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ORIGINAL ARTICLE

Culprit or multivessel revascularisation in ST-elevation myocardial infarction with cardiogenic shock

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ABSTRACT

Objective The value of multivessel revascularisation in cardiogenic shock and multivessel disease (MVD) is still not clear. We compared outcomes following culprit vessel or multivessel revascularisation in patients with ST-elevation myocardial infarction (STEMI), cardiogenic shock and MVD.

Methods From 16 620 patients with STEMI who underwent primary percutaneous coronary intervention (PCI) in a nationwide, prospective, multicentre registry between January 2006 and December 2012, 510 eligible patients were selected and divided into culprit vessel revascularisation (n=386, 75.7%) and multivessel revascularisation (n=124, 24.3%) groups. The primary outcomes were inhospital mortality and all-cause death during a median 194-day follow-up. A weighted Cox regression model was constructed to determine the HRs and 95% CIs for outcomes in the two groups.

Results Compared with culprit vessel revascularisation, multivessel revascularisation had a significantly lower adjusted risk of inhospital mortality (9.3% vs 2.4%, HR 0.263, 95% CI 0.149 to 0.462, p<0.001) and all-cause death (13.1% vs 4.8%, HR 0.400, 95% CI 0.264 to 0.606, p<0.001), mainly because of fewer cardiac deaths (9.7% vs 4.8%, HR 0.510, 95% CI 0.329 to 0.790, p=0.002). In addition, multivessel revascularisation significantly decreased the adjusted risk of the composite endpoint of all-cause death, recurrent myocardial infarction and any revascularisation (20.3% vs 18.1%, HR 0.728, 95% CI 0.55 to 0.965, p=0.026).

Conclusions This study showed that, compared with culprit vessel revascularisation, multivessel revascularisation at the time of primary PCI was associated with better outcomes in patients with STEMI with cardiogenic shock. Our results support the current guidelines regarding revascularisation in these patients.

INTRODUCTION

Patients with acute ST-elevation myocardial infarction (STEMI) and cardiogenic shock exhibit increased morbidity and mortality during hospitalisation.[1] Cardiogenic shock complicates 5%–10% of all STEMI cases.[2] To improve outcomes, early revascularisation in the infarct-related artery is strongly considered.[3] However, primary percutaneous coronary intervention (PCI) in multivessel coronary artery disease (MVD) may present difficulties. Although current guidelines recommend culprit vessel revascularisation during primary PCI in patients with STEMI, multivessel revascularisation can be performed to improve clinical outcomes in patients with STEMI with persistent cardiogenic shock.[4 5] However, given its potential disadvantages, including greater risk of stent thrombosis, ongoing ischaemia, contrast-induced nephropathy (CIN) and longer radiation exposure,[6] the decision to do multivessel revascularisation during primary PCI continues to be controversial and the support for multivessel PCI in patients with STEMI and cardiogenic shock is limited.

Thus, we compared the clinical outcomes of patients with STEMI, cardiogenic shock and MVD who were treated with culprit or multivessel revascularisation.

METHODS

Study population

In this prospective, multicentre, observational, registry-based study, data from 31 149 patients with STEMI and non-STEMI between 2006 and 2013 were retrieved from the Korean Acute Myocardial Infarction Registry (KAMIR).[7 8] Patients enrolled in the KAMIR have a similar rate of current smoking and a much lower rate of dyslipidaemia and prior ischaemic heart disease compared with other STEMI registries.[9 10] The 53 participating centres included university or community hospitals with high patient volumes and facilities for PCI and on-site cardiac surgery. The protocol conformed to the guidelines of the 1975 Declaration of Helsinki, as reflected by prior approval from each participating institution's human research committee. Informed consent for data use was obtained from each patient. Clinical follow-up was performed at 1, 2, 6 and 12 months for participants enrolled from 2006 to 2007, and for up to 24 months following hospital discharge for participants enrolled from 2008 to 2013.

