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Long-term outcome of pre-specified ECG patterns in acute coronary syndrome

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Abstract:

Background: Long-term outcome of real-life acute coronary syndrome (ACS) patients with selected ECG patterns is not well known.

Purpose: To survey the 10-year outcome of pre-specified ECG patterns in ACS patients admitted to a university hospital.

Methods: A total of 1184 consecutive acute coronary syndrome patients in 2002-2003 were included and followed up for 10 years. The patients were classified into nine pre-specified ECG categories: 1) ST elevation; 2) pathological Q waves without ST elevation; 3) left bundle branch block (LBBB); 4) right bundle branch block (RBBB) 5) left ventricular hypertrophy (LVH) without ST elevation except in leads aVR and/or V_1; 6) global ischemia ECG (ST depression ≥0.5 mm in 6 leads, maximally in leads V_{4-5} with inverted T waves and ST elevation ≥0.5 mm in lead aVR); 7) other ST depression and/or T wave inversion; 8) other findings and 9) normal ECG.

Results: Any abnormality in the ECG, especially Q waves, LBBB, LVH and global ischemia, had negative effect on outcome. In age- and gender adjusted Cox regression analysis, pathological Q waves (HR 2.28, 95%CI 1.20-4.32, p=0.012), LBBB (HR 3.25, 95%CI 1.65-6.40, p=0.001), LVH (HR 2.53, 95%CI 1.29-4.97, p=0.007), global ischemia (HR 2.22, 95%CI 1.14-4.31, p=0.019) and the combined group of other findings (HR 3.01, 95%CI 1.56-6.09, p=0.001) were independently associated with worse outcome.

Conclusions: During long-term follow-up of ACS patients, LBBB, ECG-LVH, global ischemia, and Q waves were associated with worse outcome than a normal ECG, RBBB, ST elevation or ST depression with or without associated T-wave inversion. LBBB was associated with the highest mortality rates.

Keywords: Acute coronary syndrome; ECG; left bundle branch block; prognosis; long-term mortality.

Introduction

Acute coronary syndrome (ACS) has poor long-term outcome[1, 2]. However, mortality in ACS varies widely among patients. ECG is the cornerstone of early risk assessment in ACS due to its wide availability and good diagnostic yield [3].

ACS is usually classified as ST-elevation myocardial infarction (STEMI), non-STEMI (NSTEMI) or unstable angina (UA) according to ischemic symptoms in combination with ST deviations and cardiac troponin levels [4]. There are no ECG-related criteria for NSTEMI or UA except for the lack of ischemic ST elevations (STE). Patients with NSTEMI or UA may have a wide range of ECG changes that may affect their outcome. Some of these changes may reflect myocardial ischemia and some may imply underlying cardiac pathology. The ECG changes include Q waves, ST depression (ST-D), T-wave inversions (TWI), left ventricular hypertrophy (LVH), left bundle branch block (LBBB), right bundle branch block (RBBB) and global ischemia (GI). A patient with NSTEMI or UA may as well have a normal ECG.

Long- and short-term prognosis of many ECG patterns has been widely studied. However, knowledge about the long-term prognosis of normal vs. abnormal ECG in ACS is scarce. The aim of the present study was to assess the mortality rates of several pre-specified ECG patterns, including a normal ECG, during 10 years of follow-up in ACS patients.

Material and Methods

The study protocol was previously described in detail [5]. TACOS is a real-life study of 1188 patients with acute coronary syndrome. The study was conducted in the region of Tampere University Central Hospital with a population of ~340.000. All consecutive patients with acute myocardial infarction (AMI) were recruited between 1 January 2002 and 31 March 2003. AMI was verified by elevated blood troponin (cTnI>0.2 μg/L). Troponin-negative patients with UA were recruited from 1 September 2002 to 31 March 2003. Patients discharged from the emergency department were not included. Also, the patients who died in the emergency department were excluded.

The study was observational. The treatment for each patient was chosen by the treating physician according to the regional, national and international guidelines.

Data were gathered by a study nurse and two investigators (ME and KjN). Follow-up started at the time point of the ECG used in the analysis and ended at the time of death or at the end of follow-up 31 January 2013. Median follow-up time of the survivors was 10.3 years (from 9.8 to 11.1 years). Mortality data were gathered from the Causes of Death register, maintained by Statistics Finland, which records 100% of deaths of Finnish citizens in Finland and nearly 100% abroad.

