

# Prevalence of severely impaired left ventricular ejection fraction after reperfused ST-elevation myocardial infarction

David R. Altmann<sup>a</sup>, Marcus Mutschelknauss<sup>a</sup>, Niklas Ehl<sup>b</sup>, Michael Koller<sup>b</sup>, Beat Schaer<sup>a</sup>, Lucas Jörg<sup>c</sup>, Peter Ammann<sup>c</sup>, Michael Kühne<sup>a</sup>, Hans Rickli<sup>c</sup>, Stefan Osswald<sup>a</sup>, Christian Sticherling<sup>a</sup>

<sup>a</sup> Departement of Cardiology, University of Basel Hospital, Switzerland

<sup>b</sup> Institute for Clinical Epidemiology and Biostatistics, University of Basel Hospital, Switzerland

<sup>c</sup> Departement of Cardiology, Kantonsspital St. Gallen, Switzerland

## Summary

**BACKGROUND:** Preventive implantation of an implantable cardioverter defibrillator (ICD) early after myocardial infarction failed to demonstrate a survival benefit in patients with depressed left ventricular ejection fraction (LVEF). This may be explained by early recovery of the LVEF after percutaneous coronary intervention (PCI). We sought to determine the incidence of a sustained LVEF  $\leq 35\%$  in patients with severely depressed LVEF early after a revascularised acute ST-segment elevation myocardial infarction (STEMI).

**METHODS:** LVEF was assessed in patients with an acute STEMI treated with PCI in two Swiss high-volume centres within 10 days (in-hospital LVEF) after the STEMI. Those with an in-hospital LVEF  $\leq 35\%$  were scheduled for follow-up LVEF measurement within 6–8 weeks.

**RESULTS:** A total of 330 patients were included (79% male, mean age  $63 \pm 12$  years). In-hospital LVEF measured  $3 \pm 3$  days after STEMI was  $\leq 35\%$  in 32/330 patients (10%, 95% confidence interval (CI) 13%–67%). LVEF was available in 31/32 (97%) patients at follow-up  $53 \pm 19$  days after STEMI and improved to  $>35\%$  in 19 patients (61%, 95% CI 42%–78%). The incidence of a LVEF  $\leq 35\%$  at follow-up was 39% (12/31, 95% CI 22%–56%).

**CONCLUSION:** Our data demonstrate that the incidence of severely impaired LV function 53  $\pm$  19 days after a STEMI treated with PCI is low. A severely depressed LVEF early after STEMI was present in 10% of all patients. Only 39% of these patients had a persistently impaired LVEF during follow-up. These findings support an expectant strategy be-

fore considering primary preventive ICD implantation after STEMI.

**Key words:** left ventricular ejection fraction; ST-elevation myocardial infarction; implantable cardioverter defibrillator

## Introduction

Current guidelines for the prevention of sudden cardiac death (SCD) after myocardial infarction recommend the implantation of an implantable cardioverter defibrillator (ICD) in patients with a left ventricular ejection fraction (LVEF)  $\leq 35\%$  (for patients in NYHA class II or III) or  $\leq 30\%$  (NYHA class I) not earlier than 40 days after myocardial infarction [1]. However, the landmark studies leading to these recommendations started patient recruitment in 1990 and 1997 [2, 3]. The contemporary management of myocardial infarction with aggressive early revascularisation therapy by percutaneous coronary intervention (PCI), as well as the significant improvement of medical therapy, has markedly changed and the Multicenter Automatic Defibrillator Implantation Trial (MADIT I and II) populations do not necessarily reflect today's post-myocardial infarction patients [4, 5]. The Defibrillator in Acute Myocardial Infarction Trial (DINAMIT) demonstrated no survival benefit for patients with a LVEF  $<40\%$  receiving an ICD 6 to 40 days after an acute myocardial infarction [6]. Follow-up LVEF measurements were carried out in only 47% of patients. Hence the evolution of a depressed LVEF early after myocardial infarction in the DINAMIT population is not known. The Immediate Risk Stratification Improves Survival (IRIS) trial replicated the data, including patients with an LVEF  $\leq 40\%$  5 to 31 days after an acute myocardial infarction [7]. Unfortunately, it also does not comment on the evolution of the LVEF. Stunned or hibernating myocardium may recover after revascularisation and the LVEF will improve in many patients [8]. For the clinician a common clinical problem is when to implant an ICD

