INTRODUCTION

Myocardial bridge (MB) is defined as the course of a part of the coronary artery within the myocardium. It is most common in the left anterior descending coronary artery. Although the incidence of MB has been reported as 0.5–16% in angiographic studies, it has been shown to reach up to 80% in autopsy studies. MB is known as a benign condition, but some adverse cardiac events have been reported associated with MB. Reported cardiac events have been shown to be associated with the effect of MB on coronary blood flow. Weakened myocardial blood flow in MB can cause mortal cardiac arrhythmias by impairing cardiac repolarization.1
Considering all progress in diagnostic tests and treatment in cardiology, electrocardiogram (ECG) continues to be the most important device in detecting cardiovascular diseases. Detailed assessment of the surface ECG can provide the physician a lot of important information. Cardiac repolarization abnormalities are one of them. Cardiac repolarization can traditionally be measured with QT interval, QT dispersion and corrected QT interval (QTc) from 12-lead surface ECG. On the other hands, frontal QRS-T angle (fQRSTa) is a novel indicator of repolarization and depolarization heterogeneity. The relationship of the fQRSTa in many cardiovascular diseases has been investigated, and its importance in predicting cardiovascular arrhythmias has been demonstrated. Different from the QT measurements, fQRSTa may be measured easily from surface ECG.

Previous studies demonstrated the effect of the MB on QT intervals. However, to our knowledge, no study evaluated the effect of MB on fQRSTa. In this study, we aimed to evaluate the relationship between MB and fQRSTa.

2 | MATERIAL AND METHOD

2.1 | Study design

In this study, 89 patients with isolated MB with over 70% stenosis in the coronary artery were included. Also, 91 consecutive patients who performed coronary angiography and found as normal coronary arteries were selected as control group in the same period. Coronary angiography had been performed to all patients due to a positive treadmill exercise test or myocardial scintigraphy. Patients with pulmonary hypertension, left ventricular systolic dysfunction, cardiomyopathy, severe valvular heart disease, atherosclerotic coronary artery disease (CAD), acute coronary syndrome, electrolyte imbalance, kidney failure, abnormal ECG and using antiarrhythmic and antidepressant drugs were excluded in this study. Local ethic committee approved the design of the study and informed consent was obtained from all patients.

2.2 | Coronary angiography

Coronary angiography was evaluated using radial approach with the Judkins technique. Images were recorded on a digital angiographic system (Phillips Multidagnostic IV Netherlands) at a rate of 15 frames / second. Quantitative measurements of coronary arteries were made on a digital angiographic system in Figure 1 (Phillips Multidagnostic IV Netherlands).

2.3 | Electrocardiography

After a 10-min rest in the supine position, a 12-lead surface ECG at 25mm/second and 10 mV/cm was recorded in all patients (Nihon Kohden). QRS axis, T axis and QT interval were taken from the automatic report of surface ECG. QTc interval was corrected with Bazett formula (QTc = QT/√RR). The fQRSTa was defined as the absolute difference between myocardial depolarization (QRS axis) and repolarization (T axis) (fQRSTa = |QRS axis– T axis|). An illustration of calculation of fQRSTa angle from the surface ECG is shown in Figure 2.
Echocardiographic examinations were performed with the Vivid 5 ultrasound imaging system (GE Medical Systems) in the left lateral decubitus position. All assessments were calculated in accordance with the standards published by the American Society of Echocardiography.

### 2.5 | Statistical analysis

SPSS 20.0 software program (SPSS Inc.) was used for the statistical analyses. The distribution of data was tested with Kolmogorov–Smirnov. Continuous data was expressed as mean ± standard deviation or median (interquartile range 25–75). Categorical data was expressed as numbers and percentages. Student t test and Mann–Whitney U test were used to compare continuous variables. Correlation analysis was performed to determine the correlation of fQRSTa with length of MB. Linear regression analysis was calculated to define significant predictors of fQRSTa. A p < 0.05 value was accepted statistically important.

### 3 | RESULTS

In this study was added that a total of 89 patients with MB and 91 patients with normal coronary artery. Table 1 presents the comparison of characteristics. There was no important difference between two groups in terms of characteristics and conventional echocardiographic measurements (Table 1).

