ORIGINAL ARTICLE

Patients with Tombstoning Pattern on the Admission Electrocardiography Who Have Undergone Primary Percutaneous Coronary Intervention for Anterior Wall ST-Elevation Myocardial Infarction: In-Hospital and Midterm Clinical Outcomes

Erkan Ayhan, M.D.,* Turgay Isık, M.D.,* Huseyin Uyarel, M.D.,† Mehmet Ergelen, M.D.,† Gokhan Cicek, M.D.,‡ Servet Altay, M.D.,‡ Mehmet Eren, M.D.,‡ and Charles Michael Gibson, M.S., M.D.§

*From the *Cardiology Department, School of Medicine, Balikesir University, Balikesir, Turkey; †Cardiology Department, School of Medicine, Bezmialem Vakif University, Istanbul, Turkey; ‡Cardiology Department, Siyami Ersek Cardiovascular and Thoracic Surgery Center, Istanbul, Turkey; and §Cardiovascular Division, Department of Medicine, Beth Israel Deaconess Medical Center, Boston, MA*

Background: A tombstoning pattern (T-pattern) is associated with in-hospital poor outcomes patients with ST-segment elevation myocardial infarction (STEMI), but no data are available for midterm follow-up. We sought to determine the prognostic value of a T-pattern on admission electrocardiography (ECG) for in-hospital and midterm mortality in patients with anterior wall STEMI treated with primary percutaneous coronary intervention (PCI).

Methods: After exclusion, 169 consecutive patients with anterior wall STEMI (mean age: 55 ± 12.9 years; 145 men) undergoing primary PCI were prospectively enrolled in this study. Patients were classified as a T-pattern (n = 32) or non–T-pattern (n = 137) based upon the admission ECG. Follow-up to 6 months was performed.

Results: In-hospital mortality tended to be higher in the T-pattern group compared with non–T-pattern group (9.3% vs 2.1% respectively, P = 0.05). All-cause mortality was higher in the T-pattern group than non–T-pattern group for 6 month (P = 0.004). After adjusting the baseline characteristics, the T-pattern remained an independent predictor of 6-month all-cause mortality (odds ratio: 5.18; 95% confidence interval: 1.25–21.47, P = 0.02).

Conclusion: A T-pattern is a strong independent predictor of 6-month all-cause mortality in anterior STEMI treated with primary PCI. Therefore, it may be an indicator of high risk among patients with anterior wall STEMI.

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electrocardiogram; mortality; myocardial infarction; prognosis; tombstoning ST elevation

The admission electrocardiogram (ECG) plays a pivotal role in the diagnosis of ST-elevation myocardial infarction (STEMI).[1] Among STEMI patients, a tombstoning pattern (T-pattern) on the ECG, has been associated with a poor in-hospital prognosis,[2–6] but the mid- or long-term outcomes associated with this pattern have not been evaluated.

In this study, we aimed to evaluate in-hospital clinical events and midterm all-cause mortality in T-pattern patients who were admitted with anterior

Address for correspondence: Erkan Ayhan, M.D., Pasaalanı Mah.233.Sokak, Suheda Apt. No: 3 Kat: 1 Daire: 4, Balikesir 10100, Turkey. Fax: + 90 266 6121459; E-mail: erkayh@gmail.com

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wall STEMI and underwent primary percutaneous coronary intervention (PCI).