### Abbreviations

ICD:	implantable cardioverter defibrillator
LVEF:	left ventricular ejection fraction
PCI:	percutaneous coronary intervention
SCD:	sudden cardiac death
STEMI:	ST elevation myocardial infarction

in a patient who suffered a myocardial infarction and has an impaired LVEF. Prospective data regarding the prevalence and time course of a severely impaired LVEF after STEMI are sparse. We conducted a prospective observational study in STEMI patients treated with primary PCI to assess the incidence of a depressed LVEF  $\leq 35\%$  early after a STEMI and to evaluate the evolution of the LVEF in these patients.

## Methods

This study was performed as an observational study between September 2008 and March 2010 at the University Hospital Basel (UHBS) and the Kantonsspital St. Gallen (KSSG), and was approved by the local ethics committee. Patients admitted for the treatment of STEMI were screened. The data were prospectively recorded from medical charts. Patients were included if PCI was performed within 12 hours after the onset of symptoms and if the LVEF was assessed within 10 days after PCI (in-hospital LVEF). Exclusion criteria were known LVEF  $\leq 35\%$  of any cause before the index STEMI and inability to obtain informed consent.

The diagnosis of STEMI was based on the history of chest pain lasting for 15 minutes or more in association with ST-segment elevation in two contiguous leads (cut-off points:  $\geq 0.2$  mV in leads  $V_2$ – $V_3$  and/or  $\geq 0.1$  mV in other leads), or new or presumed new left bundle-branch block, and was confirmed by the presence of an unstable coronary lesion on angiography [9, 10]. Cardiogenic shock as a clinical state of hypoperfusion was diagnosed if systolic blood pressure was  $< 90$  mm Hg, or if the central filling pressure (wedge pressure) was  $> 20$  mm Hg, or if intravenous inotropes and/or mechanical catheter-based cardiac assist devices (UHBS: Impella, Abiomed Europe; KSSG: intra-aortic balloon pump, Sensation 7 Fr IAB Catheter, Maquet Getinge Groupe) were needed to maintain a systolic blood pressure  $> 90$  mm Hg. Periprocedural resuscitation was defined as electrical cardioversion and/or mechanical resuscitation.

### LVEF measurement and mortality

LVEF assessment by echocardiography was at the discretion of the treating physician and was estimated, preferably using the Simpson biplane formula. Only LVEF measurements assessed after removal of mechanical assist devices and inotropics were recorded. Two cardiologists, blinded for the subjects study participation, reviewed all echocardiography studies. Follow-up LVEF was reassessed using echocardiography 6 to 8 weeks after the index event in patients with an in-hospital LVEF  $\leq 35\%$ . A telephone follow-up was performed to assess mortality at the same period in those with an in-hospital LVEF  $> 35\%$ . Cause of death was classified as recorded in medical records. Death, either in hospital or after hospital discharge, was assumed to be a SCD if it occurred within minutes after the onset of symptoms, resulted from a documented cardiac arrhythmia, or was witnessed and occurred unexpectedly.

### Laboratory and ECG values

Peripheral venous blood specimens were taken at the time of the PCI and during the hospital stay on a daily basis. The

first available peak creatine kinase was considered to be the peak value and was used in the analysis. The serum creatinine value used was the first available, preferably from the specimen taken at the time of PCI. QRS width was measured manually on a 12-lead surface ECG performed on the day following the PCI.