Inclusion criteria were (1) age ≥18 years, (2) acute STEMI and cardiogenic shock at presentation and (3) MVD treated with primary PCI. Exclusion criteria were (1) missing initial vital signs information and (2) a non-STEMI final diagnosis. Among

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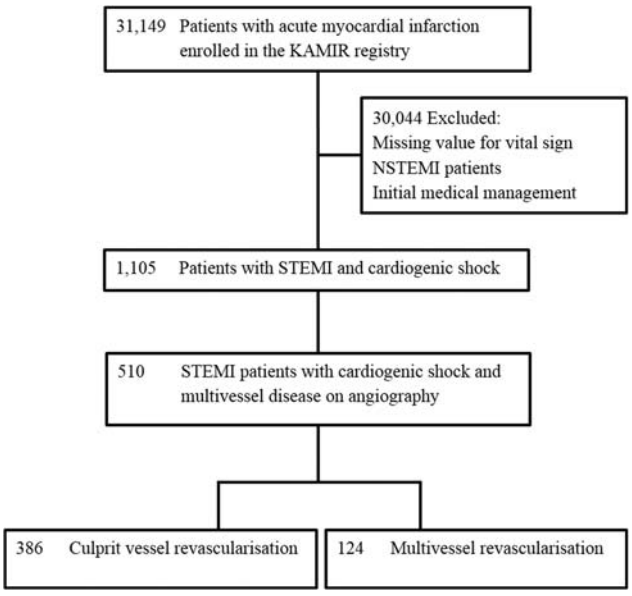

Figure 1 Study flowchart. KAMIR, the Korean acute myocardial infarction registry; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.

31 149 registered patients, 1105 had STEMI and cardiogenic shock; 510 with angiographically confirmed MVD were finally included in the study (figure 1). Subjects were divided into culprit vessel and multivessel revascularisation groups, according to the number of vessels treated.

PCI procedure

Coronary interventions were performed according to current practice guidelines. Any type of stent could be used, without restriction. The decision to perform PCI for non-culprit vessel lesions was left to the operators.

Definitions and outcomes

Cardiogenic shock was defined as[3] systolic blood pressure <90 mm Hg for >30 min or the need for supportive management to maintain systolic blood pressure ≥90 mm Hg and evidence of end-organ hypoperfusion (cool extremities, urine output <30 mL/h or altered mental status). STEMI was defined as ECG findings of ST-segment elevation ≥2 mm in two or more contiguous leads, new onset left bundle branch block or a posterior infarction with anterior ST-segment depression and at least one culprit vessel lesion on angiography. MVD was defined as the presence of an additional ≥50% diameter stenosis in at least one major non-culprit vessel on angiography. Multivessel revascularisation was defined as PCI of significant stenosis in a non-culprit vessel during admission. Procedural success was defined as thrombolysis in myocardial infarction (TIMI) flow grade 3 in the infarct-related artery and <30% diameter residual stenosis in the treated segment at the end of the procedure.

Table 1 Baseline clinical characteristics

	Culprit vessel revascularisation (n=386)	Multivessel revascularisation (n=124)	p Value
Age (years)	68.0 (57.0–76.0)	65.5 (55.0–75.0)	0.176
Male sex	254 (65.8)	88 (71)	0.287
Body mass index (kg/m^2)	23.0 (21.0–26.0)	24.0 (22.0–26.0)	0.343
Past medical conditions			
Ischaemic heart disease	50 (13.1)	18 (14.5)	0.686
Diabetes mellitus	88 (23.3)	31 (25.6)	0.599
Hypertension	208 (54.5)	65 (53.7)	0.888
Dyslipidaemia	32 (9.7)	11 (9.8)	0.969
Current smoker	178 (46.6)	59 (47.6)	0.837
Family history of ischaemic heart disease	22 (6.8)	6 (5.6)	0.657
Cardiopulmonary resuscitation at emergency department	16 (4.2)	4 (3.3)	0.650
Systolic blood pressure at presentation (mm Hg)	80.0 (70.0–90.0)	80 (73.0–90.0)	0.282
Heart rate at presentation (beats/min)	62.0 (48.0–80.0)	66.0 (50.0–81.0)	0.426
Door-to-balloon time (min)	69.0 (50.0–94.5)	75.0 (56.5–100.0)	0.121
Laboratory findings			
Serum glucose (mg/dL)	159.0 (132.0–195.0)	150.0 (128.0–194.0)	0.181
Peak troponin I (ng/mL)	35.0 (6.0–67.0)	26.5 (6.0–76.0)	0.735
Serum creatinine (mg/dL)	1.0 (1.0–1.0)	1.0 (1.0–1.0)	0.496
NT-pro-B-type natriuretic peptide (ng/mL)	573.0 (102.0–2463.0)	675.0 (142.0–2634.0)	0.743
Low-density lipoprotein cholesterol (mg/dL)	102.0 (81.0–132.0)	105.0 (84.0–129.0)	0.843
High-density lipoprotein cholesterol (mg/dL)	39.0 (34.0–46.0)	41.0 (35.0–50.0)	0.139
Left ventricular EF during hospitalisation (%)	50.0 (43.0–58.0)	50.0 (39.0–60.0)	0.917
Medications at discharge			
Aspirin	319 (98.2)	109 (99.1)	0.500
Clopidogrel	320 (98.8)	109 (99.1)	0.782
β-adrenergic blockers	122 (45)	45 (49.5)	0.463
Angiotensin-converting enzyme inhibitors	212 (79.1)	70 (78.7)	0.928
Angiotensin receptor blockers	47 (28)	27 (49.1)	0.004
Spironolactone	22 (14.2)	11 (22)	0.192
Statins	255 (91.4)	87 (87)	0.614

