Predictors of Ventricular Fibrillation at Reperfusion in Patients With Acute ST-Elevation Myocardial Infarction Treated by Primary Percutaneous Coronary Intervention.

Demidova, Marina; Carlson, Jonas; Erlinge, David; Platonov, Pyotr

Published in:
American Journal of Cardiology

DOI:
10.1016/j.amjcard.2014.11.025

2015

Predictors of Ventricular Fibrillation at Reperfusion in Patients with Acute ST-elevation Myocardial Infarction Treated by Primary Percutaneous Coronary Intervention

Marina M. Demidova, MD[1,2], Jonas Carlson, PhD[1], David Erlinge, MD, PhD [1], Pyotr G. Platonov, MD, PhD[1]

1 – Department of Cardiology, Lund University, Lund, Sweden

2 – Federal Medical Research Center, St. Petersburg, Russia

Correspondence Address:
Marina M. Demidova, MD
Department of Cardiology
Lund University
22185, Lund
Sweden
fax 4646157857; tel. 046172435
e-mail: marina.demidova@med.lu.se

Abstract

Ventricular fibrillation during reperfusion (rVF) in ST-elevation myocardial infarction (STEMI) is an infrequent, but serious event that complicates coronary interventions. Our aim was to analyse clinical predictors of rVF in an unselected population of STEMI patients treated with percutaneous coronary intervention (PCI). Consecutive STEMI patients admitted to a tertiary care hospital for primary PCI during 2007-2012 were retrospectively assessed for the presence of rVF. Admission ECG stored in a digital format, were analysed for a maximal ST elevation in a single lead (maxST) and a sum of ST-deviations in all leads (sumST). Clinical, ECG and angiographic characteristics were tested for association with rVF using logistic regression analysis. Among 3724 STEMI patients admitted during 2007-2012, 71 (1.9%) suffered from rVF. In univariate analysis, history of myocardial infarction (MI), aspirin and beta-blocker use, VF before PCI, left main coronary artery disease, inferior MI localization, symptom-to-balloon time less than 360 minutes, maxST greater than 300 μV and sumST > 1500 μV were associated with increased risk of rVF. In a multivariate analysis, sumST > 1500 μV (OR 3.7; p=0.006, 95%CI 1.45-9.41) before PCI remained independent predictor of rVF. In-hospital mortality was 18.3% in rVF group vs. 3.3% in NoVF group (p<0.001), however rVF was not an independent predictor of in-hospital death. In conclusion, the magnitude of ST-elevation before PCI for STEMI independently predicts rVF and should be considered in periprocedural arrhythmic risk assessment. Despite higher in-hospital mortality in rVF patients, reperfusional VF itself has no independent prognostic value for prognosis.

Key words: ST-elevation myocardial infarction (STEMI), ventricular fibrillation, reperfusion

Ventricular fibrillation (VF) during reperfusion for ST-elevation myocardial infarction (STEMI) is an infrequent event, however it complicates coronary interventions and subsequent hospital stay (1). Because of its relatively low incidence, the predictors and prognostic value of VF at reperfusion are usually analysed together with other VF episodes at any time of acute STEMI (2-4). Some studies divided VF on early and late (2, 5), some of them dealt with prereperfusion (6), periprocedural (1) or postprocedural (7) VF. While experimental studies have been searching for specific underlying mechanisms of reperfusional arrhythmias on cellular level, clinical studies focused on VF at reperfusion for STEMI in unselected populations, to the best of our knowledge, are lacking. Most clinical studies analysed clinical and angiographic predictors of VF, whereas the data on dynamic electrocardiographic changes that can predict VF, especially VF at reperfusion, are scarce. Our aim was to analyse electrocardiographic (ECG) characteristics associated with VF during reperfusion in unselected STEMI patients treated with percutaneous coronary intervention (PCI).

Methods.

