present study was to investigate the relationship between fQRS in leads V4-V6 and LVAT formation in patients with first acute anterior MI.

METHODS

Study population

Retrospective, single-center study was performed at Diyarbakır Gazi Yaşargil Education and Research Hospital, a tertiary referral hospital with 24/7 primary percutaneous coronary intervention (PCI) service. Included were all 356 consecutive patients admitted to cardiac intensive care unit with diagnosis of first acute anterior MI between October 2011 and November 2015. Patients were required to meet the following criteria: (1) age <70 years, (2) chest pain lasting ≥30 minutes, (3) more than 2 mm ST segment elevation in at least consecutive anterior precordial leads of ECG, (4) initial echocardiogram performed within 14 days following admission. Patients were excluded if they did not have transthoracic echocardiogram within 14 days of admission or had poor echocardiographic window (n=67). In addition, patients were excluded if they had preserved left ventricular ejection fraction (LVEF) or no anterior wall motion akinesia or dyskinesia (n=82), and 32 patients who were managed conservatively or with thrombolytic therapy were also excluded. An additional 27 patients were excluded due to known connective tissue disease, previous history of MI, advanced liver or kidney disease, known infectious disease, or malignancy. Of the 356 patients screened, 148 (42%) patients met our prespecified inclusion criteria.

Abbreviations:

ACS	*Acute coronary syndrome*
CAD	*Coronary artery disease*
CI	*Confidence interval*
ECG	*Electrocardiogram*
FQRS	*Fragmented QRS*
LV	*Left ventricular*
LVAT	*Left ventricular apical thrombus*
LVEF	*Left ventricular ejection fraction*
MI	*Myocardial infarction*
OR	*Odds ratio*
PCI	*Percutaneous coronary intervention*

Patients were treated according to American College of Cardiology Foundation/American Heart Association Guidelines for the Management of Patients With ST elevation MI, which was defined by the following criteria: typical chest pain, ST-segment elevation of 1 mm at least in 2 contiguous anterior electrocardiographic leads or new onset left bundle branch block, and transient elevation of serum cardiac biomarkers.[12,13] All patients were treated in the emergency department with dual antiplatelet therapy consisting of aspirin (loading dose of 300 mg followed by 100 mg/day) and clopidogrel (loading dose of 300-600/mg followed by 75 mg/day), as well as unfractionated or low molecular weight heparin. Primary PCI was performed in patients with symptoms of 12 hours in duration as well as in patients with symptoms lasting 12 to 24 hours in duration, if symptoms continued to persist at time of admission. Post-PCI anticoagulant treatment was deferred unless echocardiographic examination demonstrated LVAT formation. Treatment with low molecular weight heparin was

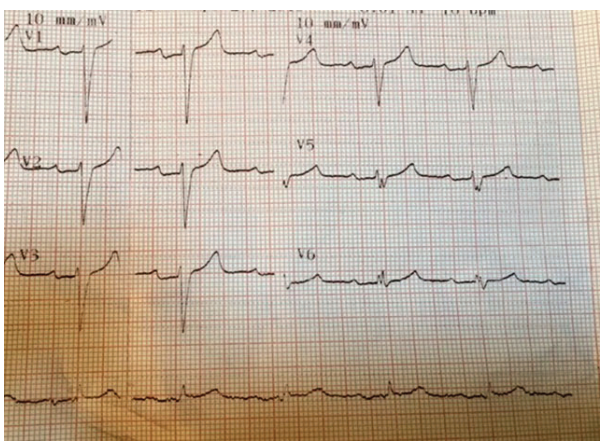

Figure 1. Fragmented QRS in leads V5-V6.

initiated and supplemented with warfarin for patients with echocardiographic evidence of LVAT, aiming for target international normalized ratio of 2 to 3 for 3 to 6 months.

Echocardiography

Baseline comprehensive 2-dimensional transthoracic echocardiography, including M-mode and Doppler echocardiography, was performed by single experienced cardiologist blinded to angiographic data using Philips IE-33 (Philips Healthcare, Inc., Andover, MA, USA) instrument with S5-1 transducer. Second harmonic imaging was used to optimize endocardial visualization. Parasternal long and short axis, apical, and 2- and 4-chamber views were obtained using standard transducer positions. Special consideration was given to apical and low parasternal echocardiographic windows. Echocardiograms were interpreted independently by 2 expert investigators. LVAT was defined as echo-dense mass adjacent to abnormally contracting (akinetic or dyskinetic) myocardial segment. Measurements were performed according to guidelines of American Society of Echocardiography. [14] LVEF was calculated using Simpson biplane formula.