### Percutaneous coronary intervention

PCI was performed in standard fashion. All patients received a loading dose of 600 mg clopidogrel, and aspirin-naïve patients were administered aspirin 250 mg intravenously before PCI. Glycoprotein IIb/IIIa receptor antagonists (abciximab was used at UHBS and tirofiban at KSSG) were given at the discretion of the physician performing the intervention, but was routinely administered in the presence of reduced coronary flow after successful PCI as assessed with the Thrombolysis in Myocardial Infarction (TIMI) grading system. Heparin was given as a bolus of 5,000 IU before primary PCI and all patients had either a bare metal or drug eluting stent implanted. Postprocedural antiplatelet therapy consisted of aspirin 100 mg/day and clopidogrel 75 mg/day for at least 12 months.

The initial and postprocedural blood flow in the infarct-related artery was graded by the physician performing the PCI according to the Thrombolysis in Myocardial Infarction (TIMI) grading system [11]. The diagnosis of a “no reflow phenomenon” required angiographic evidence of a patent artery after successful PCI with no evidence of residual stenosis ( $< 50\%$ ) after intracoronary administration of nitroglycerine and exclusion of dissection, spasm, or thrombus and a TIMI flow  $< 3$ , at least 10 minutes after PCI.

### Outcome measure

The primary outcome was a persistently depressed LVEF  $\leq 35\%$  between two time intervals, namely in-hospital ( $\leq 10$  days post-PCI) and at follow-up (6–8 weeks post-PCI) in patients with an in-hospital LVEF  $\leq 35\%$ . Mortality during the study period was a secondary outcome.

### Statistical analysis

Continuous variables are expressed as mean with standard deviation (SD) if normally distributed or as median with interquartile range (IQR) in the case of deviation from normality. An incidence of patients with a LVEF  $\leq 35\%$  and the 95% confidence interval (CI) is given for in-hospital and follow-up LVEF measurements. The number of patients with available in-hospital and follow-up LVEF measurements were taken as denominator. Differences between patients with and without depressed LVEF were compared using the chi-square test or Fisher exact test for categorical variables. Normally distributed numerical variables were analysed using the Student t-test and in case of deviation from normality with the Mann-Whitney U test. A two-tailed p value of  $< 0.05$  was considered to indicate statistically significant differences. Analyses were performed using Prism software package version 5.0 (Graph Pad Software for Mac OS X, Inc.)

## Results

Patient recruitment and group outcomes are summarised in figure 1.

From a total of 560 patients admitted with suspected STEMI during the study period, 230 patients were excluded for following reasons:

Ten patients in whom LVEF was not known died. Causes of death in these patients were progressive heart failure (n = 5), incessant ventricular fibrillation (n = 1), cerebral hypoxia after resuscitation (n = 3), and noncardiac (n = 1). PCI was not successful in nine, not performed in six and a diagnosis other than STEMI (e.g. Takotsubo cardiomyopathy) was made in ten patients. LVEF was known to be  $\leq 35\%$  before the index STEMI in three patients and forty-six patients had no LVEF measurement before hospital discharge. In 146 patients informed consent could not be obtained.

Thus, the study population consisted of 330 patients (table 1). In-hospital LVEF measured  $3 \pm 3$  days after PCI was  $\leq 35\%$  in 32/330 patients (10%, 95% CI 13%–67%). LVEF was obtained using the Simpson's biplane formula in 249/330 (76%) patients and assessed visually in the remaining. Patients with an in-hospital LVEF  $\leq 35\%$  presented with higher creatine kinase levels and more often in cardiogenic shock. Use of cardiac assist devices and left anterior descending coronary artery as culprit vessel or multi-vessel disease were more frequent. Length of stay at the intensive care unit and QRS duration were longer. For history of previous myocardial infarction or revascularisation (PCI or surgical), use of antithrombotic agents during PCI and symptom-to-balloon time, no difference between the groups was found. Most patients (82%) had complete revascularisation, defined as no residual coronary artery stenosis  $>75\%$  after the index PCI. In the remaining patients, revascularisation was performed on average  $23 \pm 18$  days following the index PCI (75% PCI, 25% coronary artery bypass graft [CABG]). In the group of patients with an in-hospital LVEF  $\leq 35\%$ , ten were revascularised  $20 \pm 15$  days following STEMI.