Data are shown as medians (25th–75th IQR) or numbers and percentages.

Primary outcomes were inhospital mortality and all-cause death during follow-up. Secondary outcomes included cardiac death, recurrent myocardial infarction (MI), any revascularisation and major adverse cardiac events (MACE), consisting of all-cause death, recurrent MI and any revascularisation during follow-up.

Statistics

Continuous variables were compared using the Student's t test or the Wilcoxon rank-sum test, while categorical variables were analysed using the χ^2 or Fisher's exact test. Unadjusted cumulative event rates were estimated by the Kaplan–Meier method and compared by log-rank and Wilcoxon tests.

To reduce the impact of treatment selection bias and potential confounding factors, weighted Cox proportional hazard models with robust SEs were used to derive HRs of outcomes between culprit vessel and multivessel revascularisations. Weighted Cox models were constructed using the inverse probability of treatment weighting (IPTW) and adjusting IPTW approach.[11] In this model, weights were stabilised by marginal probability for both groups. Stabilised weights for patients undergoing culprit vessel revascularisation were the product of the marginal probability for the culprit vessel revascularisation group and the inverse of (1-propensity score), while stabilised weights for patients undergoing multivessel revascularisation were the product of the marginal probability for the multivessel revascularisation group and the inverse of the propensity score.[12] Adjusting IPTW (<10) was used to reduce overestimated probability of variables for weighted Cox models. Variables used for weighted Cox models included age, sex, body mass index, cardiopulmonary resuscitation on arrival, initial systolic blood pressure, initial heart rate, overt pulmonary oedema, ischaemic heart disease, hypertension, diabetes mellitus, smoking, number of diseased vessels, infarct-related artery, preprocedural and postprocedural TIMI flow grades, stent length, stent diameter, number of implanted stents, result of PCI, use of an intra-aortic balloon pump and serum glucose and creatinine levels. The Kaplan–Meier method was used repeatedly to estimate clinical outcome incidences between groups in weighted Cox models. Logistical regression was used to identify independent predictors of multivessel revascularisation. Covariates were the same variables used for weighted Cox models.

A multiple Cox regression analysis was performed to predict risk factors of inhospital mortality and all-cause death during follow-up using baseline and angiographic characteristics associated with inhospital mortality and all-cause death in the simple Cox regression analysis ($p<0.1$) with >90% data availability.

Two-sided p values <0.05 were considered statistically significant. Analyses were performed with R V.3.1.1 using freely distributed statistical packages.

RESULTS

Baseline clinical and procedural characteristics

Three-hundred and eighty-six patients (75.7%) underwent culprit vessel revascularisation and 124 (24.3%) underwent multivessel revascularisation. Baseline and procedural characteristics are shown in tables 1 and 2.