Consecutive STEMI patients admitted to Lund University Hospital for primary PCI during 2007-2012 were retrospectively assessed for the presence of VF during reperfusion. The information about cardiopulmonary resuscitation or defibrillation for VF was retrieved from the Swedish National Register of Information and Knowledge about Swedish Heart Intensive Care Admissions (RIKS-HIA) and information about reperfusion arrhythmias was obtained from Swedish Coronary Angiography and Angioplasty Register (SCAAR). Then medical histories of patients were reviewed to crosscheck VF occurrence and its timing in relation to infarct-related artery (IRA) opening. Relevant clinical information was taken from the RIKS-HIA Register, angiographic characteristics were determined from the SCAAR Register.

ECG stored in digital format either in GE Marquette MUSE system (GE Medical Systems, Milwaukee, Wisconsin) or Infinity MegaCare ECG Management System (Dräger, Lübeck, Germany) databases were analysed for predictors of VF during reperfusion. We looked for

admission ECG and a historical ECG recorded prior coronary event.

A previously recorded standard 12-lead ECG unrelated to STEMI available for interpretation was defined as historical ECG. Most recent ECG was used for analysis if several historical ECGs were available. Apart from the standard criteria (P, PR, QRS, QT, QTc intervals, presence of right or left bundle branch block (RBBB/LBBB)), the presence of J-point elevation at least 1mm above baseline in two contiguous either inferior or lateral leads was analysed.

An ECG recorded after onset of STEMI, but prior to coronary intervention was defined as admission ECG. If several ECGs were recorded prior to PCI, the latest ECG was considered as admission ECG - either in-hospital or pre-hospital ECG (in those cases when in-hospital ECG prior to PCI was not taken or not saved in the database). The ECGs with paced rhythm were excluded. ECGs with complete RBBB/LBBB were excluded from analysis of parameters characterizing ventricular repolarization (i.e. ST-level, QT, QTc). On the base of admission ECG, the maximal ST elevation in a single lead with most prominent elevation (STmax), the sum of ST-deviations in all 12 leads (sumST), including ST-elevation and reciprocal depression, as well as Anderson-Wilkins and Sclarowsky-Birnbaum scores were calculated.

In brief, the Anderson-Wilkins acuteness score (8) takes into consideration the presence of abnormal Q-waves to the Selvester QRS scoring system (9) and the morphology of T-wave, classified as tall, positive, flat or negative. The acuteness of each standard lead with ST-elevation is classified from no points to 4 points, then the total sum in all leads is divided by the number of leads involved to correct for the overall extent of the myocardial involvement.

Sclarowsky-Birnbaum score (10) assess depolarization changes during ischemia progression. Tall peaked T-waves is classified as grade I ischemia, the ST-elevation is present at grade II and changes in the terminal part of the QRS complex appear at grade III ischemia. Criteria for grade III include disappearance of S-waves in leads with Rs configuration and J point/R ratio ≥ 0.5 in leads with qR configuration.

To identify clinical factors associated with VF at reperfusion, relevant clinical,

angiographic and electrocardiographic factors were compared across groups using chi-square or Fisher's exact test for categorical variables and Student's t -test for continuous variables with an approximate normal distribution, or Man-Whitney U-test, as appropriate. Significantly associated covariates were further evaluated in univariate logistic regression models with estimation of odds ratios and likelihood-ratio tests. To determine independent factors of risk, factors significantly associated with reperfusional VF in univariate models were included in a stepwise regression analysis with backwards elimination. p -values <0.05 were considered significant. All analyses were performed using SPSS 22.0 (SPSS Inc., Chicago, Illinois, USA).

Results

Among 3274 STEMI patients admitted for primary PCI during 2007-2012, 71 (1.9%) suffered from VF during reperfusion. The incidence of reperfusion VF did not differ in different years: 13/627 (2.1%) in 2007, 11/538 (2.0%) in 2008, 11/553 (2.0%) in 2009, 12/633 (1.9%) in 2010, 10/678 (1.5%) in 2011 and 14/735 (1.9%) in 2012.

All patients suffered from VF at reperfusion during 2007-2012 comprised rVF group, and 614 consecutive patients without arrhythmias admitted during 2007 were taken as controls (No rVF group). Clinical characteristics were analysed for all 685 patients (71 in rVF group and 614 in No rVF group), however admission ECGs were not available for 17% thus leaving 567 patients included in analysis of ECG characteristics (55 patients in rVF group and 512 in No rVF group). Among them 108 were ambulance ECGs and 459 in-hospital pre-PCI ECGs. Two No rVF patients were excluded from ECG-analysis because of paced rhythm. Assessment of repolarization, including the level of ST-segment and QT interval was performed in patients without LBBB/RBBB – in total in 489 patients (42 in rVF group and 447 in No rVF group).