## Follow-up LVEF

Follow-up LVEF was available for 31/32 (97%) patients  $53 \pm 19$  days following STEMI (one patient declined a follow-up visit) and improved to  $>35\%$  in 19/31 patients (61%, 95% CI 42%–78%) (LVEF  $42\% \pm 4\%$  vs  $30\% \pm 5\%$ ). The incidence of a LVEF  $\leq 35\%$  at follow-up was therefore 39% (12/31, 95% CI 22%–56%).

The LVEF recovered to  $>35\%$  in seven of ten patients revascularised during the follow-up period. Patients without LVEF improvement had longer symptom-to-balloon time (290 min [194–593] vs 200 min [135–285]; table 2) when compared with those with an improved LVEF  $>35\%$ . No patient died during follow-up. The demographic characteristics of the two groups are depicted in table 2. Adherence to heart failure therapy prescribed at discharge was 100% in all patients at follow-up. An aldosterone antagonist was prescribed for four patients during follow-up.

## Discussion

The incidence of a severely depressed LVEF  $\leq 35\%$  was 10% within days following revascularisation of an acute STEMI and improved to  $>35\%$  within 7 weeks in 61% of these patients, resulting in an incidence of sustained depressed LVEF  $\leq 35\%$  of 39% (12/31, 95% CI 22%–56%).

### Left ventricular dysfunction early after STEMI and its evolution

Although overall survival was not improved by early primary preventive ICD therapy in the DINAMIT trial, death due to arrhythmia occurred more often in controls on medical therapy (hazard ratio for ICD group 0.42,  $p = 0.009$ ) and deaths from nonarrhythmic causes in the ICD group were more frequent, not least due to device-related complications [6]. However, a high probability of left ventricular (LV) function improvement following revascularised acute coronary syndrome explains the missing benefit of early primary preventive ICD therapy in this population.

A recent study evaluated infarction size and the evolution of LV function by sestamibi scintigraphic imaging following reperfused STEMI [8]. Reduction of the preinterventional infarction size of 25% to 8% at 6 months follow-up was associated with improvement of LV function and long-term survival, although subjects had a preserved LVEF at baseline. The MISSION AMI trial identified 8% of patients with a severely depressed LVEF  $\leq 35\%$  using gated SPECT 3 months after reperfused acute coronary syndrome [12]. Of those, 4% had a LVEF  $\leq 30\%$  and overall 6% of patients were candidates for ICD implantation  $<1$  year after myocardial infarction. In CARISMA an improvement of LVEF following myocardial infarction from baseline  $31\% \pm 6\%$  to  $35\% \pm 10\%$  was observed during a follow up of 6 weeks [13]. LVEF showed fewer recovery properties in the subgroup of patients with an arrhythmic event compared with patients without. In a recent series from Zwolle, 13% of 2,544 patients treated with PCI for a STEMI had a LVEF of  $<30\%$  more than 30 days after infarction [14]. Although the investigators did not provide an initial LVEF, they identified multivessel disease and reinfarction within 1 year as

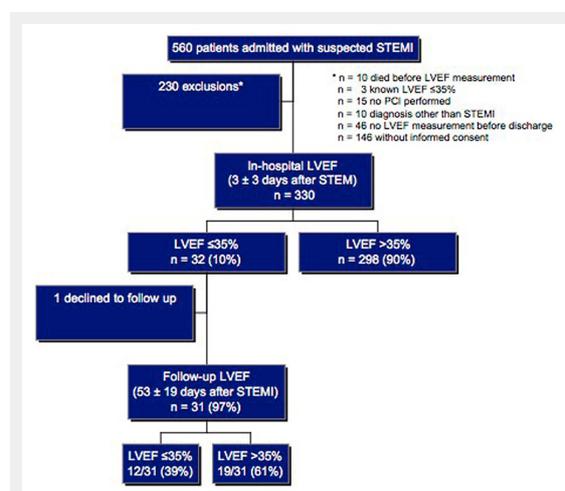


Figure 1

Study flow chart – patient recruitment and group outcome.  
LVEF = left ventricular ejection fraction; STEMI = ST-segment elevation myocardial infarction

risk factors for subsequent death, and 40% of the deaths were attributed to SCD.