Table 2 Procedural characteristics

	Culprit vessel revascularisation (n=386)	Multivessel revascularisation (n=124)	p Value
Extent of diseased vessel			0.315
Two-vessel disease	219 (56.7)	60 (48.4)	
Three-vessel disease	154 (39.9)	57 (46)	
Left main and other vessel disease	10 (2.6)	6 (4.8)	
Infarct-related artery			0.001
Left anterior descending	100 (25.9)	39 (31.5)	
Left circumflex	29 (7.5)	20 (16.1)	
Right coronary	254 (65.8)	61 (49.2)	
Left main tract	3 (0.8)	3 (2.4)	
Preprocedural TIMI flow grade*			0.023
0	274 (72.7)	69 (58.5)	
1	29 (7.7)	17 (14.3)	
2	35 (9.3)	16 (13.6)	
3	39 (10.3)	16 (13.6)	
Number of treated vessels	1.00	2.17	<0.001
Number of stents	1.32±0.60	2.38±1.09	<0.001
Use of drug-eluting stent	318 (82.4)	103 (83.1)	0.962
Stent diameter (mm)†	3.2±0.5	3.1±0.4	0.047
Stent length (mm)†	25.8±6.4	24.9±5.7	0.159
Use of intra-aortic balloon pump	63 (16.3)	23 (18.5)	0.811
Successful PCI	367 (95.1)	120 (96.8)	0.432
Postprocedural TIMI flow grade*			0.734
0	2 (0.5)	0 (0)	
1	6 (1.6)	3 (2.5)	
2	36 (9.8)	8 (6.8)	
3	324 (88.0)	107 (90.7)	

Data are shown as mean±SD or numbers and percentages.
*Percentages are calculated for available data only.
†Data from infarct-related artery intervention only.
PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

Overall, there was a similar prevalence of cardiovascular risk factors and history of coronary artery disease between the groups. Initial systolic blood pressure and heart rates were also similar. Both groups were treated similarly at discharge with antiplatelet therapy, β-blockers, angiotensin-converting enzyme inhibitors and statins, and vessel disease extent on coronary angiography was similar. The right coronary artery as the infarct-related artery was more common in patients undergoing culprit vessel revascularisation compared with those undergoing multivessel revascularisation (65.8% vs 49.2%), for whom the left anterior descending artery as the infarct-related artery was more common (25.9% vs 31.5%). Preprocedural TIMI flow grade was significantly different between groups, but there was no difference in successful PCI and final postprocedural TIMI flow grade. The use of intra-aortic balloon pumps was similar between groups (16.3% vs 18.5%).

Unadjusted clinical outcomes

Death within 24 h of hospitalisation occurred in 19 (3.7%) patients. Inhospital mortality was 13.5% (69 patients), with no group differences (14.5% vs 10.5%, log-rank p=0.252; figure 2A). Unadjusted risks of inhospital mortality did not differ significantly between groups (HR 0.704, 95% CI 0.385 to 1.288, p=0.255; table 3).

Most patients (94.1%) were admitted to the coronary care unit, where they stayed for a median 3 days (IQR 2–5 days). There were no group differences in ventricular tachycardia/fibrillation incidence (10.9% vs 9.7%, p=0.705), cerebrovascular accident (0.5% vs 0.8%, p=0.715), CIN (1.3% vs 1.6%, p=0.791), major bleeding (1.0% vs 0.0%, p=0.255) or multiorgan failure (1.0% vs 1.6%, p=0.604) during hospitalisation.

All-cause death and MACE occurred in 85 (16.7%) and 122 patients (23.9%), respectively, during a median follow-up of 194 days (IQR 14–374 days). There were no significant group differences in unadjusted incidences of all-cause death (17.9% vs 12.9%, log-rank p=0.181; figure 2A), recurrent MI (0.8% vs 2.4%, log-rank p=0.185), any revascularisation (6.2% vs 7.3%, log-rank p=0.984) or MACE (24.4% vs 22.6%, log-rank p=0.491). Unadjusted risks of all-cause death, cardiac death, recurrent MI, any revascularisation and MACE did not differ significantly between groups (table 3).