ECG

ECGs taken in the ambulance, referring health center or emergency department were screened for the study. For each patient, the acute stage ECG with maximal ischemic changes was chosen for the analysis. The patients were classified based on the ECG findings according to the QRST morphology as follows: STE; ST-D and/or TWI (ST-D/TWI); GI; Q wave; LBBB; RBBB; LVH; other ECG changes – and normal ECG. An example of each group is shown in Figure 1.

STE was defined as ST-segment elevation in two adjacent leads: in leads $V_{1\text{-}6}$ $\geqslant$1.5 mm with $\geqslant$2 mm in at least one lead, in leads II, III, aVF, I and aVL $\geqslant$1 mm. The T-P interval was used as the reference line.

ST-D was defined as a negative shift of at least 0.5mm from the baseline at the J-point in at least two contiguous leads. The cut-off for TWI was 0.5mm. A biphasic T wave was defined as inverted, if the terminal portion of the wave was negative. Patients with ST-D were classified as GI in case of: ST-D ≥0.5 mm in ≥6 leads, maximally in leads V_{4-5} with inverted T waves and STE ≥0.5 mm in lead aVR.

In the Q wave group, STE fulfilling the abovementioned criteria were not allowed. The definition of pathological Q waves was: 1) in leads $V_{1\text{-}3}$ any Q wave $\geqslant$30 ms in duration; 2) in

leads I, II, aVL, aVF, V_{4-6} Q wave ⩾1 mm deep and ⩾30 ms in duration in ⩾2 adjacent leads; and 3) in leads V_{1-2} R wave duration >40 ms and R/S ratio >1 in the absence of pre-excitation, right ventricular hypertrophy or right bundle branch block.

LBBB was defined as QRS≥120ms, broad and notched or slurred R waves in leads aVL, V_5, and V_6, absent Q waves in leads I, V_5, and V_6, and R-wave peak time prolongation of >60 ms in leads V_5 and V_6 [6].

RBBB was defined as 1) QRS≥120ms; 2) in leads V1 or V2 rsr´, rsR´ or rSR´ configuration OR normal R peak time in V5-V6 but >50ms in V1 in the presence of pure dominant R wave in V1; 3) S wave duration greater than R wave duration or S wave duration >40ms in leads I and V6.

For ECG-LVH two criteria were used, the Sokolow-Lyon voltage criteria: S wave in V1 + R wave in V5-6 >35 mm and R-wave voltage >11 mm in lead aVL. ST-T changes secondary to LVH (ST-D in leads I, aVL, V5, V6 and STE in V1-V2) or other ST-D and/or TWI were included in this category.

The group of other ECG changes comprises patients with changes in their QRS complex or ST-T changes other than those mentioned above. These changes included intraventricular conduction defect (n=36), ventricular paced rhythm (n=18), ventricular rhythm (n=4), ST changes not fulfilling the criteria for STE or ST-D (n=3), high T waves (n=3), pre-excitation (n=1) and extreme left axis deviation (n=1).

Normal ECG was defined as an ECG with normal QRST configuration. Left anterior and posterior fascicular blocks were included in the normal ECG group.

We excluded patients with missing ECG (n=1) and heart rate >130 (n=3). The final study population comprises 1184 patients.

The ECGs were analyzed by three investigators (KjN, ME, KK).

Statistics

In the baseline characteristics, we present numbers of patients and percentages for categorical variables. Chi square is used for the statistical analyses in categorical variables. When applicable, we used Fisher´s exact test. For continuous variables, we present median values with interquartile ranges (IQR). We used the Kruskal-Wallis test for the statistical analyses of continuous variables. Survival of patients in the different ECG groups is illustrated by a Kaplan-Meier curve, where the difference between the groups was tested with the Log Rank test. To adjust survival with age and gender, we performed forward stepwise Cox regression analysis. We present hazard ratios with 95% confidence intervals (CI). Statistical analyses were done with SPSS 25.

Ethics

All patients gave a written informed consent. The study protocol was approved by the Ethics Committee of Tampere University Hospital. The study was done according to the principles of Declaration of Helsinki.