Risk of persistently severe LVEF dysfunction can be explained by the magnitude of LV dysfunction and infarct size following reperfusion.

Patients with persistently severe LV dysfunction in our study showed a trend for lower LVEF early after revascularisation (see table 2). Peak creatine kinase was not different between the groups, but was higher in the group with an LVEF improvement during follow-up. This finding might be explained by more rapid coronary reperfusion as demonstrated earlier in patients with patent coronary arteries following thrombolysis-induced reperfusion compared with those with a reduced coronary flow, suggesting a washout phenomenon [15]. A lower baseline LVEF and

larger infarct size have been associated with failure of LV recovery [8].

Patients with LV remodelling presented more often with a lower LVEF and larger infarct size seen on magnetic resonance imaging and as indicated by higher creatine kinase levels [16]. Peak and cumulative creatine kinase levels have been correlated to infarct size, short- and long-term impairment of LVEF, and death [17, 18]. Nienhuis et al. studied the prognostic value of creatine kinase in large-scale, prospective observational studies performed in STEMI patients treated with primary PCI. They found that patients with anterior wall myocardial infarction are at increased risk for higher creatine kinase and that the peak creatine kinase is an independent predictor of LV dysfunction and 1-year mortality [19, 20].

**Table 1:** Baseline characteristics of the total study population and by in-hospital LVEF  $\leq 35\%$  and  $>35\%$ .

	Total (n = 330)	LVEF $\leq 35\%$ (n = 32)	LVEF $>35\%$ (n = 298)	p-value
Age (SD), y	63 $\pm$ 12	65 $\pm$ 13	63 $\pm$ 12	0.4
Men, n (%)	260 (79)	23 (72)	237 (80)	0.4
BMI [IQR], kg/m <sup>2</sup>	26 [24–29]	27 [24–29]	26 [24–28]	0.8
Medical history, n (%)				
Hypertension	180 (55)	19 (59)	162 (54)	0.7
Hyperlipidaemia	201 (62)	24 (88)	180 (60)	0.1
Smoker	197 (60)	19 (59)	178 (60)	1.0
Diabetes mellitus	42 (13)	3 (9)	39 (13)	0.8
Family history positive	90 (27)	8 (25)	82 (28)	0.8
Previous myocardial infarction	27 (8)	4 (13)	23 (8)	0.3
Previous PCI	31 (9)	5 (16)	26 (9)	0.2
Previous CABG	6 (2)	2 (6)	4 (1)	0.1
Procedural characteristics				
Creatinine [IQR], $\mu$ g/L	81 [70–92]	83 [72–88]	81 [70–92]	1.0
Peak creatine kinase [IQR], U/l	1946 [980–3709]	4493 [2493–6535]	1853 [955–3224]	<0.0001
Cardiogenic shock, n (%)	15 (5)	4 (13)	11 (4)	0.05
Assist device, n (%)	14 (4)	4 (13)	10 (3)	0.04
Culprit vessel LAD, n (%)	164 (50)	28 (88)	136 (46)	<0.0001
Multivessel disease, n (%)	178 (54)	23 (72)	155 (52)	0.04
Postprocedural TIMI flow $<III$ , n (%)	41 (12)	7 (22)	34 (11)	0.1
Mechanical ventilation, n (%)	18	3 (9)	15 (5)	0.4
GP IIb/IIIa-inhibitor use, n (%)	248 (75)	24 (75)	224 (75)	1
Symptom to balloon time [IQR], minutes	195 [145–300]	212 [150–348]	194 [144–300]	0.3
Duration of ICU care [IQR], d	2 [1–2]	2 [2–5]	2 [1–2]	0.0002
Treatment with hypothermia, n (%)	7 (2)	0	7 (2)	n/a
Echocardiographic characteristics*				
Left ventricular ejection fraction (SD), %	49 $\pm$ 10	32 $\pm$ 4	51 $\pm$ 8	< 0.0001
LVEDD, (SD), mm	48 $\pm$ 8	52 $\pm$ 8	48 $\pm$ 7	0.01
LVESD, (SD), mm	34 $\pm$ 8	40 $\pm$ 10	33 $\pm$ 7	0.009
QRS width [IQR], ms	96 [90–104]	102 [91–113]	96 [90–104]	0.04
QRS width $\geq 120$ (SD), ms	31 (9)	4 (13)	24 (8)	0.3
Left bundle branch block, n (%)	4 (1)	0	4 (1)	0.6
Medication at hospital discharge, n (%)				
Aspirin	329 (99)	32 (100)	297 (99)	1.0
Clopidogrel	330 (100)	32 (100)	298 (100)	1.0
ACE inhibitor or ARB	267 (81)	28 (88)	243 (82)	0.5
Beta-blocker	307 (93)	29 (91)	278 (93)	0.5
Statin	328 (99)	32 (100)	296 (99)	1.0
Diuretic	37 (11)	11 (34)	26 (9)	0.0002
Anticoagulant	18 (6)	5 (16)	13 (4)	0.02