PATIENTS AND METHODS

Patient Population

We prospectively evaluated 169 consecutive patients with anterior wall STEMI who were admitted to the emergency department of our hospital and treated by urgent cardiac catheterization procedures. Patients fulfilling the following criteria were included in the study: (1) presentation within 12 hours from the onset of symptoms (typical chest pain lasting for >30 minutes); (2) the amplitude of the ST segments measured 60 ms after J-point being ≥2 mm in at least two contiguous ECG leads; (3) the patient was treated with primary PCI. The exclusion criteria were as follows: no indication for angioplasty, treatment with coronary bypass surgery, presence of advanced valve disease, left or right bundle branch block, pacemaker rhythm, Wolf-Parkinson-White syndrome, left ventricular hypertrophy, nonanterior wall infarction, >120 milliseconds width of the QRS complex, and infarct history. Note that, 12-lead ECGs (having the rate of 25 mm/s and the calibration at amplitude of 1.0 mV/10 mm) were recorded in all patients soon after their admission to the hospital. The patients were subdivided into two groups based on the admission ECG: those with a T-pattern and those with non–T-pattern (Figs. 1A and B, respectively). The T-pattern was identified according to modified criteria of Wimalaratna[2]: (1) the R wave is absent or its duration is <0.04 seconds with minimal amplitude; (2) the ST segment is convex upward and merges with the descending limb of the R wave or the ascending limb of the QS wave; (3) the peak of the convex ST segment is higher than whatever remains of the R wave; and (4) the convex ST segment merges with the ascending limb of the T wave. The above changes had to be present in at least two adjacent ECG leads. The amplitudes of ST-segment elevations in all patients were recorded. A repeat 12-lead ECG was obtained 60 minutes and 24 hours after primary PCI. The sum of ST-segment elevations were measured in leads V_1 through V_6. The difference between two measurements (admission and 60 minutes) was accepted as resolution of the sum of ST-segment elevation and expressed as ST-segment resolution ($\sum$STR). According to the classification of Schroder et al.,[7] patients with $\sum$STR ≥ 50% were accepted as no-reflow phenomenon (negative), and patients with $\sum$STR < 50% were accepted as no-reflow phenomenon (positive). ECGs were analyzed by two independent readers blinded to the outcome data. There was 99% concordance for ECG interpretation for the presence of T-pattern and non–T-pattern. The study protocol was approved by the Local Ethics Committee.

Data Sources

Demographic data and the clinical history concerning risk factors such as age, sex, diabetes mellitus (DM), hypertension, hyperlipidemia, smoking, family history for coronary artery disease (CAD), myocardial infarction, and previous drug use were obtained from patients and medical records. Reperfusion time, door-to-balloon time, and the presence of prodromal angina were recorded. In addition, heart rate and blood pressure were measured at initial presentation. A physical examination was also performed. Blood values were determined at initial presentation (before catheterization procedures) and on a daily basis during the hospital stay. Transthoracic echocardiography (TTE) was performed within the first 24 hours after admission to the intensive cardiologic care unit. TTE was performed by using a system V (Vingmed, GE, Horten, Norway) with a 2.5 MHz phased-array transducer. The left ventricular ejection fraction (EF) was measured using a modified Simpson's rule.[8]

Coronary Angiography, Primary Angioplasty, and Stent Implantation

All patients received chewable aspirin (300 mg, unless contraindicated) and clopidogrel (300 mg, loading dose) before primary PCI. Angiographic data was obtained from the records of the cardiac catheterization laboratory. Emergency coronary angiography was performed by the percutaneous femoral approach. In all cases, nonionic low-osmolality contrast media was used. The first injection was performed in the contralateral artery. Flow in the infarct-related artery (left anterior descending artery) was graded according to the Thrombolysis in Myocardial Infarction (TIMI) classification.[9] Heparin (10,000 U) was administered following the evaluation of coronary anatomy. A coronary artery stenosis of more than 50% was considered clinically significant. Occlusion of the infarct-related artery was crossed by using a