Results

The baseline characteristics of each ECG group are shown in Table 1. Most patients were male (58.4%). The proportion of females differed among the groups being highest in GI (56.7%). Smoking was most common among patients with STE (24.9%) and least frequent among those with LBBB (5.1%). Hypertension was most common in the GI and LVH groups. Type 1 diabetes was infrequent in all groups (0-3.1%), while the proportion of type 2 diabetes varied between 15% (Normal ECG) and 34.9% (RBBB); the proportion was high in the LBBB (31.4%), other ECG changes (34.8%) and GI (30.2%) groups as well. The rate of prior AMI was highest in the Q wave (34.8%) and Other ECG changes (38.5%) groups and lowest in the Normal ECG group (10%). The ranges of in-hospital PCI and CABG were 4.2-24.6% and 3.0%-27.8%, respectively. Medications on admission and at discharge are shown in Table 1.

The median age of the study population was 72 years (IQR 63-80). Age differed remarkably among the ECG groups. The youngest patients were in the Normal ECG category (median 60, IQR 53-69) and the oldest in the GI (median 77, IQR 72-82), RBBB (median 77, IQR 71-83), LBBB and LVH (median 77, IQR 71-84 for both) categories. Median creatinine values varied between 74 (Normal ECG) and 103 μmol/l (Other ECG change). Median CRP values were between four (Normal ECG) and 22 (Q wave) mg/l. Systolic blood pressure was clearly highest in the LVH group (median 160, IQR 143-189).

The Kaplan-Meier curve (Fig. 2) shows the survival benefit of normal ECG compared to all other groups throughout the follow-up. The ECG groups STE and ST-D/TWI had similar long-term survival rates. The patients with RBBB, Q waves, other ECG changes, LVH, GI and LBBB had the lowest survival rates. The poor outcome of the patients with LBBB was evident from the beginning to the end of follow-up. The p-value for the difference between the groups is <0.001 (Log Rank).

To adjust survival with age and gender, we performed Cox regression analysis. The results are shown in Table 2. Adjusted survival was worst for LBBB (HR 3.25, 95% CI 1.65-6.40, p=0.001). Other ECG groups with high mortality rates in the adjusted model were GI (HR 2.22, 95% CI 1.14-4.31, p=0.019), Q waves (HR 2.28, 95%CI 1.20-4.32, p=0.012), LVH (HR 2.53, 95% CI 1.29-4.97, p=0.007) and other ECG changes (HR 3.01, 95% CI 1.56-6.09, p=0.001). RBBB, STE and ST-D/TWI did not differ from normal ECG in the adjusted model.

Discussion

The present study of consecutive ACS patients evaluated the prognostic significance of several pre-specified ECG manifestations, including a normal ECG, at presentation. The pre-specified ECG groups had clear differences in their baseline characteristics and medication at hospital admission. It is not surprising that patients with Q waves in the ECG are more likely to have a history of previous AMI or that patients with ECG-LVH are more likely to have a history of hypertension. Therefore, each ECG pattern to some part reflects the complete patient profile instead of representing an independent phenomenon.

In the present study, a normal ECG predicted favorable outcome as compared to any studied QRST change. Normal ECG does not rule out ischemia or cardiac pathology but is a known predictor of favorable outcome in suspected AMI [7]. In the patients with normal ECG, 45% had elevated troponin levels. Ischemia may have resolved at the moment of the ECG recording or it may not be severe enough to be reflected in the ECG. It may also be that human eye is blind to subtle ischemic ECG changes. It was recently reported that deep neural network analysis reliably predicted death from an ECG considered normal by cardiologists in a large electronic health record database [8]. However, our results imply that normal ECG – as the human eye sees it – is a reliable predictor of favorable outcome in ACS.