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blockers; BMI = body mass index; CABG = coronary artery bypass graft; GP = glycoprotein; IQR = interquartile range; LAD = left anterior descending artery; LVEDD = left ventricular end-diastolic diameter; LVEDV = left ventricular end-diastolic volume; LVESD = left ventricular end-diastolic diameter; LVESV = left ventricular end-systolic volume; PCI = percutaneous coronary intervention; SD = standard deviation; TIMI = thrombolysis in myocardial infarction. \* Left ventricular diameter was available in 295/330 (89%) of subjects.

The median time from the onset of symptoms to the first balloon inflation was 3 hours in our study and patients with a persistently reduced LVEF  $\leq 35\%$  at follow-up had longer symptom-to-balloon times. Other authors reported significant longer symptom-to-reperfusion times (mean 287 vs 258 min,  $p = 0.03$ ) in subjects with an in-hospital LVEF  $\leq 40\%$  and inducible ventricular tachycardia in an electrophysiology study performed before hospital discharge as compared with those without. In one multicentre registry, the LVEF measured before discharge was significantly lower with a prolonged symptom-to-balloon time of  $>240$  min but did not differ at 1 month [21, 22].

Although arrhythmic risk is of concern early after STEMI, those who received appropriate shocks in the DINAMIT trial had more episodes of heart failure and myocardial infarction, and nonarrhythmic deaths were more frequent, which offset the observed sudden death reduction [6, 23].

### Limitations

Owing to the observational character of this study, not all eligible patients were analysed because of missing data on LV function before hospital discharge. Furthermore, since the two hospitals performing PCI are tertiary centres with early transfer of the patients to the referring centres, a considerable number of patients could not provide informed consent. In addition, a fraction of the study population with an in-hospital LVEF  $\leq 35\%$  might have presented with a hitherto unknown LVEF  $\leq 35\%$ . Furthermore as LVEF measurement was at the discretion of the treating physician, some eligible patients were discharged without in-hospital LVEF assessment. The lower enzymatic infarct size in these patients as compared with patients in whom LVEF was measured during hospital stay might have influenced the physician's decision (see table 1). Therefore, a selection bias can be assumed in the presented study population.