Historical ECGs were available for 447 patients: 40 from rVF group and 407 from No rVF group. The time from historical ECG to STEMI did not differ between groups: 27±32 month in rVF group and 40±50 in No rVF group, p=0.12.

Patients with VF at reperfusion were more likely to have a history of myocardial infarction and more often used beta-blockers and aspirin than those without VF (**Table 1**). Patients with VF at reperfusion more often suffered from VF before PCI, either out of hospital, during ambulance transport or in hospital prior to the balloon inflation. 11 patients from 71 in rVF group had VF both before and during reperfusion, whereas 60 patients had no VF before reperfusion and suffered from reperfusion VF only. There were no differences between the groups in regard to age, gender, smoking, presence of hypertension, diabetes, anamnesis of stroke, history of congestive heart failure, using digitalis at admission, proportion of patients with Killip class >1, and serum creatinine at admission. The level of potassium at admission was significantly lower in rVF group, though within normal range. Patients with VF at reperfusion had shorter symptom-to-balloon time, more often had inferior localization of myocardial infarction and left main stenosis on angiography. The percentage of multivessel disease and IRA distribution did not differ between groups.

The patients suffered from VF at reperfusion were more likely to have longer QRS interval on historical ECG before event (**Table 2**). Only one patient from rVF group had J-point elevation in lateral leads meeting criteria for early repolarization on historical ECG. The patients with VF at reperfusion had higher ST elevation before PCI, but did not differ in either Anderson-Wilkins acuteness score or Sclarovsky-Birnbaum score comparing with No rVF group. The percentage of LBBB/RBBB and conventional ECG-criteria did not differ between groups.

In a univariate regression analysis, the following factors were associated with increased risk of VF during reperfusion: history of myocardial infarction, aspirin and beta-blockers use, VF before PCI, potassium level at admission, left main coronary artery disease, inferior localization of MI, duration of QRS on historical ECG, symptom-to-balloon time less than 360 minutes, ST-elevation in a single lead greater than 300 μV and sum of ST-deviations in all leads greater than 1500 μV. **(Table 3).** In a multivariate analysis, sum of ST-deviations in all leads greater than 1500 μV before PCI and left main stenosis by angiography remained independent predictor of

VF during reperfusion.

Since pre-reperfusional VF appeared to be a strong predictor of rVF, we also performed a separate analysis of the predictors of lone rVF in patients who did not suffer from VF before IRA opening (**Table 4**). In the univariate regression analysis, the following factors were associated with increased risk of lone VF during reperfusion: history of myocardial infarction, aspirin and beta-blocker use, hypopotassiemia at admission, left main coronary artery disease, duration of QRS on historical ECG, symptom-to-balloon time less than 360 minutes, ST-elevation in single lead greater than 300 μV and sum of ST-deviations in all leads greater than 1500 μV. In a multivariate analysis, sum of ST-deviations in all leads greater than 1500 μV, aspirin use and QRS duration on historical ECG remained independent predictor of lone VF during reperfusion.

In-hospital mortality in whole rVF group was 18.3%. In-hospital mortality in patients with lone VF at reperfusion was found to be 16.7% comparing with 3.3% in patients with no VF before and during reperfusion ($p<0.001$ for both comparisons). In the univariate analysis, age, history of myocardial infarction, Killip class>1 at admission, VF before reperfusion, VF during reperfusion, left main stenosis and multivessel disease were associated with increased in-hospital mortality (**Table 5**). In the multivariate analysis, age, heart failure at admission, VF before reperfusion and left main stenosis were independent predictors of poor in-hospital prognosis. However, reperfusion VF was not an independent predictor of in-hospital mortality.