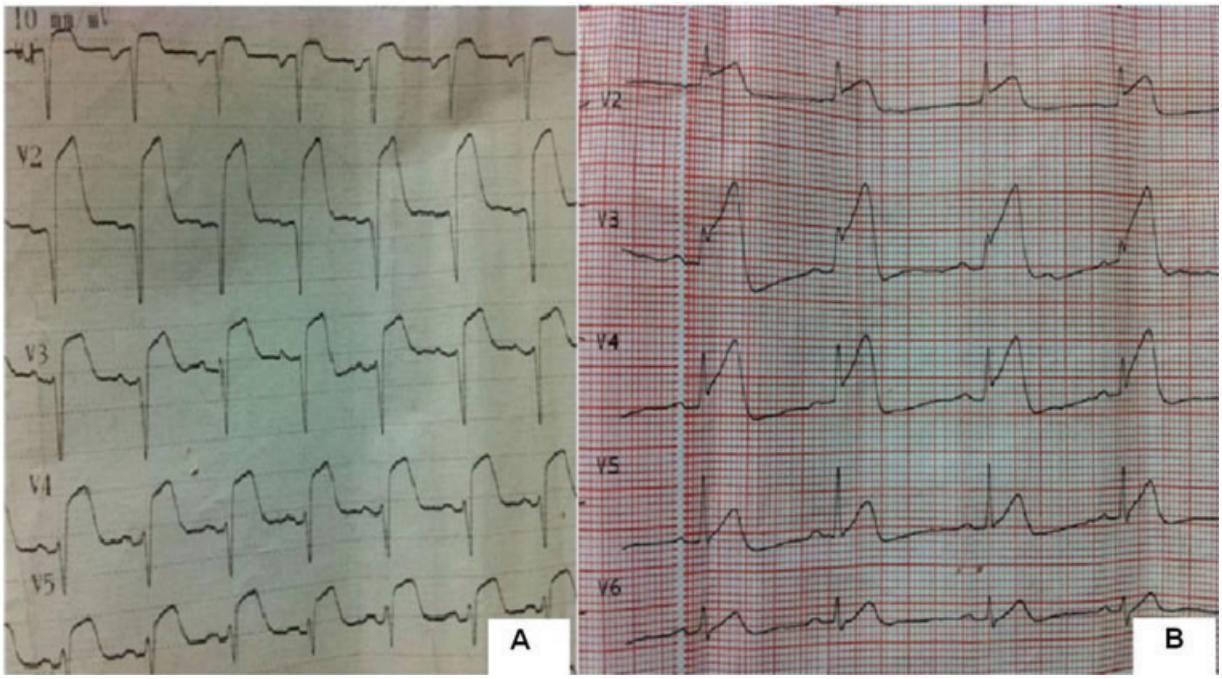

Figure 1. T-pattern (A) and non–T-pattern (B) of STEMI.

0.014-inch guidewire. Primary PCI, including balloon angioplasty, and/or stent implantation were performed only in the infarct-related artery as determined by the lesion anatomy. A successful intervention was defined as a reduction in the stenosis or obstruction to less than 50% with TIMI grade 2 or 3 flow after primary PCI.

After angioplasty, all patients were admitted to the coronary care unit, where 500 U/hour of intravenous heparin or 1 mg/kg per day of subcutaneous low-molecular-weight heparin were administered. Aspirin (100 mg/day) and clopidogrel (75 mg/day) were continued in all patients. The use of tirofiban was left to the discretion of the operator. All the patients were prescribed acetylsalicylic acid, clopidogrel, beta-blockers, an angiotensin converting enzyme inhibitor, and a statin on discharge. During the follow-up period, these five groups of drugs were administered to all patients.

Definitions

DM was considered to be present in patients with diabetes controlled by diet, oral hypoglycemic agents, or insulin, as well as in cases discharged from the hospital with a diagnosis of DM and/or prescription of hypoglycemic agents. Hyperlipidemia was defined as either the use of lipid-lowering agents, a total serum cholesterol level >240 mg/dL, or a serum triglyceride level >200 mg/dL. Reperfusion time was defined as the interval from the onset of chest pain symptoms to the first balloon inflation. Door-to-balloon time was defined as the time between hospital admission and balloon inflation. Cardiogenic shock was defined as marked and persistent (>30 minutes) hypotension with a systolic arterial pressure lower than 80 mmHg, in combination with signs of hypoperfusion due to left ventricular dysfunction and mechanical complications. Patients were also evaluated according to the Killip classification.[10] Multivessel disease was defined as presence of a stenosis greater than 50% in three major epicardial coronary arteries.