The mean length of MB was 21.5 ± 5.6 mm. Electrocardiographic values of the study subjects are demonstrated in Table 2. Patients with MB had importantly increased fQRSTa (27 [20–41] vs. 23 [12–37], p = 0.007), QT (366 ± 2 vs. 358 ± 3, p = 0.030) and QTc (411 ± 2 0 vs. 407 ± 12, p = 0.007) compared to the control group. In correlation analysis, fQRSTa was positively correlated with MB length (r = 0.411, p < 0.001). Multivariate linear regression analysis was performed to determine the significant predictor of fQRSTa. It was shown that MB length was independently related with the fQRSTa (β = 0.247, p = 0.041) (Table 3).

### 4 | DISCUSSION

In the presented study, we showed that the fQRSTa, QTc and QT were importantly increased in patients with MB compared with the control group. In addition, we revealed that fQRSTa was the just considerable predictor of MB length. To our knowledge, ours is the first research indicating relation with fQRSTa and MB.

The incidence of MB has been reported as 0.5–16% in angiographic studies. Similarly, the incidence of muscular bridge was 2.3% in our institution. It is a congenital anomaly of the coronary arteries and often tend to be asymptomatic. However, it can cause chest pain, sudden cardiac death, myocardial infarction and life-threatening arrhythmias. Although the main angiographic finding of MB is narrowing of the coronary arteries during systole, it has been shown to persist during the diastole period. Therefore, severe ischemia may be occurred in patients with MB. Increased heart rate due to different reasons can shorten the diastole time and increase the cardiac oxygen demand. Therefore, high heart rate can be expected to tendency to ischemia in patients with MB. Another possible cause of ischemia in these patients is coronary vasospasm. Studies have shown that coronary vasospasm is present in the proximal segment of MB. In addition, some histopathological studies performed after sudden death revealed interstitial edema and myocardial fibrosis in the segment with MB. It has been suggested that
TABLE 1 Basal characteristics of study populations

| Variables                      | Myocardial bridge group n = 89 | Control group n = 91 | p     |
|--------------------------------|--------------------------------|----------------------|-------|
| Age (years)                    | 49.2 ± 6.8                     | 47.8 ± 9.8           | 0.068 |
| Male gender (%)                | 74 (83.1)                      | 79 (86.8)            | 0.491 |
| BMI (kg/m²)                    | 25.8 ± 3.2                     | 25.4 ± 4.1           | 0.809 |
| SBP (mmHg)                     | 116.1 ± 2.2                    | 119.2 ± 11.1         | 0.085 |
| DBP (mmHg)                     | 74.2 ± 9.4                     | 74.0 ± 9.6           | 0.887 |
| Heart rate (per/ min)          | 74.0 ± 10.1                    | 76.6 ± 12.8          | 0.136 |
| DM (%)                         | 12 (14)                        | 18 (20)              | 0.257 |
| HT (%)                         | 28 (32)                        | 30 (33)              | 0.829 |
| Lipidemia (%)                  | 26 (29)                        | 20 (22)              | 0.348 |
| Smoking (%)                    | 52 (58)                        | 47 (52)              | 0.361 |
| Hemoglobin (g/dl)              | 13.5 ± 2.2                     | 13.9 ± 1.6           | 0.098 |
| Potassium (mEq/L)              | 4.1 ± 0.6                      | 4.1 ± 0.5            | 0.991 |
| Sodium (mEq/L)                 | 137.3 ± 2.9                    | 137.9 ± 3.0          | 0.256 |
| Total cholesterol (mg/dl)      | 182(159–211)                   | 172(150–200)         | 0.220 |
| LDL cholesterol (mg/dl)        | 101.1 ± 25.7                   | 101.2 ± 22.7         | 0.981 |
| Triglyceride (mg/dl)           | 124 (86–178)                   | 127(89–173)          | 0.917 |
| HDL cholesterol (mg/dl)        | 38 (33–46)                     | 40 (35–45)           | 0.629 |
| LVEF (%)                       | 56.9 ± 6.6                     | 56.9 ± 6.5           | 0.981 |
| LVDD (mm)                      | 46.3 ± 3.5                     | 46.8 ± 3.9           | 0.633 |
| LVSD (mm)                      | 31.6 ± 3.5                     | 31.1 ± 3.0           | 0.308 |
| IVS (mm)                       | 8.955 ± 1.9                    | 9.066 ± 1.9          | 0.703 |
| PWD (mm)                       | 8.2 ± 1.8                      | 8.3 ± 1.9            | 0.639 |
| LA (mm)                        | 33.8 ± 2.9                     | 34.3 ± 3.2           | 0.357 |
| E/A                            | 1.4 ± 0.5                      | 1.3 ± 0.5            | 0.866 |
| MB length (mm)                 | 21.5 ± 5.6                     | -                    | -     |