STEMI is often considered the most acute and severe form of ACS. However, long-term outcome of STEMI does not go hand in hand with its bad reputation [1]. New STE in the ECG – especially with reciprocal ST-D – is usually due to acute coronary occlusion but STE may be caused by non-ischemic causes, such as pericarditis, early repolarization syndrome or Brugada syndrome [4]. Total occlusion of the culprit artery is more often seen in STEMI than in NSTEMI, while the opposite is true for multivessel disease [9-12]. It was somewhat surprising that the outcome of STE patients in the present study was almost as good as in those with a normal ECG; the same was true for ST-D/TWI. After adjusting with age and gender, STEMI patients did not have significantly higher 10-year mortality than those with a normal ECG (Table 2). The aforementioned difference in coronary disease severity may explain the relatively good outcome of STE. Especially compared with the patients with LBBB or GI, those presenting with STE less often had comorbidities such as hypertension or type 2 diabetes (Table 1). It is noteworthy that 8.3% of the patients with STE were troponin-negative. These patients did not have STEMI but most probably either more persistent or transient ST elevation with subsequently normal troponin levels. These troponin-negative patients contributed to the relatively favorable outcome of the STE group. As mentioned above, STE may also be caused by non-ischemic causes. However, patients with final diagnosis other than ACS were not included in this study.

ST-D and TWI often appear simultaneously in ACS patients. We therefore combined these ECG manifestations as one group. TWI may reflect many different conditions, but in patients with symptoms indicating ACS, ischemia is the likely cause [13, 14]. A large registry data study showed that in-hospital mortality in NSTEMI patients with isolated TWI was lower than in

those with a normal ECG [15]. Ischemic ST-Ds are thought to reflect regional subendocardial ischemia [16]. However, ST-D has low or moderate sensitivity and specificity for AMI [17]. In a study by Savonitto et al. ACS patients with ST-D had higher 6-month mortality than patients with STE. As compared to patients with both STE and ST-D, mortality was similar [11]. It is noteworthy that in that study, LBBB was classified as STE, and it is likely that the outcome of STE would have been even better without LBBB. In the present study, the outcome of ST-D/TWI patients was worse than in those with a normal ECG. However, in the Cox regression analysis, the difference was not statistically significant.

GI in the ECG typically reflects complex coronary artery disease, typically either left main or three-vessel disease [18, 19]. Due to the complex disease, mortality is high in GI [20], and this was also the case in the present study. GI patients more often had elevated troponin levels (99%), and hypertension (62.5%) than the other groups (Table 1).

Q waves are traditionally thought to reflect myocardial necrosis and scar [21]. In the acute phase of ACS this may not always be the case. In STEMI, Q waves imply larger infarcts but they may be transient [22]. Q waves also imply higher risk of death in STEMI [23, 24]. There is also study data indicating worse outcome in non-Q-wave MI than in Q-wave MI. [25]. It has to be pointed out that we excluded patients with STE from the Q-wave group. Therefore, our results cannot be directly compared with the results of studies comparing Q-wave and non-Q-wave MI. Based on this, our Q-wave patient group probably consists of both “late comers” with a first ACS and those with an acute event “on top of” one or more old MIs (Table 1). In these patients, Q waves probably represent myocardial necrosis/scar rather than a great ischemic area at risk. This group of patients has not been well studied before. The 10-year mortality of patients with Q waves was nearly twice as high as in those with a normal ECG.

The left bundle branch of the cardiac conduction system is perfused via septal branches of the left anterior descending coronary artery and distal branches of the right or left circumflex coronary artery. Thus, new-onset LBBB may imply multivessel disease [26, 27]. LBBB may imply underlying structural heart disease, which may have significant negative impact on patient outcome [28, 29] both with [30, 31] and without [32, 33] ACS. The results for the present study confirm previous study results, but they also give new information; of several

pre-specified ECG presentations, LBBB proved to be associated with the highest mortality rates during long-term follow-up.

The right bundle branch is perfused dominantly by branches of the left anterior descending artery (LAD) [34]. In the setting of ACS, RBBB may thus reflect infarct of the LAD territory. RBBB is a predictor of higher mortality in STEMI [35] and NSTEMI [36]. In a study by Widimsky et al, AMI patients with new or presumably new RBBB had higher in-hospital mortality than patients with LBBB or old RBBB. TIMI 0 flow of the infarct-related artery was more common in RBBB than in LBBB. [37] According to this study, ESC 2017 STEMI guidelines recommend considering invasive strategy in patients with ischemic symptoms and RBBB [38], although this was recently questioned [39]. Timoteo et al found higher one-year mortality in patients with RBBB than LBBB [40]. Contrary to that finding, the present study showed remarkably lower long-term mortality in RBBB than in LBBB. However, we didn´t define RBBB as old or presumably new.