Prodromal angina was defined as typical chest pain episode (s) persisting <30 minutes either at rest or during effort 24 hours before the onset of STEMI. A positive family history of CAD was defined as documented evidence of CAD in a parent or sibling before 60 years of age. Acute stent thrombosis was defined as an abrupt onset of cardiac symptoms (i.e., an acute coronary syndrome) along with elevated biomarker levels or electrocardiographic evidence of myocardial injury after stent deployment within the first 24 hours, accompanied by angiographic evidence of a flow-limiting

thrombus near a previously placed stent. Cardiovascular mortality was defined as sudden death or mortality associated with acute myocardial infarction, heart failure, or arrhythmia. Reinfarction was described as the elevation of serum creatine kinase-MB enzyme levels by twice the upper limit of normal values along with ST-segment reelevation. Target vessel revascularization (TVR) was defined as an angioplasty or a coronary artery bypass surgery due to restenosis or reocclusion in the infarct-related artery. Major adverse cardiac events (MACE) were defined as cardiovascular mortality, reinfarction, and repeat TVR (percutaneous or surgical).

In-Hospital and Postdischarge Follow-Up

Serious ventricular arrhythmias (ventricular tachycardia and/or fibrillation), cardiopulmonary resuscitation, temporary pacing, intra-aortic balloon pump, acute stent thrombosis, mortality, dialysis, cardiogenic shock, and MACE were evaluated during the hospital stay, although data regarding 6-month follow-up were obtained from hospital records or by interviewing patients (directly or by telephone), their families, or their personal physicians.

Statistical Analysis

Quantitative variables were expressed as mean value ± SD, and qualitative variables were expressed as percent (%). Comparison of parametric values between two groups were performed by means of two tailed Student's *t*-test. Categorical variables were compared by the likelihood ratio χ^2 test or Fisher's exact test. Backward stepwise multivariate Cox regression analysis which included variables with $P < 0.1$ was performed to identify independent predictors of midterm all-cause mortality. Unsuccessful procedure, female gender, T-pattern, body mass index >30 kg/m^2, anemia at admission, multivessel disease, ST-segment elevation ≥10 mm, and Killip >1 were entered into the model. The cumulative survival curves for midterm all cause mortality were constructed with the use of the Kaplan-Meier method with differences assessed with the log-rank test. A P value <0.05 was considered statistically significant. All statistical studies were carried out with SPSS program (version 15,0, SPSS, Chicago, IL, USA).

RESULTS

Clinical, Demographic Characteristics, and Biochemical Parameters

The baseline characteristics of the patients are shown in Table 1. When the two groups were compared (32 patients with T-pattern and 137 patients with non–T-pattern), T-pattern patients were found to be older (mean age: 61.2 ± 15.7 years vs 53.6 ± 11.8 years, $P = 0.03$). Although history of hypertension was more prevalent higher in the T-pattern group ($P = 0.02$), the non–T-pattern group had more smokers and prodromal angina. At first presentation, creatinine and creatine kinase MB levels were higher in the T-pattern group compared with the non–T-pattern group.

Angiographic and Procedural Characteristics

Angiographic and procedural characteristics for both of the groups are shown in Table 2. There was no significant difference between the two groups in terms of multivessel disease, pre-PCI TIMI flow grade, post-PCI TIMI flow grade, left ventricular EF, duration of hospital stay, use of tirofiban, stent deployment, or procedural success.

In-Hospital and Follow-Up Outcomes

In-hospital mortality tended to be higher in T-pattern group compared with non–T-pattern group (Table 3; 9.3% vs 2.1%, respectively, $P = 0.05$). The need for dialysis, inotropic agent usage, and intraaortic balloon pump usage were found to be of significantly more frequent in the T-pattern group; however, MACE, TVR, cardiopulmonary resuscitation, serious ventricular arrhythmias, success of the procedure, and the frequency of acute stent thrombosis were similar in both groups. Midterm results are shown in Table 4. Six-month cardiovascular mortality was 15.6% versus 2.9% in T-pattern and non–T-pattern groups, respectively ($P = 0.004$). The incidence of heart failure requiring hospitalization was also higher in the T-pattern group ($P = 0.004$). Midterm survival curves for both groups are shown in Figure 2.