Adjusted clinical outcomes

Weighted Cox regression analysis using the IPTW method was performed to adjust for possible confounding factors. The c-statistic for the IPTW model was 0.87. Adjusted incidences of inhospital mortality (2.4% vs 9.3%, p<0.001) and all-cause death during follow-up (4.8% vs 13.1%, p<0.001) were significantly lower in the multivessel versus culprit vessel revascularisation group based on IPTW-adjusted survival curves (figure 2B). Adjusted incidences of cardiac death (4.8% vs 9.7%, p=0.002) and MACE (18.1% vs 20.3%, p=0.026) were significantly lower in the multivessel versus culprit vessel revascularisation group (figure 3A, D). The adjusted incidence of recurrent MI was significantly higher (3.6% vs 0.4%, p=0.017) in the multivessel revascularisation group, while the adjusted incidence of

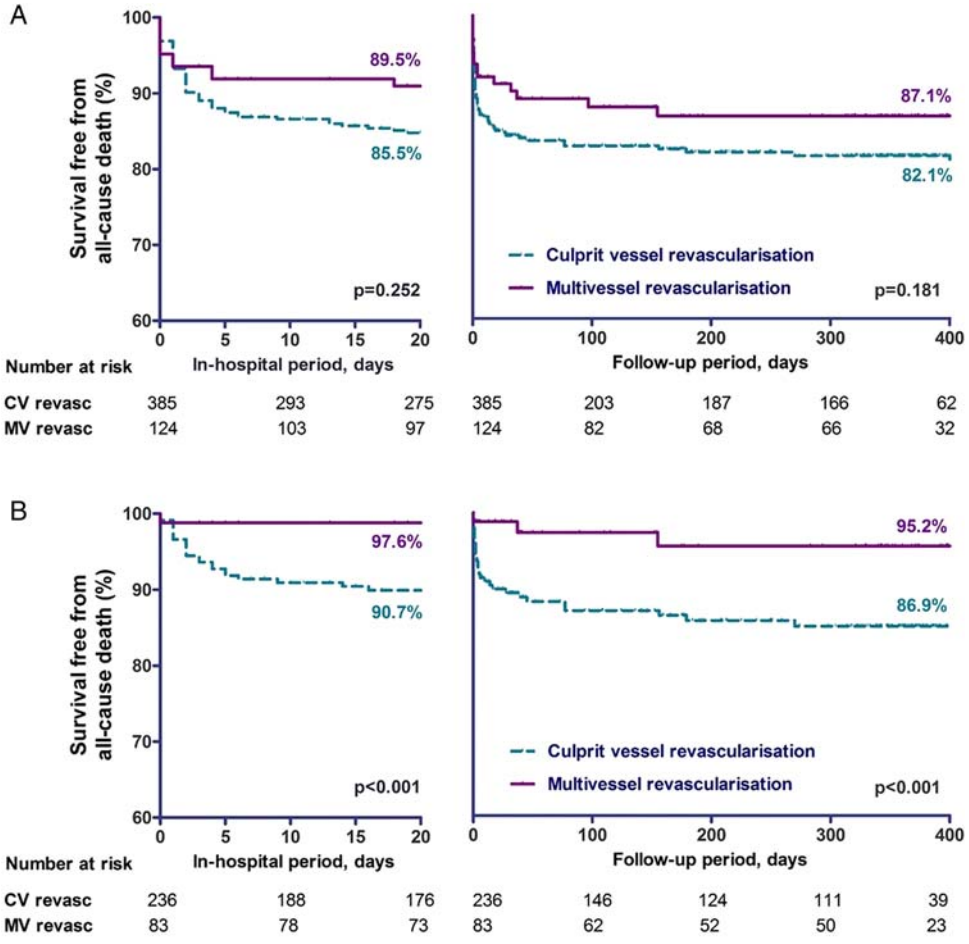

Figure 2 Survival curves free from inhospital mortality and all-cause death during follow-up between culprit vessel (CV) and multivessel (MV) revascularisation in the overall population (A) and after adjusting for the inverse probability of treatment weight (B).