Cardiac troponins (cTn) are the preferred biomarker to detect myocardial necrosis and have been shown to be of predictive value in the setting of STEMI [24]. However, the enzymatic infarct size in acute STEMI patients measured as the cumulative creatine kinase release seems to correlate

with peak cTnT [17]. As different cTn measurements were used in the two participating hospitals, we did not evaluate the impact of cTn.

Coronary flow following PCI was assessed angiographically using the Thrombolysis In Myocardial Infarction (TIMI) flow grade. This method is widely used clinically to describe coronary flow before and after revascularisation, but it mainly describes epicardial blood flow and neglects myocardial blood flow, and hence coronary microcirculation. However, because of the observational character of this study, quantitative assessment of coronary microcirculation (e.g. using contrast echocardiography, magnetic resonance imaging, doppler flow wires or combined pressure and temperature-tipped guidewires) was not performed [25].

As we recorded only LVEF, we are not aware of other mechanisms that might have contributed to a reduced LVEF in the in-hospital and follow-up phase (e.g. role of functional mitral regurgitation). Finally, cardiogenic shock was not necessarily due to LV dysfunction but probably due to right ventricular dysfunction in the setting of right ventricular myocardial infarction.

The low event rate of sustained reduced LVEF  $\leq 35\%$  might have influenced the statistical power.

### Conclusion

Our data demonstrate that the incidence of severely impaired LV function early after STEMI treated with acute PCI is 10% and that the likelihood of LVEF improvement within weeks is high, with the majority of patients having an initially severely depressed LVEF improving to  $>35\%$ . These findings support an expectant strategy early after STEMI before considering primary preventive ICD implantation.

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**Table 2:** Baseline characteristics of patients with LVEF  $\leq 35\%$  and  $>35\%$  at follow-up.

	LVEF $\leq 35\%$ (n = 12)	LVEF $>35\%$ (n = 19)	p-value
Age (SD), y	66 $\pm$ 15	64 $\pm$ 11	0.2
Previous myocardial infarction	1 (8)	3 (16)	1.0
Previous revascularisation	2 (17)	3 (16)	1.0
Creatinine [IQR], $\mu\text{g/L}$	80 [72–86]	84 [72–93]	0.6
Peak creatine kinase [IQR], U/l	4041 [2666–5839]	6280 [2484–6690]	0.4
Cardiogenic shock, n (%)	1 (8)	3 (16)	1.0
Culprit vessel LAD, n (%)	10 (83)	17 (90)	0.6
Multivessel disease, n (%)	4 (33)	5 (26)	0.7
Postprocedural TIMI flow $<III$ , n (%)	2 (17)	4 (21)	1.0
Symptom to balloon time [IQR], minutes	290 [194–593]	200 [135–285]	0.01
Duration of ICU care [IQR], d	2.5 [2–7]	2 [2–2]	0.2
In-hospital LVEF, %	30 $\pm$ 4	33 $\pm$ 3	0.09
In-hospital LVEDD, (SD), mm	54 $\pm$ 8	50 $\pm$ 6	0.1
In-hospital LVESD, (SD), mm	43 $\pm$ 10	37 $\pm$ 8	0.07
QRS width [IQR], ms	103 [86–112]	102 [94–110]	0.1
QRS width $\geq 120$ (SD), ms	2 (17)	1 (5)	0.5
Left bundle branch block, n (%)	0	4 (21)	0.1