Discussion

The occurrence of VF at reperfusion in our study (1.5-2.1% in different years) is lower compared with previously reported incidence of periprocedural VF in STEMI. In APEX AMI enrolled 5745 STEMI patients, periprocedural VT/VF occurred in 180 patients – 3.1% (11). In PAMI, included 3065 patients, 133 of them (4.3%) suffered from VT/VF in cardiac catheterization laboratory (1). These differences can be explained by several factors, that distinguish our study from abovementioned. First, we took into consideration only VF and VT demanding defibrillation, not all sustained VT and VF as in (1, 4, 11). Secondly, we consider

only VF occurring after IRA opening, not all periprocedural VF as in (1). Then, these studies included STEMI patients admitted within 6 (11) or 12 (1) hours from symptom onset, and APEX AMI included high-risk STEMI only, whereas our study analysed an unselected STEMI cohort. In our previous study we showed that reperfusional VF accounted for 22% of VF occurring during the first 48 hours of STEMI treated by primary PCI (3).

The occurrence of VF at reperfusion in our study was higher in inferior STEMI localization. This is in line with previously published data concerning VF in STEMI, but not directly related to reperfusion (11-13). The higher incidence of VF in inferior STEMI, especially with right ventricular involvement can be explained by much more prominent I_{to} in epicardium of the right ventricle than the left one (14). We also observed a tendency to the prevalence of RCA as IRA that previously had been reported to be predictive for VF (1). Notably, in our cohort, patients with inferior STEMI having higher risk of rVF had lower max ST elevation than patients with anterior STEMI (309±219 vs 501±339 μV, $p<0.001$).

In earlier studies, TIMI 0 before PCI has been reported to be predictive for VF in catheterization laboratory (1). In our study all 71 patients who suffered from rVF had total acute coronary occlusions, while there were no cases of non-occlusive stenoses in this group. The rate of thrombectomy in VF group was 53% comparing to 12% in No VF group ($p<0.001$).

We have not found any association of VF at reperfusion with multivessel disease, which is in accordance with the APEX-AMI trial, concerning VF not directly related to reperfusion (11). The patients with reperfusion VF were more likely to have left main stenosis, which could lead to more intensive ischemia and larger area of myocardium at risk involved into ischemia-reperfusion. We believe that the higher rate of beta-blocker and aspirin treatment in VF group should be considered as a more sensitive indicator of underlying cardiovascular comorbidities than anamnestic information.

Hypokalemia is known to be a predictor of VF during STEMI, especially in regard to VF occurring shortly after symptom onset (15). We analysed the association of the potassium level

at admission and reperfusion VF, and found that despite the differences between rVF group and No rVF group, the average potassium level was normal in both groups. The percentage of patients with hypokalaemia at admission tended to be higher in rVF group (5.8% vs 2.7%), but the difference has not reached statistical significance. The symptom-PCI time was shorter in rVF group, in agreement with PAMI trial results (1). VF before PCI was predictive for VF during reperfusion, reflecting interindividual variation in vulnerability to ventricular arrhythmias in the settings of ischemia-reperfusion. At the same time, 85% of patients who suffered from VF during reperfusion had experienced no VF before reperfusion.

In earlier studies, the sum ST deviation at ECG was predictive for VF at any time of STEMI (11), but did not influence the occurrence of postprocedural VF (7). We found that the magnitude of ST-elevation, reflecting ischemia intensity before primary PCI, predicts VF during reperfusion. Since myocardial ischemia affects not only ventricular repolarization but also depolarization process, we scrutinized admission ECGs for Sclarovsky-Birnbaum ischemia grades. The terminal distortion of QRS-complex corresponding to grade 3 of ischemia is believed to reflect severe and prolonged ischemia that affects Purkinje fibres (10) and correlates to larger infarct size and less myocardial salvage at reperfusion (16, 17). In our study, the percentage of patients with Birnbaum 3 ischemia grade on admission did not differ between groups despite the shorter symptom to PCI time in rVF group. We believe that it might be explained by the presence of patients with fast progression myocardial infarction due to poor collateral flow or absence of preconditioning in the rVF group (10).