Table 3 Outcome following multivessel revascularisation compared with culprit vessel revascularisation

	Crude		Inverse probability of treatment weighted		Adjusting inverse probability of treatment weighted	
	HR (95% CI)	p Value	HR (95% CI)	p Value	HR (95% CI)	p Value
Inhospital mortality	0.704 (0.385 to 1.288)	0.255	0.203 (0.088 to 0.464)	<0.001	0.263 (0.149 to 0.462)	<0.001
All-cause death	0.691 (0.401 to 1.19)	0.184	0.601 (0.374 to 0.964)	0.034	0.400 (0.264 to 0.606)	<0.001
Cardiac death	0.924 (0.527 to 1.619)	0.783	0.766 (0.4656 to 1.261)	0.295	0.510 (0.329 to 0.790)	0.002
Recurrent myocardial infarction	2.815 (0.568 to 13.95)	0.205	2.25 (0.418 to 12.11)	0.345	4.307 (1.152 to 16.11)	0.03
Any revascularisation	1.008 (0.991 to 1.468)	0.983	0.550 (0.286 to 1.057)	0.073	0.985 (0.630 to 1.54)	0.949
Target lesion revascularisation	2.293 (0.769 to 6.831)	0.136	1.052 (0.381 to 2.905)	0.922	2.055 (0.979 to 4.313)	0.056
Target vessel revascularisation	2.785 (0.174 to 44.53)	0.469	3.17 (0.355 to 28.22)	0.301	4.301 (0.715 to 25.84)	0.111
Non-target vessel revascularisation	0.358 (0.081 to 1.567)	0.173	0.170 (0.045 to 0.622)	0.007	0.327 (0.153 to 0.698)	0.003
Major adverse cardiac events	0.861 (0.564 to 1.314)	0.49	0.642 (0.443 to 0.930)	0.019	0.728 (0.55 to 0.965)	0.026

any revascularisation did not differ between groups (9.6% vs 7.2%, p=0.949; figure 3B, C). Multivessel revascularisation was associated with significantly lower adjusted risks of inhospital mortality and all-cause and cardiac death during follow-up (table 3) but a significantly higher adjusted risk of recurrent MI. While the adjusted risk of any revascularisation did not differ between groups, multivessel revascularisation was marginally associated with a higher adjusted risk of target lesion revascularisation (p=0.056) and a significantly lower adjusted risk of non-target vessel revascularisation. Multivessel revascularisation was associated with a significantly lower adjusted risk of MACE.

Predictors of multivessel revascularisation during primary PCI

Multivariable analysis showed that multivessel revascularisation during primary PCI was associated with the presence of overt pulmonary oedema (OR 2.388, 95% CI 1.252 to 4.557, p=0.008) and non-right coronary artery infarct-related artery (OR 0.457, 95% CI 0.228 to 0.914, p=0.027).

Association of baseline characteristics and treatment with inhospital mortality and all-cause death during follow-up

The simple Cox regression analysis of the overall cohort showed that old age, female sex, cardiopulmonary resuscitation on arrival, overt pulmonary oedema, history of hypertension, diabetes mellitus, postprocedural TIMI flow grade 2–3, intra-aortic balloon pump, low left ventricular EF, high serum glucose or creatinine at presentation and inhospital complications, such as ventricular tachycardia/fibrillation, CIN and major bleeding, were associated with inhospital mortality and all-cause death during follow-up (tables 4 and 5). After adjustment, old age, postprocedural TIMI flow grade 2–3, intra-aortic balloon pump, low left ventricular EF, high serum creatinine and CIN had a