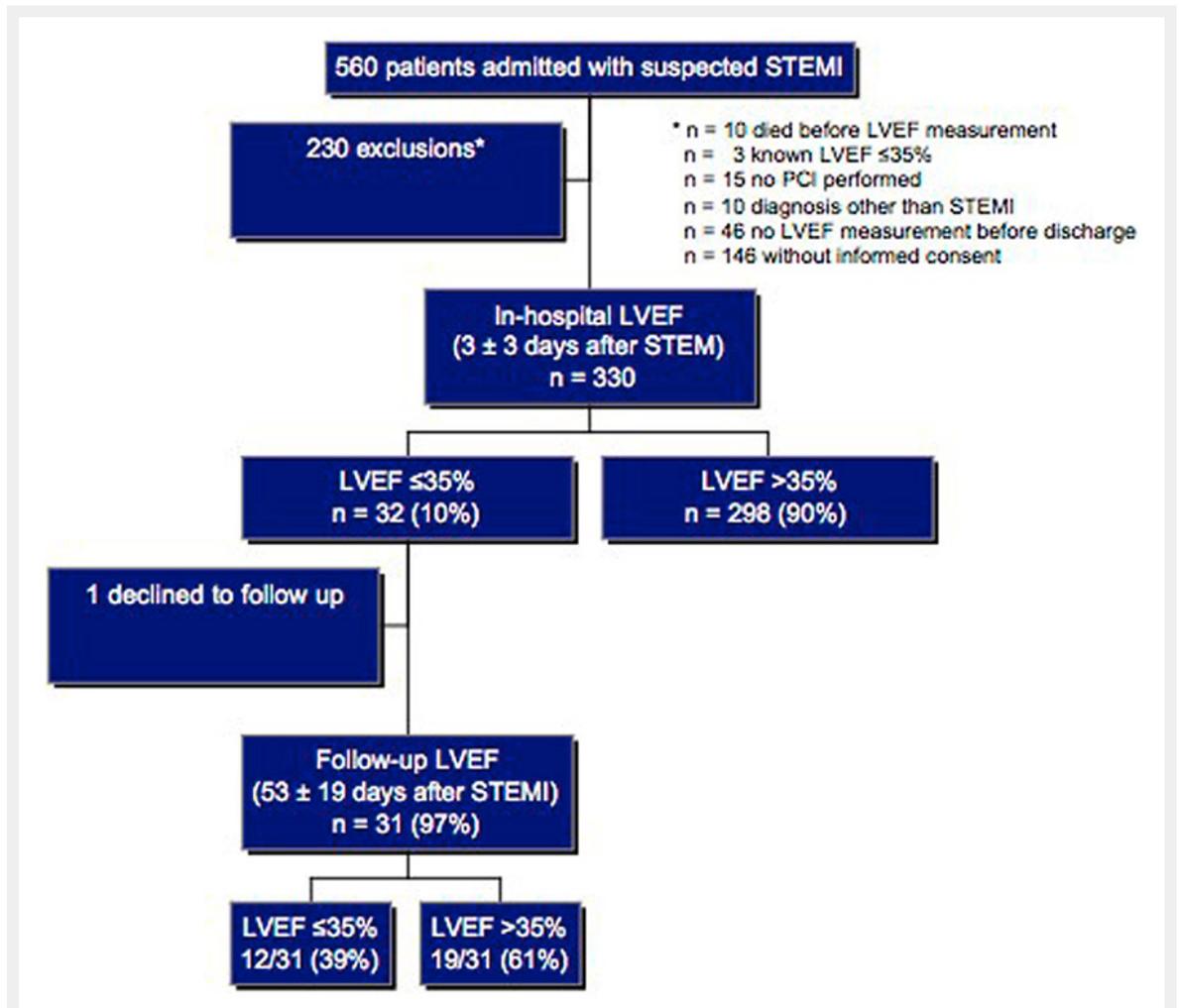
IQR = interquartile range; LAD = left anterior descending artery; LVEDD = left ventricular end-diastolic diameter; LVESD = left ventricular end-systolic diameter; PCI = percutaneous coronary intervention; SD = standard deviation; TIMI = thrombolysis in myocardial infarction

**Correspondence:** Christian Sticherling, MD, FESC, University Hospital Basel, Division of Cardiology, Petersgraben 4, CH-4031 Basel, Switzerland, [christian.sticherling\[at\]usb.ch](mailto:christian.sticherling[at]usb.ch)

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Figures (large format)



Figure

Study flow chart – patient recruitment and group outcome.

LVEF = left ventricular ejection fraction; STEMI = ST-segment elevation myocardial infarction