We reviewed not only admission ECGs, but also historical ECG before STEMI. We demonstrated that QRS duration before the coronary event is a predictor of VF during reperfusion for subsequent STEMI. This finding is in agreement with results of Tikkanen (18) on longer QRS duration in future victims of sudden death during acute coronary event.

Recently, a number of studies reported the association between J-wave pattern on historical ECG and life-threating ventricular arrhythmias and sudden death during acute ischemic event

(18-21). The association of a initially existing J-wave pattern with future arrhythmic complications during acute STEMI was explained by the presence of heterogeneity of ventricular repolarization as a substrate predisposing to the development of ventricular arrhythmias in the setting of an acute ischemia trigger (22). In our study J-wave point elevation was observed in 0,2%, that is much lower than in the above mentioned studies - 11-16% (18, 19), and than the reported early repolarization (ER) prevalence in general population – 4.5% (23). However, ER is known to be an age-dependant phenomenon, and in age group 60-70 years, corresponding the average age at the moment of historical ECG in our study, its prevalence appears to be in the range of 1.5% in women and 2-3% in man (23) thus being in line with our observations.

In-hospital mortality in rVF group was five times as high as in No rVF group as earlier reported by others (2, 6, 24). The majority of cases of in-hospital death in both rVF and No rVF groups were due to heart failure, among other causes were mechanical complications, cerebral injury and reinfarction, that is in accordance with a literature (1). Despite markedly increased in-hospital mortality in rVF group in our study, reperfusion VF has not been found to have an independent prognostic value for in-hospital mortality.

Because of the low prevalence of reperfusion VF in PCI-treated STEMI we have chosen to compare the unselected cohort of patients admitted with STEMI during 2007 with all those who had reperfusion VF during 2007-2012, which may affect interpretation of our findings since standards of care might have been modified over the six-year period. However, invasive strategy as the first-choice approach in STEMI has been adopted long before 2007, the annual incidence of reperfusion VF and symptoms-to-balloon time remained similar during study period thus suggesting that this would affect study findings in a limited extent.

Funding sources:

This study was supported by the Swedish Institute (research scholarship for MMD), Donations funds at Skåne University Hospital (PP, MMD) and The Swedish Heart Lung Foundation (PP, MMD).

References:

1. Mehta RH, Harjai KJ, Grines L, Stone GW, Boura J, Cox D, O'Neill W, Grines CL. Sustained ventricular tachycardia or fibrillation in the cardiac catheterization laboratory among patients receiving primary percutaneous coronary intervention: incidence, predictors, and outcomes. *J Am Coll Cardiol* 2004;43:1765-1772.

2. Bougouin W, Marijon E, Puymirat E, Defaye P, Celermajer DS, Le Heuzey JY, Boveda S, Kacet S, Mabo P, Barnay C, Da Costa A, Deharo JC, Daubert JC, Ferrieres J, Simon T, Danchin N. Incidence of sudden cardiac death after ventricular fibrillation complicating acute myocardial infarction: a 5-year cause-of-death analysis of the FAST-MI 2005 registry. *Eur Heart J* 2014;35:116-122.

3. Demidova MM, Smith JG, Hoijer CJ, Holmqvist F, Erlinge D, Platonov PG. Prognostic impact of early ventricular fibrillation in patients with ST-elevation myocardial infarction treated with primary PCI. *Eur Heart J Acute Cardiovasc Care* 2012;1:302-311.

4. Newby KH, Thompson T, Stebbins A, Topol EJ, Califf RM, Natale A. Sustained ventricular arrhythmias in patients receiving thrombolytic therapy: incidence and outcomes. The GUSTO Investigators. *Circulation* 1998;98:2567-2573.

5. Volpi A, Cavalli A, Santoro L, Negri E. Incidence and prognosis of early primary ventricular fibrillation in acute myocardial infarction--results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-2) database. *Am J Cardiol* 1998;82:265-271

6. Piccini JP, Berger JS, Brown DL. Early sustained ventricular arrhythmias complicating acute myocardial infarction. *Am J Med* 2008;121:797-804.