Independent Predictors of Midterm All-Cause Cardiovascular Mortality

Independent predictors of midterm all-cause cardiovascular mortality are shown in Table 5. On

Table 1. Baseline Characteristics of Study Patients

	T-Pattern (-) (n = 137)	T-Pattern (+) (n = 32)	P-Value
Age, years	53.6 (11.8)	61.2 (15.7)	0.03
Age ≥75 years	9 (6.5)	7 (21.8)	0.008
Hyperlipidemia	46 (33.5)	7 (21.8)	0.19
Diabetes mellitus	23 (16.7)	7 (21.8)	0.49
Current smoker	96 (70.1)	12 (37.5)	0.001
Family history for CAD	60 (43.7)	11 (34.3)	0.33
Hypertension	52 (37.9)	19 (59.3)	0.02
SBP, mmHg	127.6 (21.4)	129 (25.7)	0.76
Heart rate, beats/min	80.2 (12.5)	84.6 (14.2)	0.08
Killip class	>16 (4.3)	4 (12.5)	0.08
Female	18 (13.1)	6 (18.7)	0.41
Prodromal angina	37 (27)	3 (9.3)	0.03
Reperfusion time (minute)	228.9 (176.8)	258.9 (178.6)	0.38
Door-to-balloon time (minute)	33.7 (11.7)	36.4 (17.9)	0.28
Pathologic Q wave in the lead precordial (24 hours)	14 (10.2)	6 (18.7)	0.17
∑STR (<50%)	10 (7.2)	5 (15.6)	0.13
Fractionation of the QRS	3 (2.1)	1 (3.1)	0.75
Prior aspirin use	19 (13.8)	5 (15.6)	0.79
Prior beta-blocker use	21 (15.3)	7 (21.8)	0.37
Prior statin use	8 (5.8)	0 (0)	0.16
Prior ACE/ARB use	32 (23.3)	11 (34.3)	0.19
Prior CCB	9 (6.5)	2 (6.2)	0.94
Admisson creatinine (mg/dL)	0.85 (0.22)	0.98 (0.47)	0.03
Peak CK-MB, U/L	184.6 (162.2)	255.12 (149.3)	0.05
Admission glucose (mg/dL)	160.6 (70.7)	171.5 (62.9)	0.42
Total cholesterol (mg/dL)	199.1 (51.6)	190.3 (52.4)	0.39
LDL-cholesterol (mg/dL)	125.5 (41.6)	116.4 (39.8)	0.26
HDL-cholesterol (mg/dL)	41.1 (13.2)	44.7 (11.3)	0.08
Triglycerides (mg/dL)	157.7 (82.6)	141.4 (92.1)	0.32
Hemoglobin (g/dL)	14.6 (1.7)	14.2 (1.4)	0.26

Mean values (SD) and % (n) are reported for continuous and categorical variables, respectively. T-pattern = tombstoning pattern; CAD = coronary artery disease; BMI = body mass index; SBP = systolic blood pressure; PCI = percutaneous coronary intervention; CCB = calcium channel blocker; STR = ST-segment resolution; ACE/ARB = angiotensin converting enzyme/angiotensin receptor blocker; CK-MB = creatinine kinase-MB; HDL = high-density lipoprotein; LDL = low-density lipoprotein.

multivariate analysis, high Killip class, presence of multivessel disease, presence of anemia on admission, and T-pattern on ECG were documented as independent predictors of all cause cardiovascular mortality. A T-pattern alone, in addition to ST-segment elevation, was independently increasing all-cause cardiovascular mortality (odds ratio: 5.18, 95% confidence interval: 1.25–21.47, $P = 0.02$).

DISCUSSION

A T-pattern of ST elevation has been related to a higher in-hospital event rate.[2–6,11] In previous studies,[2,3,6,11] fibrinolytic therapy was more commonly performed rather than primary PCI. The major finding of the present study is that among patients treated with primary PCI in the modern era, a T-pattern is associated with a higher risk for not only in-hospital but also 6-month all-cause mortality. The T-pattern was one of the independent predictors of 6-month all-cause mortality. Furthermore, hospitalization for heart failure was more common in the T-pattern group.