Electrocardiography

ECG was performed initially and during intensive

Table 1. Baseline characteristics of the study groups

	Left ventricular apical thrombus		*p*
	No (n=116)	Yes (n=32)	
Age (year)	59.8±10.5	61.2±11.2	0.201
Male gender	91 (78.4)	21 (65.6)	0.136
Diabetes mellitus	44 (38)	14 (44)	0.559
Hypertension	34 (30)	10 (31)	0.832
Hyperlipidemia	37 (32)	13 (40)	0.366
Smoking	71 (61)	23 (72)	0.274
Family history of CAD	17 (15)	9 (26)	0.105
Body mass index (kg/m^2)	29.8±3.2	26.2±2.8	0.150
Glomerular filtration rate (mL/minutes)	86±18	83±13	0.242
Time from symptoms to treatment (hours)	3.1±1.3	2.9±1.5	0.424
Prehospital anticoagulation	99 (85)	27 (84)	0.906
Unfractionated heparin	86 (74)	24 (75)	0.921
Low molecular weight heparin	30 (26)	8 (25)	0.921
Successful PCI	109 (94)	24 (75)	0.002
Admission troponin levels (ng/ml)	2.7±1.6	2.9±1.4	0.882
Peak troponin levels (ng/ml)	11.2±4.3	12.8±4.8	0.105
White blood cell (×10^3/mm^3)	11.7±2.9	12.6±1.2	0.021
Neutrophil (×10^3/mm^3)	7.7±2.0	8.3±1.3	0.015
Lymphocyte (×10^3/mm^3)	2.6±0.8	2.6±0.4	0.028
Neutrophil/lymphocyte ratio	2.6±0.2	2.9±0.6	0.043
Red cell distribution width (%)	13.4±0.4	13.6±0.5	0.048
Hemoglobin (g/dL)	14.3±1.5	14.2±1.2	0.702
Mean platelet volume (%)	9.4±0.4	9.7±0.7	0.004
Platelet count (×10^3/mm^3)	251±44	261±35	0.122
Fragmented QRS	26 (22.4)	17 (53.1)	<0.001

Continuous variables are presented as mean (standard deviation) and categorical variables as number (percentage). PCI: Percutaneous coronary intervention. CAD: Coronary artery disease.

care follow-up at 12th, 24th, and 48th hour post PCI. ECG from 48th hour was interpreted for fragmentation. Twelve-lead ECG results (Cardiofax M ECG-1350K, filter range 0.5 Hz to 150 Hz, AC filter 60 Hz, 25 mm/second, 10 mm/mV; Nihon-Kohden Corp., Tokyo, Japan) were analyzed by 2 independent cardiologists who were blinded to patient data. fQRS was defined as presence of different RSR' patterns (QRS duration <120 milliseconds), which included additional R wave (R' prime) or notching of R wave or S wave, or presence of more than 1 R' prime without typical bundle branch block in 2 contiguous leads corresponding to major coronary artery territory (Figure 1).[15,16] All ECGs were interpreted using standard criteria.

Statistical analysis

Continuous variables were presented as mean (SD) and categorical variables as number (percentage). Distribution of continuous variables across study groups was tested with Kolmogorov–Smirnov test. Continuous variables were compared using Student's t-test. Categorical data were compared using chi-square or Fisher's exact tests, as needed. Univariate and multivariate logistic regression analyses were conducted to assess association of fQRS and LVAT. In stepwise multivariate regression analysis (backward, Wald), effect size was adjusted for all variables with univariate significance level of <0.2. Adjusted odds ratios (OR) and 95% confidence intervals (CIs) were presented. Two-tailed p value of <0.05 was considered statistically significant. All statistical analyses were performed using IBM SPSS software (IBM SPSS Statistics for Windows, Version 21.0; IBM Corp., Armonk, NY, USA).