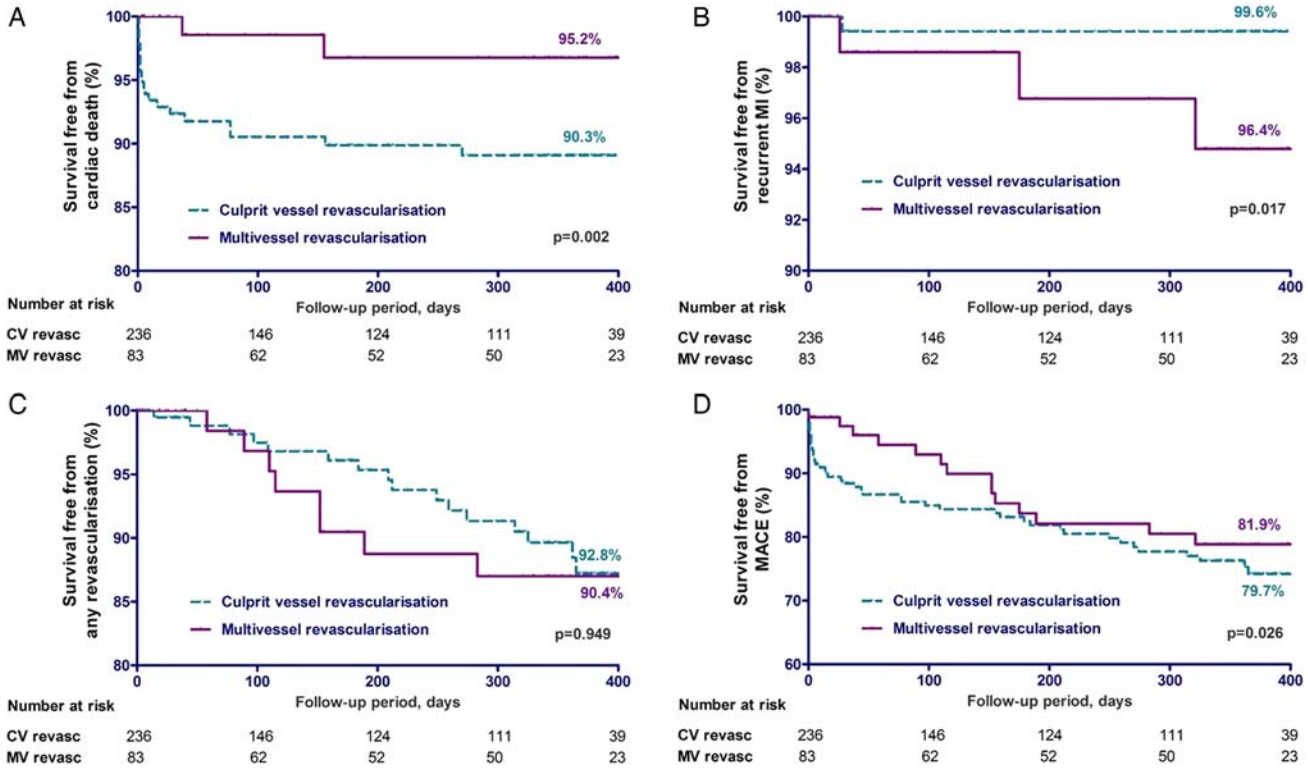

Figure 3 The inverse probability of treatment weight-adjusted survival curves free from cardiac death (A), recurrent myocardial infarction (MI; B), any revascularisation (C) and major adverse cardiac events (MACE; D) between culprit vessel (CV) and multivessel (MV) revascularisation.

Table 4 Predictors of inhospital mortality

	Simple Cox regression		Multiple Cox regression	
Variable	**HR (95% CI)**	**p Value**	**HR (95% CI)**	**p Value**
Age (1-year increase)	1.073 (1.049 to 1.098)	<0.001	1.064 (1.019 to 1.110)	0.005
Female sex	2.693 (1.676 to 4.328)	<0.001		
Cardiopulmonary resuscitation	4.165 (2.065 to 8.400)	<0.001		
Systolic blood pressure	0.993 (0.984 to 1.002)	0.108		
Overt pulmonary oedema	3.332 (1.981 to 5.603)	<0.001		
Ischaemic heart disease	0.515 (0.207 to 1.282)	0.154		
Hypertension	1.688 (1.010 to 2.822)	0.046		
Diabetes mellitus	1.722 (1.028 to 2.884)	0.039		
Dyslipidaemia	0.175 (0.024 to 1.270)	0.085		
Preprocedural TIMI flow grade 0–1	1.146 (0.844 to 1.556)	0.381		
Postprocedural TIMI flow grade 2–3	0.228 (0.136 to 0.384)	<0.001	0.242 (0.085 to 0.685)	0.008
Use of intra-aortic balloon pump	5.000 (3.116 to 8.023)	<0.001	3.286 (1.350 to 7.997)	0.009
Low left ventricular EF	0.918 (0.890 to.0948)	<0.001	0.938 (0.903 to 0.974)	0.001
Serum glucose level	1.006 (1.004 to 1.008)	<0.001		
Serum creatinine level	1.577 (1.372 to 1.813)	<0.001	1.816 (1.249 to 2.639)	0.002
Ventricular tachycardia/fibrillation*	3.636 (2.143 to 6.169)	<0.001		
Contrast-induced nephropathy*	8.391 (3.625 to 19.422)	<0.001	6.165 (1.977 to 19.222)	0.002
Major bleeding*	4.236 (1.037 to 17.297)	0.044		