ECG-LVH may be caused by various cardiac conditions, such as hypertensive heart disease or aortic stenosis, all of which may affect the prognosis [41]. ECG criteria for LVH have low sensitivity but good specificity for left ventricular hypertrophy confirmed with autopsy or cardiac imaging [42-44]. ECG-LVH has been associated with poor prognosis both in patients with AMI [45] and in those without AMI [46]. Thus, ECG-LVH should be considered as a tool to assess prognosis rather than a tool to diagnose LVH. In the present study, the prognosis of patients with ECG-LVH was among the worst of the ECG categories.

The ECG group “Other” was associated with relatively poor long-term outcome and a high rate of comorbidity at baseline. As this was a heterogeneous group from the ECG point of view, it is difficult to draw any firm clinical conclusions of the significance of different ECG changes. The group included patients with broad QRS other than RBBB or LBBB. Previous studies have showed higher mortality in ACS patients with wider QRS [47, 48]. In a study by Lev et al, ACS patients with undetermined ECG pattern had higher mortality and more comorbidities than patients with determined ECG pattern (STE or non-ST-elevation) [49]. However, LBBB was defined as undetermined ECG which may contribute to the poor prognosis.

Limitations

As with other studies, which have explored the long-term outcome of ACS patients, also this study has the limitations associated with changes in the treatment of ACS. The use of emergent and urgent invasive evaluation and of anti-thrombotic and statin treatment has changed a lot since the time when the study was performed. At the time of the study, in-hospital PCI was not routine treatment in ACS. Partly the low percentage is due to the all-comer nature of the study. Some patients may have been too old or co-morbid to be suitable candidates for coronary angiography.
Therefore, it is challenging to assess the long-term outcome of ACS. However, the difference in outcome between the different ECG manifestations is likely to prevail despite the changing treatment.

Conclusions

During 10-year follow-up of ACS patients, LBBB, ECG-LVH, GI, and Q waves were associated with worse outcome than a normal ECG, RBBB, STE or ST-D/TWI. LBBB was associated with the highest mortality rates.

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Table 1. Baseline characteristics based on ECG groups.