Although the exact mechanism remains unknown, there are several hypotheses as to why a tombstone-like pattern occurs on the ECG. Several of the proposed mechanisms are as follows: extremely rapid myocardial damage, multivessel disease, insufficient myocardial-protective effect of prodromal angina, and elevated wall stress.[2–6] This study suggests that the T-pattern may be

Table 2. Angiographic and Procedural Characteristics of Patients

	T-Pattern (-) (n = 137)	T-Pattern (+) (n = 32)	P-Value
Multivessel disease	19 (13.8)	5 (15.6)	0.85
Pre-TIMI grade			0.79
0/1	135 (98)	32 (100)	
2	1 (0.7)	0 (0)	
3	1 (0.7)	0 (0)	
Post-TIMI grade			0.81
0/1	2 (1.5)	1 (3.1)	
2	13 (5.6)	3 (9.3)	
3	122 (92.9)	28 (87.5)	
Stent	28 (20.4)	4 (12.5)	0.26
Stent length (mm)	18.5 (4.7)	20.6 (3.8)	0.05
Stent diameter (mm)	3.1 (0.35)	3.1 (0.31)	0.39
Proximal location of the lesion	99 (72.2)	25 (78.1)	0.5
Tirofiban	62 (45.2)	10 (31.2)	0.15
Success of procedure	129 (94.1)	31 (96.8)	0.54

Mean values (SD) and % (n) are reported for continuous and categorical variables, respectively. T-pattern = tombstoning pattern; TIMI = Thrombolysis in Myocardial Infarction.

Table 3. In-Hospital Cardiac Events and Complications

	T-Pattern (-)(n = 137)	T-Pattern (+) (n = 32)	P-Value
In-hospital mortality	3 (2.1)	3 (9.3)	0.05
Reinfarction	3 (2.1)	1 (3.1)	0.75
Target-vessel revascularization	1 (0.7)	0 (0)	0.63
MACE	6 (4.3)	4 (12.5)	0.08
Serious ventricular arrhythmia	13 (9.4)	6 (18.7)	0.16
Cardiopulmonary resuscitation	6 (4.3)	4 (12.5)	0.08
Cardiogenic shock	6 (4.3)	4 (12.5)	0.08
Intraaortic balloon pump	0 (0)	2 (6.2)	0.003
Renal failure requiring dialysis	0 (0)	1 (3.1)	0.04
New atrial fibrillation	1 (0.7)	3 (9.3)	0.004
Complete atrioventricular block requiring transient pacemaker	1 (0.7)	0 (0)	0.63
Acute stent thrombosis	4 (2.9)	1 (3.1)	0.95
Time of hospital stay (days)	7.8 (5)	6.5 (3.3)	0.12
LVEF (%)	41.8 (7)	40.8 (9.3)	0.5

Mean values (SD) and % (n) are reported for continuous and categorical variables, respectively. T-pattern = tombstoning pattern; MACE = major adverse cardiac events (cardiovascular death, reinfarction, target-vessel revascularization); LVEF = left ventricular ejection fraction.

explained at least in part by the absence of prodromal angina, which is in accordance with the study by Balci et al.[3,11] Because prodromal angina exerts protective effects on the myocardium to limit infarct size and ischemic preconditioning, lack of these positive effects might be associated with more prominent ST-segment elevations.[12,13] Although, Tomcsányi et al. proposed that impaired TIMI grade flow was associated with a T-pattern, this study does not support this hypothesis.[5]

Although prior studies[3,6] conducted in the fibrinolytic era documented in-hospital mortality rates of 38.2% and 26% among patients with and without a T-pattern, respectively, a study conducted in the era of primary PCI by Tomcsányi et al.[5] provided a similar in-hospital mortality ratio (13%) to that of our study (9.3%). Thus, in both fibrinoytic and primary PCI patients, the all-cause cardiovascular mortality rate among patients with a T-pattern remains much higher, compared to that of a non–T-pattern.