RESULTS

Total of 148 patients were included in the study (mean age: 60.1±10.1 years; male: 75.6%). Of these, 32 (21.6%) had LVAT (mean age: 61.2±11.2 years; 21 men, 11 women). Baseline characteristics of the study groups are provided in Table 1. Patients with LVAT had higher prevalence of fQRS (53.1% vs 22.4%; p<0.001) and lower rate of successful PCI (75% vs 94%; p=0.002), compared with patients without LVAT. White blood cell, neutrophil and lymphocyte count, neutrophil/lymphocyte ratio, red cell distribution width, and mean platelet volume were significantly higher in patients with LVAT (p<0.05 for all; Table 1). Groups were similar with respect to other baseline characteristics shown in Table 1 (p>0.05 for all).

In patients with LVAT, LV dimensions were greater than in patients without LVAT (p<0.05 for all; Table 2). More patients in LVAT group had LVEF of <30% (87.5% vs 65.5%; p=0.010) (Table 2).

Presence of fQRS on 12-lead ECG was an independent predictor of LVAT (OR, 2.795; 95% CI, 1.058–7.396) in multivariable logistic regression analysis (Table 3). Other independent predictors of LVAT were mean platelet volume (OR, 2.737; 95% CI, 1.258–5.955) and LVEF of <30% (OR, 4.715; 95% CI, 1.347–16.513). Unsuccessful PCI had borderline significance for presence of LVAT in multivariate analysis (OR, 3.605; 95% CI, 0.991–13.113).

DISCUSSION

In this study, among patients presenting with first acute anterior MI, patients with LVAT had significant-

Table 2. Echocardiographic characteristics of the study groups

	Left ventricular apical thrombus		*p*
	No (n=116)	Yes (n=32)	
Left ventricular end-diastolic diameter (mm)	4.8±0.45	5.1±0.50	0.031
Left ventricular end-systolic diameter (mm)	3.3±0.50	3.5±0.30	0.010
Interventricular septal wall thickness (mm)	1.0±0.22	1.1±0.15	0.204
Posterior wall thickness (mm)	0.9±0.18	0.9±0.16	0.302
Left atrial diameter (mm)	3.7±0.45	3.6±0.27	0.105
Left ventricular ejection fraction ≤30	76 (65.5)	28 (87.5)	0.010

Continuous variables are presented as mean (standard deviation) and categorical variables as number (percentage).

Table 3. Stepwise multivariate analysis for the presence of left ventricular apical thrombus

	Odds Ratio	95% CI	*p*
STEP 1			
Age	0.989	0.947–1.033	0.622
Gender	0.444	0.149–1.323	0.145
Neutrophil/lymphocyte ratio	2.342	0.332–16.548	0.393
Red cell distribution width	1.927	0.761–4.880	0.166
Mean platelet volume	2.385	1.062–5.356	0.035
Left ventricular ejection fraction ≤30	4.876	1.324–17.961	0.017
Unsuccessful percutaneous coronary intervention	5.023	1.260–20.035	0.022
Fragmented QRS	2.033	0.721–5.733	0.180
STEP 2			
Gender	0.483	0.171–1.361	0.168
Neutrophil/lymphocyte ratio	2.437	0.351–16.923	0.368
Red cell distribution width	1.882	0.747–4.741	0.180
Mean platelet volume	2.407	1.072–5.404	0.033
Left ventricular ejection fraction ≤30	4.748	1.307–17.251	0.018
Unsuccessful percutaneous coronary intervention	5.042	1.265–20.092	0.022
Fragmented QRS	2.053	0.728–5.783	0.174
STEP 3			
Gender	0.498	0.178–1.392	0.184
Red cell distribution width	1.939	0.775–4.854	0.157
Mean platelet volume	2.471	1.105–5.524	0.028
Left ventricular ejection fraction ≤30	5.202	1.435–18.862	0.012
Unsuccessful percutaneous coronary intervention	4.639	1.206–17.838	0.026
Fragmented QRS	2.217	0.802–6.128	0.125
STEP 4			
Red cell distribution width	1.769	0.715–4.377	0.217
Mean platelet volume	2.670	1.204–5.918	0.016
Left ventricular ejection fraction ≤30	4.935	1.393–17.482	0.013
Unsuccessful percutaneous coronary intervention	3.842	1.040–14.196	0.044
Fragmented QRS	2.449	0.901–6.658	0.079
STEP 5			
Mean platelet volume	2.737	1.258–5.955	0.011
Left ventricular ejection fraction ≤30	4.715	1.347–16.513	0.015
Unsuccessful percutaneous coronary intervention	3.605	0.991–13.113	0.052
Fragmented QRS	2.795	1.058–7.396	0.038

CI: Confidence interval.

ly higher prevalence of fQRS on ECG compared with patients without LVAT. Moreover, fQRS was independently associated with LVAT in patients presenting with first acute anterior MI.