7. Mehta RH, Yu J, Piccini JP, Tcheng JE, Farkouh ME, Reiffel J, Fafy M, Mehran R, Stone GW. Prognostic significance of postprocedural sustained ventricular tachycardia or

fibrillation in patients undergoing primary percutaneous coronary intervention (from the HORIZONS-AMI Trial). *Am J Cardiol* 2012;109:805-812.

8. Wilkins ML, Pryor AD, Maynard C, Wagner NB, Elias WJ, Litwin PE, Pahlm O, Selvester RH, Weaver WD, Wagner GS. An electrocardiographic acuteness score for quantifying the timing of a myocardial infarction to guide decisions regarding reperfusion therapy. *Am J Cardiol* 1995;75:617-620.

9. Selvester RH, Wagner GS, Hindman NB. The Selvester QRS scoring system for estimating myocardial infarct size. The development and application of the system. *Arch Intern Med* 1985;145:1877-1881.

10. Birnbaum Y, Drew BJ. The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis. *Postgraduate medical journal* 2003;79:490-504.

11. Mehta RH, Starr AZ, Lopes RD, Hochman JS, Widimsky P, Pieper KS, Armstrong PW, Granger CB. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. *JAMA* 2009;301:1779-1789.

12. Volpi A, Maggioni A, Franzosi MG, Pampallona S, Mauri F, Tognoni G. In-hospital prognosis of patients with acute myocardial infarction complicated by primary ventricular fibrillation. *N Engl J Med* 1987;317:257-261.

13. Berger PB, Ruocco NA, Ryan TJ, Frederick MM, Podrid PJ. Incidence and significance of ventricular tachycardia and fibrillation in the absence of hypotension or heart failure in acute myocardial infarction treated with recombinant tissue-type plasminogen activator: results from the Thrombolysis in Myocardial Infarction (TIMI) Phase II trial. *J Am Coll Cardiol* 1993;22:1773-1779.

14. Yan GX, Lankipalli RS, Burke JF, Musco S, Kowey PR. Ventricular repolarization components on the electrocardiogram: cellular basis and clinical significance. *J Am Coll Cardiol* 2003;42:401-409.

15. Nordrehaug JE, von der Lippe G. Hypokalaemia and ventricular fibrillation in acute myocardial infarction. *Br Heart J* 1983;50:525-529.

16. Birnbaum Y, Mahaffey KW, Criger DA, Gates KB, Barbash GI, Barbagelata Al, Clemmensen P, Sgarbossa EB, Gibbons RJ, Rahman MA, Califf RM, Granger CB, Wagner GS. Grade III ischemia on presentation with acute myocardial infarction predicts rapid progression of necrosis and less myocardial salvage with thrombolysis. *Cardiology* 2002;97:166-174.

17. Birnbaum Y, Kloner RA, Sclarovsky S, Cannon CP, McCabe CH, Davis VG, Zaret BL, Wackers FJT, Braunwald E. Distortion of the terminal portion of the QRS on the admission electrocardiogram in acute myocardial infarction and correlation with infarct size and long-term prognosis (Thrombolysis in Myocardial Infarction 4 Trial). *Am J Cardiol* 1996;78:396-403.

18. Tikkanen JT, Wichmann V, Junttila MJ, Rainio M, Hookana E, Lappi OP, Kortelainen ML, Anttonen O, Huikuri HV. Association of early repolarization and sudden cardiac death during an acute coronary event. *Circulation Arrhythm Electrophysiol* 2012;5:714-718.

19. Naruse Y, Tada H, Harimura Y, Hayashi M, Noguchi Y, Sato A, Yoshida K, Sekiguchi Y, Aonuma K. Early repolarization is an independent predictor of occurrences of ventricular fibrillation in the very early phase of acute myocardial infarction. *Circulation Arrhythm Electrophysiol* 2012;5:506-513.

20. Patel RB, Ilkhanoff L, Ng J, Chokshi M, Mouchli A, Chacko SJ, Subacius H, Bhojraj S, Goldberger JJ, Kadish AH. Clinical characteristics and prevalence of early repolarization associated with ventricular arrhythmias following acute ST-elevation myocardial infarction. *Am J Cardiol* 2012;110:615-620.