	Normal ECG n(%) n=40	ST elevation n(%) n=353	STD/TWI n(%) n=160	Global ischemia n(%) n=97	Q wave n(%) n=272	LBBB n(%) n=71	RBBB n(%) n=43	LVH n(%) n=82	Other ECG change n(%) n=66	All n(%) n=1184	p value
Female	14 (35.0)	128 (36.3)	83 (51.9)	55 (56.7)	91 (33.5)	37 (52.1)	15 (34.9)	43 (52.4)	41 (37.6)	492 (41.6)	<0.001
Smoking	8 (21.6)	83 (24.9)	30 (20.0)	10 (11.9)	55 (22.3)	3 (5.1)	3 (7.7)	9 (12.2)	3 (5.6)	204 (18.9)	<0.001
Hypertension	18 (45.0)	177 (50.3)	88 (55.7)	60 (62.5)	136 (50.7)	42 (59.2)	19 (45.2)	51 (62.2)	41 (62.1)	632 (53.8)	0.100
Diabetes											0.040
Type 1	0 (0)	2 (0.6)	1 (0.6)	3 (3.1)	3 (1.1)	0 (0)	1 (2.3)	0 (0)	2 (3.0)	12 (1.0)	
Type 2	6 (15.0)	77 (21.8)	35 (22.0)	29 (30.2)	67 (24.7)	22 (31.4)	15 (34.9)	15 (18.3)	23 (34.8)	289 (24.5)	
Prior AMI	4 (10.0)	53 (15.2)	28 (17.9)	30 (30.9)	93 (34.8)	19 (27.1)	13 (30.2)	21 (25.6)	25 (38.5)	286 (24.5)	<0.001
PCI	6 (15.0)	87 (24.6)	17 (10.6)	10 (10.3)	33 (12.1)	3 (4.2)	3 (7.0)	4 (4.9)	9 (13.6)	172 (14.5)	<0.001
CABG	4 (10.0)	26 (7.4)	10 (6.3)	27 (27.8)	27 (9.9)	5 (7.0)	4 (9.3)	6 (7.3)	2 (3.0)	111 (9.4)	<0.001
TnI positive	18 (45.0)	324 (91.8)	113 (70.6)	96 (99.0)	234 (86.0)	60 (84.5)	34 (79.1)	68 (82.9)	47 (71.2)	994 (84.0)	<0.001
Medication at admission											
β blocker	16 (41.0)	142 (40.3)	87 (54.4)	65 (67.0)	135 (49.6)	35 (49.3)	22 (51.2)	45 (54.9)	38 (57.6)	585 (49.5)	<0.001
Diuretic	6 (15.4)	70 (19.9)	49 (30.6)	49 (50.5)	94 (34.6)	42 (59.2)	16 (37.2)	37 (45.1)	36 (54.5)	399 (33.8)	<0.001
Statin	9 (23.1)	65 (18.4)	47 (29.4)	26 (26.8)	55 (20.2)	14 (19.7)	8 (18.6)	17 (20.7)	20 (30.3)	261 (22.1)	0.121
ACE inhibitor	2 (5.1)	54 (15.3)	26 (16.3)	27 (27.8)	69 (25.4)	23 (32.9)	12 (27.9)	19 (23.2)	24 (36.4)	256 (21.7)	<0.001
ARB	4 (10.3)	19 (5.4)	13 (8.1)	5 (5.2)	21 (7.7)	6 (8.5)	1 (2.3)	11 (13.4)	3 (4.5)	83 (7.0)	0.232
ASA	19 (48.7)	124 (35.1)	72 (45.3)	54 (56.3)	124 (45.6)	32 (45.7)	20 (46.5)	41 (50.0)	41 (62.1)	527 (44.7)	0.001
Clopidogrel	1 (2.6)	4 (1.1)	2 (1.3)	1 (1.0)	2 (0.7)	1 (1.4)	1 (2.3)	0 (0)	0 (0)	12 (1.0)	0.888
Nitrate	19 (48.7)	118 (33.4)	75 (46.9)	69 (71.1)	128 (47.1)	48 (67.6)	24 (55.8)	44 (54.3)	39 (59.1)	564 (47.7)	<0.001
CCB	10 (25.6)	71 (20.2)	35 (21.9)	27 (27.8)	45 (16.5)	16 (22.5)	12 (27.9)	16 (19.5)	16 (24.2)	248 (21.0)	0.379
Digoxin	1 (2.6)	19 (5.4)	15 (9.4)	18 (18.6)	29 (10.7)	19 (26.8)	8 (18.6)	25 (30.5)	9 (13.6)	143 (12.1)	<0.001
Warfarin	1 (2.6)	21 (5.9)	17 (10.6)	15 (15.5)	38 (14.0)	17 (23.9)	5 (11.6)	12 (14.6)	17 (25.8)	143 (12.1)	<0.001

Medication at discharge											
β blocker	34 (85.0)	333 (94.3)	148 (92.5)	93 (95.9)	256 (94.1)	64 (90.1)	39 (90.7)	77 (93.9)	56 (84.8)	1100 (92.9)	0.067
Diuretic	13 (32.5)	157 (44.5)	80 (50.0)	87 (89.7)	187 (68.8)	59 (83.1)	28 (65.1)	63 (76.8)	45 (68.2)	719 (60.7)	<0.001
Statin	25 (62.5)	256 (72.5)	86 (53.8)	58 (59.8)	146 (53.7)	25 (35.2)	15 (34.9)	36 (43.9)	29 (43.9)	676 (57.1)	<0.001
ACE inhibitor	4 (10.0)	167 (47.3)	56 (35.0)	38 (39.2)	159 (58.5)	43 (60.6)	18 (41.9)	37 (45.1)	29 (43.9)	551 (46.5)	<0.001
ARB	4 (10.0)	21 (5.9)	16 (10.0)	6 (6.2)	22 (8.1)	5 (7.0)	1 (2.3)	10 (12.2)	4 (6.1)	89 (7.5)	0.446
ASA	35 (87.5)	332 (94.1)	141 (88.1)	80 (82.5)	241 (88.6)	55 (77.5)	34 (79.1)	71 (86.6)	51 (77.3)	1040 (87.8)	<0.001
Clopidogrel	9 (22.5)	115 (32.6)	29 (18.1)	16 (16.5)	43 (15.8)	6 (8.5)	5 (11.6)	9 (11.0)	7 (10.6)	239 (20.2)	<0.001
Nitrate	22 (55.0)	252 (71.4)	103 (64.4)	81 (83.5)	204 (75.0)	52 (73.2)	35 (81.4)	65 (79.3)	44 (66.7)	858 (72.5)	0.003
CCB	14 (35.0)	54 (15.3)	32 (20.0)	25 (25.8)	35 (12.9)	14 (19.7)	10 (23.3)	22 (26.8)	12 (18.2)	218 (18.4)	0.003
Digoxin	2 (5.0)	27 (7.6)	17 (10.6)	30 (30.9)	49 (18.0)	19 (26.8)	6 (14.0)	26 (31.7)	16 (24.2)	192 (16.2)	<0.001
Warfarin	4 (10.0)	58 (16.4)	33 (20.6)	22 (22.7)	90 (33.1)	28 (39.4)	7 (16.3)	25 (30.5)	21 (31.8)	288 (24.3)	<0.001