Kukla et al. reported[6] the presence of a T-pattern was related to increased in-hospital mortality independent of ST-segment elevation, as in our study. Also, in our study peak creatine kinase-MB values were increased in patients with a T-pattern. Previous studies[14,15] demonstrated that peak creatine kinase-MB values were associated with poor outcomes in patients with STEMI. This relationship may help us to understand pathogenesis poor prognosis patients with T-pattern.

CONCLUSION

Although primary PCI is quite successful in a general population patients, a T-pattern among

Table 4. 6-Month Follow-Up Outcomes

	T-Pattern (-)(n = 137)	T-Pattern (+)(n = 32)	P-Value
All-cause mortality	4(2.9)	5(15.6)	0.004
Cardiac death	4(2.9)	5(15.6)	0.004
Non-cardiac death	0	0	–
Fatal reinfarction	1(0.7)	0(0)	0.63
Target-vessel revascularization	21(15.3)	3(9.3)	0.39
MACE	26(18.9)	8(25)	0.44
HF requiring hospitalization	1(0.7)	3(9.3)	0.004

Mean values (SD) and % (n) are reported for continuous and categorical variables, respectively. T-pattern = tombstoning pattern; MACE = major adverse cardiac events (cardiovascular death, reinfarction, target-vessel revascularization); HF = heart failure.

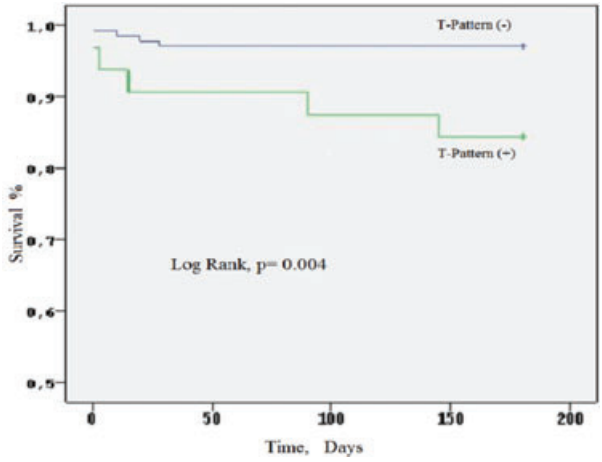

Figure 2. Kaplan-Meier curve for all cause cardiovascular mortality of patients with and without T-pattern at midterm.

patients with an anterior STEMI is strongly associated with increased hospitalization for heart failure and midterm all-cause mortality.

Limitations of the Study

Several limitations should be taken into consideration while assessing the results of our study. First, a relatively small number of patients were enrolled, but compared with previous studies our study population is larger. Second, keeping in mind that the T-pattern develops in the early stages of myocardial infarction,[3,6] we might have underestimated the prevalence for T-pattern in patients because of the fact that the pattern might have already been changed by the time patients were admitted to the emergency department. However, the percentage of T-pattern patients we included in our study (18.9%) was in accordance with the previous studies.[3,6] Third, myocardial viability was not assessed during follow-up (using stress echocardiography, magnetic resonance, etc.). Holter monitoring was not used to assess for arrhythmic events. Fourth, the follow-up period in our study was restricted to 6 months. Fifth, we have no data to support proposed explanations regarding the underlying mechanism.

Table 5. Predictors of Midterm Cardiovascular Mortality

	Odds Ratio	95% CI	P-Value
Univarite predictors			
Unsuccessful procedure	4.23	1.06–16.9	0.04
Female	5.1	1.36–18.8	0.01
T-pattern	5.63	1.5–20.9	0.01
BMI >30 kg/m^2	3.1	0,85–11.82	0.08
Anemia at admission	3.7	0.78–18.1	0.09
Multivessel disease	4.17	0.84–20.66	0.08
ST-segment elevation ≥10mm	13.08	3.6–48.7	<0.001
Killip >1	49.28	12.19–199.25	0.001
Independent predictors			
Killip >1	44.75	10.19–196.6	<0.001
Anemia at admission	7.82	1.13–53.87	0.03
T-pattern	5.18	1.25–21.47	0.02
Multivessel disease	4,41	0.91–21.4	0.06

T-pattern = tombstoning pattern; BMI = body mass index; CI = confidence interval.

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