21. Kim SH, Kim DH, Park SD, Baek YS, Woo SI, Shin SH, Kwan J, Park KS. The Relationship Between J Wave on the Surface Electrocardiography and Ventricular Fibrillation during Acute Myocardial Infarction. *J Korean Med Sciens* 2014;29:685-690.

22. Tikkanen JT, Anttonen O, Junttila MJ, Aro AL, Kerola T, Rissanen HA, Reunanen A, Huikuri HV. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med* 2009;361:2529-2537.

23. Noseworthy PA, Tikkanen JT, Porthan K, Oikarinen L, Pietila A, Harald K, Peloso GM, Merchant FM. Jula A, Väänänen H, Hwang SJ, O'Donnell CJ, Salomaa V, Newton-Cheh C, Huikuri HV. The early repolarization pattern in the general population: clinical correlates and heritability. J *Am Coll Cardiol* 2011;57:2284-2289.

24. Behar S, Goldbourt U, Reicher-Reiss H, Kaplinsky E. Prognosis of acute myocardial infarction complicated by primary ventricular fibrillation. Principal Investigators of the SPRINT Study. *Am J Cardiol* 1990;66:1208-1211.

Table 1. Clinical and angiographic characteristics

Variable	VF at reperfusion		p-value
	Yes (n=71)	No (n=614)	
Age (years)	68±12	66±12	0.36
Man	52 (73%)	430 (70%)	0.68
Previous myocardial infarction	**15 (22%)**	**80 (13%)**	**0.04**
Hypertension	31 (46%)	244 (40%)	0.36
Stroke	6 (9.0%)	46 (7.5%)	0.63
Chronic heart failure	4 (6.0%)	17 (2.8%)	0.14
Diabetes mellitus	5 (8%)	67 (11%)	0.53
B-blockers	**25 (40%)**	**159 (27%)**	**0.02**
Aspirin	**24 (38%)**	**149 (25%)**	**0.03**
Digitalis	2 (3.2%)	8 (1.3%)	0.24
Past or present smoker	24 (73%)	392 (67%)	0.57
HF at admission Killip>1	7 (13%)	47 (10%)	0.045
Creatinin at admission (mmol/l)	97±51	87±30	0.15
K^+ at admission (mmol/l)	**3.8±0.5**	**3.9±0.4**	**0.002**
Hypokalemia at admission	4 (5.8%)	14 (2.7%)	0.14
VF before reperfusion	**11 (16%)**	**26 (4%)**	**0.001**
Symptom-to-balloon time	**185 (164)**	**227 (254)**	**0,006**
Symptom-to-balloon time<360 min	**51 (84%)**	**359 (70%)**	**0.025**
Multivessel disease	42 (64%)	303 (55%)	0.19
Left main stenosis	**10 (15%)**	**34 (6%)**	**0.02**
RCA as IRA	34 (49%)	226 (41%)	0.2

Categorical variables are presented in%, for continuous - mean±SD (if normally distributed) or median [IQ] is presented. Abbreviations: HF-heart failure; RCA-right coronary artery; IRA-infarct related artery.

Table 2 Electrocardiographic characteristics.

	VF at reperfusion		p-value
	Yes	No	
Historical ECG:			
P duration (ms)	107±28	110±19	0.63
PR duration (ms)	164±30	164±30	0.97
QRS duration (ms)	**102±20**	**96±16**	**0.02**
QTc duration (ms)	421±28	415±26	0.34
J-point elevation in lateral leads	1 (2.1%)	0 (0%)	0.08
Admission ECG:			
P duration (ms)	105±29	110±21	0.3
PR duration (ms)	176±39	171±34	0.45
RBBB	7 (13%)	41 (8%)	0.60
LBBB	6 (11%)	22 (4%)	0.15
Inferior MI localization	**44 (67%)**	**188 (51%)**	**0.02**
ST max (μ V)	**498 [330]**	**300 [261]**	**<0.001**
ST max >300 μ V	**47 (84%)**	**253 (52%)**	**<0.001**
Sum ST (μ V)	**2289 [1933]**	**1518 [1205]**	**<0.001**
Sum ST >1500 μ V	**48 (87%)**	**249 (52%)**	**<0.001**
Birnbaum grade 3	13 (45%)	138 (40%)	0.72
Anderson-Wilkins score	2.5±0.9	2.5±1.1	0.94