	Normal ECG median (IQR)	ST elevation median (IQR)	STD/TWI median (IQR)	Global ischemia median (IQR)	Q wave median (IQR)	LBBB median (IQR)	RBBB median (IQR)	LVH median (IQR)	Other ECG change median (IQR)	All median (IQR)	p value
Age	60 (53-69)	68 (56-77)	72 (59-79)	77 (72-82)	73 (64-80)	77 (71-84)	77 (71-83)	77 (71-84)	75 (69-79)	72 (63-80)	<0.001
Creatinine	74 (67-95)	84 (71-99)	81 (67-99)	92 (75-115)	90 (75-112)	100 (81-127)	84 (73-114)	90 (71-120)	103 (85-135)	87 (72-109)	<0.001
Maximum CRP	4 (2-9)	10 (3-37)	9 (2-50)	19 (4-67)	22 (5-69)	14 (5-67)	13 (4-100)	16 (4-68)	11 (3-33)	12 (3-57)	<0.001
Systolic BP	145 (133-168)	144 (126-166)	150 (132-172)	144 (122-170)	141 (121-161)	146 (124-160)	156 (137-187)	160 (143-189)	139 (117-163)	145 (126-167)	<0.001
Diastolic BP	84 (71-91)	80 (70-91)	80 (70-90)	77 (62-91)	81 (70-90)	79 (66-90)	78 (69-94)	83 (66-98)	76 (65-83)	80 (69-91)	0.093

AMI=acute myocardial infarction; PCI=percutaneous coronary intervention; CABG=coronary artery bypass grafting; TnI=troponin I; ACE=angiotensin converting enzyme; ARB=angiotensin receptor blocker; CCB=calcium channel blocker; CRP=C-Reactive Protein; BP=blood pressure

Table 2. Cox regression. Adjusted hazard ratios for 10-year mortality are shown.

	HR	95% CI	p value
Normal ECG	Ref.	Ref.	Ref.
ST-D/TWI	1.45	0.74-2.81	0.277
STE	1.49	0.78-2.83	0.225
RBBB	1.84	0.89-3.82	0.100
GI	2.22	1.14-4.31	0.019
Q	2.28	1.20-4.32	0.012
LVH	2.53	1.29-4.97	0.007
Other ECG changes	3.01	1.56-6.09	0.001
LBBB	3.25	1.65-6.40	0.001
Age	1.07	1.06-1.08	<0.001
Gender (female)	0.90	0.77-1.06	0.199

ST-D=ST depression; TWI=T-wave inversion; STE=ST elevation; Q=Q wave; GI=global ischemia; LVH=left ventricular hypertrophy; LBBB=left bundle branch block