LV thrombus formation is a major complication after acute anterior MI. Previous studies have reported that incidence of LV thrombus is 27% to 46% in patients with acute anterior MI who have apical akinesia or dyskinesia.[17–19] However, in another recent study, prevalence of LV thrombus was much lower than pre-

viously reported, possibly as result of improvement in management of acute MI with widespread use of reperfusion therapies and appropriate antiplatelet therapies.[20] Predictors of LVAT development after acute MI must be well known; incidence of embolic stroke in patients with documented LV thrombi on echocardiography increased 5.5 times after anterior acute MI.[3] In previous reports, some clinical and echocardiographic parameters, such as large infarct size, anterior location, low LVEF, delayed reperfusion, and low Thrombolysis In Myocardial Infarction (TIMI) flow grade, have been well established for LVAT development.[21,22] However, relationship of electrocardiographic characteristics and LVAT development has not been well studied.

fQRS was defined by unexpected deviations in QRS morphology. It is a sign of structural (fibrosis) and functional (ischemia, inflammation) myocardial abnormality.[23] Specific cause of this fractionation on surface ECG and determinations of this phenomenon are not completely understood yet; myocardial fibrosis and/or ischemia is generally accepted as being responsible for fQRS formation through altered homogeneity of myocardial electrical activity.[24,25] First of all, it was found to be associated with increased cardiac mortality and morbidity in patients with CAD,[7] acute coronary syndromes (ACS),[8,23] and ischemic and nonischemic cardiomyopathy.[26,27] Secondly, fQRS was found to be associated with ventricular arrhythmias in various conditions, such as ischemic and nonischemic cardiomyopathy,[27] hypertrophic cardiomyopathy,[28] Brugada syndrome,[29] acquired long QT syndrome,[30] and arrhythmogenic right ventricular dysplasia.[31,32] Das et al. reported that in patients with CAD, all-cause mortality and cardiac events rates were significantly higher in fQRS group.[7] Research of Ari et al. in a cohort study suggests that in patients with ST-elevation MI, presence of fQRS on ECG performed at 48th hour was significant predictor of major cardiac events.[23] Presence or absence of fQRS on admission to emergency department has been demonstrated in some clinical trials to be related to prognosis and irreversible ischemia in patients with ACS.[15,23,33] These findings not only establish relationship between fQRS and fibrosis, but also myocardial ischemia in patients with ACS. Some recent studies have demonstrated role of inflammation in fQRS formation.[34,35] Systemic inflammation has an important role in occurrence of thrombosis and conduction abnormalities, and this was attributed to myocardial inflammation, focal fibrosis, or ischemia in the conduction system. In studies that used gadolinium to delay enhancement of cardiac magnetic resonance imaging in order to determine myocardial structure, fQRS was found to be related to extensive myocardial scar.[36,37]

In the setting of acute anterior MI, ischemia and/or inflammation cause anteroapical aneurysm, akinesia, or dyskinesia that results in reduced LVEF. LV tries to adapt to new condition and becomes vulnerable to thrombus formation.

Limitations

This study has several limitations. Primary limitation is its retrospective nature. We didn't evaluate power analysis. Small sample size is another limitation. We didn't evaluate patients with magnetic resonance imaging or myocardial perfusion scintigraphy to detect myocardial abnormalities attributable to fQRS. Finally, we did not study relationship of fQRS and LVAT-related complications.

Conclusion

Presence of fQRS in leads V4-V6 is independently associated with LVAT in patients presenting with first acute anterior MI. It may help in predicting LVAT formation after acute MI.

Conflict-of-interest issues regarding the authorship or article: None declared

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