Categorical variables are presented in%, for continuous - mean±SD (if normally distributed) or median [IQ] is presented. Abbreviations: RBBB-right bundle brunch block; LBBB –left bundle brunch block; MI-myocardial infarction; ST max – maximal ST in a lead with most prominent elevation; Sum ST- sum of ST deviations in all leads

Table 3. Clinical factors associated with Ventricular fibrillation during reperfusion

Charasteristics at admission	Univariate analysis			Multivariate analysis		
	HR	95% CI	p-value	HR	95% CI	p-value
Previous MI	1.93	1.04-3.58	0.039	-	-	-
QRS duration at historical ECG	1.02	1.003-1.04	0.020	-	-	-
K^+ at admission	0.40	0.22-0.73	0.003	-	-	-
VF before PCI	4.15	1.95-8.81	<0.001			
Medications:						
Aspirin	1.88	1.09-3.22	0.023	-	-	-
B-blockers	1.86	1.09-3.19	0.024	-	-	-
Symptom-to-baloon time <360 min	2.19	1.08-4.42	0.029	-	-	-
Left main stenosis	2.60	1.22-5.54	0.013	4.47	1.19-18.80	0.027
Inferior MI	1.89	1.09-3.29	0.023			
ST max >300 μ V	4.87	2.34-10.16	<0.001	-	-	-
Sum ST >1500 μ V	6.44	2.86-14.53	<0.001	4.00	1.52-10.54	0.005

Abbreviations: MI -myocardial infarction; VF-ventricular fibrillation; PCI-percutaneous coronary intervention; Left main stenosis- left main coronary artery stenosis; ST max – maximal ST in a lead with most prominent elevation, Sum ST- sum of ST deviations in all leads

Table 4. Clinical factors associated with ventricular fibrillation during reperfusion in a subgroup of patients without arrhythmias before percutaneous coronary interventions (n=60)

Charasteristics at admission	Univariate analysis			Multivariate analysis		
	HR	95% CI	p-value	HR	95% CI	p-value
Previous MI	2.33	1.22-4.48	0.011	-	-	-
QRS duration at historical ECG	1.02	1.00-1.04	0.014	1.02	1.00-1.05	0.042
K^+ admission	0.40	0.21-0.77	0.006	-	-	-
Medications:						
Aspirin	1.99	1.12-3.54	0.020	2.97	1.29-6.80	0.010
B-blockers	2.12	1.20-3.74	0.009	-	-	-
Symptom-to-baloon time <360 min	2.21	1.05-4.62	0.036	-	-	-
Left main stenosis	2.29	1.01-5.22	0.048	-	-	-
ST max >300 μ V	4.99	2.29-10.85	<0.001	-	-	-
Sum ST >1500 μ V	6.80	2.84-16.30	<0.001	4.40	1.57-12.28	0.005

Abbreviations: MI, myocardial infarction; Left main stenosis, left main coronary artery stenosis; ST max – maximal ST in a lead with most prominent elevation, Sum ST- sum of ST deviations in all leads

Table 5. Clinical factors associated with in-hospital mortality

Charasteristics at admission	Univariate analysis			Multivariate analysis		
	HR	95% CI	p-value	HR	95% CI	p-value
Age	1.08	1.04-1.12	<0.001	1.07	1.04-1.09	<0.001
Previous MI	1.60	1.01-2.54	0.046	-	-	-
HF >Killip1	3.21	1.30-7.91	0.011	3.56	1.98-6.39	<0.001
VF before reperfusion	9.03	4.07-20.04	<0.001	3.38	1.91-6.00	<0.001
VF at reperfusion	4.87	2.39-9.96	<0.001	-	-	-
Left main stenosis	4.97	3.07-8.03	<0.001	2.30	1.23-4.30	0.009
Multivessel coronary disease	1.60	1.10-2.54	<0.001	-	-	-

Abbreviations: MI- myocardial infarction; HF- heart failure; VF-ventricular fibrillation; Left main stenosis- left main coronary artery stenosis.