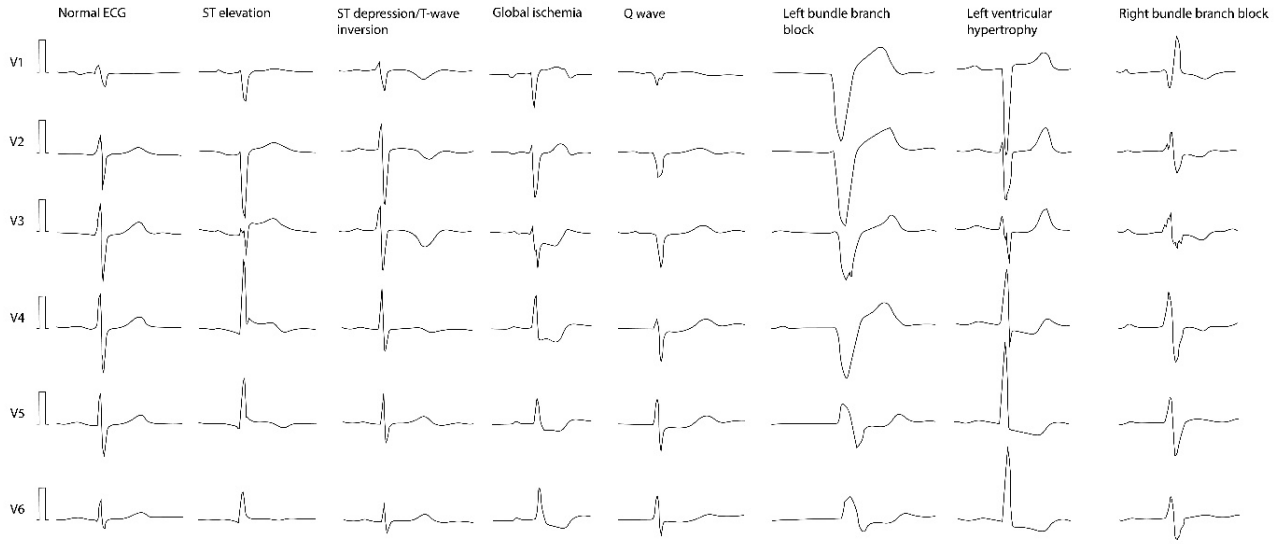

Figure 1. An example of each ECG group (50mm/s). Precordial leads are shown.

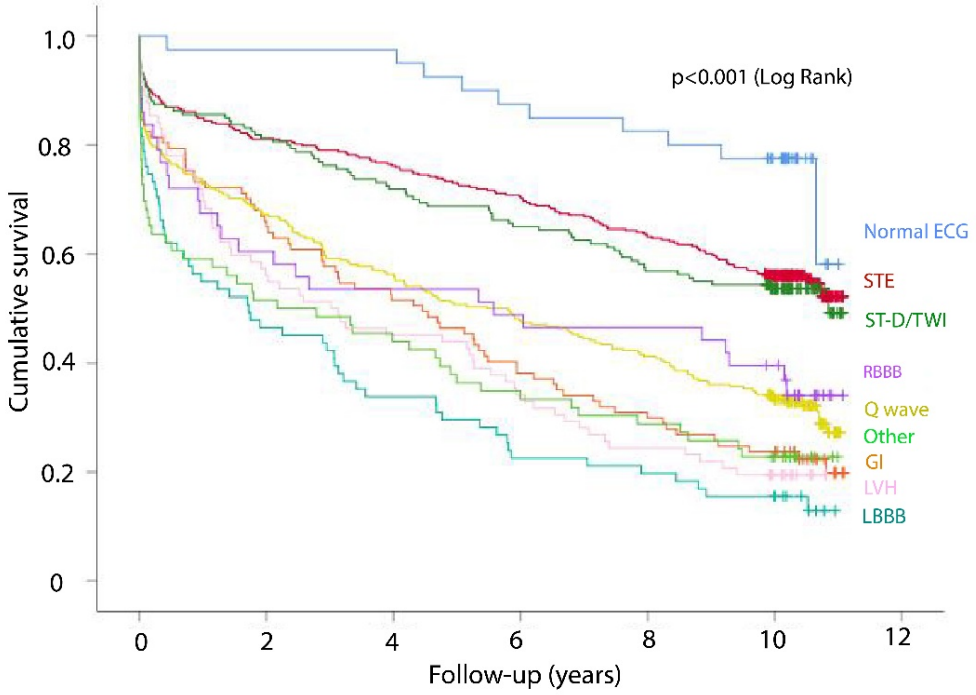

Figure 2. The Kaplan–Meier analysis showing the survival of patients according to the ECG groups during ten-year follow-up.