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; DM, diabetes mellitus; HDL, high density lipoprotein; HT, hypertension; IVS, interventricular septum thickness; LA, left atrium; LDL, low density lipoprotein; LVDD, left ventricular end diastolic dimension; LVEF, left ventricular ejection fraction; LVSD, left ventricular end systolic dimension; MB, myocardial bridge; PWD, posterior wall thickness; SBP, systolic blood pressure.

myocardial fibrosis is associated with increased arrhythmic events in MB patients. As the length of the segment with MB increases, it can be proposed that the area of myocardial fibrosis also increases. Increased fibrosis disrupts the homogeneity of the myocardium and can lead to repolarization disorder.

Cardiac repolarization is traditionally estimated according to the QT and QTc interval. Previous studies demonstrated the association of MB with QT and QTc interval measurement. In this study, we also showed that QTc and QT interval were significantly increased in patients with MB.

Although QT intervals measurements is most commonly used markers to assess myocardial repolarization, the measurements of these markers are not easy and may require additional tools such as caliper, magnifying glass and software programs. Also, these parameters have poor reproducibility. Therefore, the investigators focused on novel indicators which may be obtained easily from the surface ECG. The fQRSTa is a novel indicator of myocardial repolarization and depolarization heterogeneity. It is determined as the absolute angle difference between the directions of ventricular depolarization and repolarization. The fQRSTa has been found to be more repeatable, less responsive to interference than QT interval measurements. In this study, we also found that only fQRSTa was higher in patients with MB compared to control group among all repolarization parameters investigated in this study. In accordance with this finding, previous studies also indicated that fQRSTa was more sensitive than other repolarization parameters for evaluating myocardial repolarization.

Myocardial repolarization abnormalities and ischemia increase the fQRSTa. Last research have showed the usefulness of fQRSTa in various patient populations. Increased fQRSTa has also showed to be related with an increased risk of arrhythmic event. In the present study, we also assessed the fQRSTa in patients with MB. We revealed that fQRSTa increased importantly in patients with MB according to control group. As far as we know, this is the first study showing its effect on the fQRSTa angle in patients with MB. We think that the possible mechanisms of fQRSTa increase in these patients may be due to (I) circulatory abnormalities in the area perfused by the bridged artery, (II) myocardial ischemia, and (III) increased fibrosis. These conditions may disrupt the homogeneity of myocardium and lead to myocardial repolarization disorder.

Previous studies demonstrated that higher fQRSTa values (generally >90°) was associated with poor prognosis. In our study, fQRSTa was mildly increased (27 [20–41]) in patients with MB and no patients developed fatal arrhythmia during the study period. Because the increase in fQRSTa was relatively small in our study, it may be difficult to consider this increase is clinically significant and may not be suggested that these patients would develop fatal arrhythmias in short term follow-up. However, fQRSTa may be increased more during the long-term follow-up in these patients. This small increase in fQRSTa in patients with MB may possible be due to myocardial ischemia. The fact that we found a significant correlation between the length of MG and fQRSTa supports this finding (longer MB segment may cause more ischemia as well as increased fQRSTa).

4.1 Limitations

The most important limitation of our study was that the sample size was limited and that it was a single center study. More accurate results can be obtained in a larger study group. In addition, the relationship among MB, fQRSTa and ventricular arrhythmia have not been evaluated. Monitoring the patients with long-term Holter ECG could have provided additional contribution to the study. Moreover,
the incidence of MB is not rare (reach up to 80% in autopsy studies), but clinical problems are very limited. In our study, we included only MB patients that caused ischemia. Therefore, our results cannot be generalized to all MB patients. Prospective studies with large-scale and long follow-ups are needed to better elucidate the relationship between MB and fQRSTa.

5 | CONCLUSIONS

The fQRSTa is a novel indicator of repolarization and depolarization heterogeneity. In our study, we demonstrated that MB length was independently associated with fQRSTa.

CONFLICT OF INTEREST

No conflict interest.

ETHICS APPROVAL STATEMENT

Harran University Faculty of Medicine Clinical Research Ethics Committee. No: 16.12.2019; 07/16.

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