*Complications during hospitalisation.
TIMI, thrombolysis in myocardial infarction.

significant association with inhospital mortality and all-cause death during follow-up.

DISCUSSION

Our major finding was that adjusted inhospital mortality and all-cause death during follow-up were significantly lower following multivessel versus culprit vessel revascularisation during primary PCI in patients with STEMI with cardiogenic shock and MVD. Additionally, multivessel revascularisation was associated with a lower adjusted risk of the composite of all-cause death, recurrent MI and any revascularisation. More patients with overt pulmonary oedema at presentation and non-right coronary artery infarct-related artery on angiography underwent multivessel revascularisation during primary PCI. Old age, procedural failure, use of an intra-aortic balloon pump, low left ventricular EF, high serum creatinine and CIN were independent predictors of inhospital mortality and all-cause death during follow-up.

Results of multivessel PCI in patients with STEMI with cardiogenic shock are somewhat conflicting. Cavender *et al*[10] reported that multivessel compared with culprit vessel revascularisation during primary PCI was associated with higher inhospital mortality (36.5% vs 27.8%, OR 1.54, 95% CI 1.22 to

Table 5 Predictors of all-cause death during follow-up

	Simple Cox regression		Multiple Cox regression	
Variable	**HR (95% CI)**	**p Value**	**HR (95% CI)**	**p Value**
Age (1-year increase)	1.071 (1.049 to 1.093)	<0.001	1.079 (1.040 to 1.120)	<0.001
Female sex	2.131 (1.392 to 3.261)	<0.001		
Cardiopulmonary resuscitation	3.445 (1.723 to 6.888)	<0.001		
Systolic blood pressure	0.994 (0.986 to 1.003)	0.182		
Overt pulmonary oedema	3.387 (2.124 to 5.401)	<0.001		
Ischaemic heart disease	0.859 (0.443 to 1.665)	0.653		
Hypertension	1.611 (1.019 to 2.547)	0.041		
Diabetes mellitus	1.632 (1.021 to 2.607)	0.041		
Dyslipidaemia	0.563 (0.205 to 1.545)	0.265		
Preprocedural TIMI flow 0–1	1.668 (0.905 to 3.077)	0.101		
Postprocedural TIMI flow 2–3	0.264 (0.162 to 0.428)	<0.001	0.336 (0.142 to 0.793)	0.013
Use of intra-aortic balloon pump	4.451 (2.891 to 6.853)	<0.001	2.531 (1.246 to 5.141)	0.010
Low left ventricular EF	0.932 (0.909 to 0.955)	<0.001	0.948 (0.921 to 0.976)	<0.001
Serum glucose level	1.005 (1.003 to 1.007)	<0.001		
Serum creatinine level	1.597 (1.403 to 1.819)	<0.001	1.784 (1.326 to 2.402)	<0.001
Ventricular tachycardia/fibrillation*	3.297 (2.010 to 5.407)	<0.001		
Contrast-induced nephropathy*	9.078 (4.167 to 19.778)	<0.001	5.928 (2.149 to 16.355)	0.001
Major bleeding*	3.699 (0.909 to 15.056)	0.068		

*Complications during hospitalisation.
TIMI, thrombolysis in myocardial infarction.

1.95) among patients with STEMI with cardiogenic shock (n=3087) from the National Cardiovascular Data Registry (NCDR), whereas an analysis by Bauer *et al*[13] found that multivessel revascularisation had no impact on inhospital mortality (OR 1.28, 95% CI 0.72 to 2.28, p=0.07) after adjustment for confounding factors. These results might have been due to the increased risk of procedural-related complications, such as bleeding or renal failure, distal embolisation associated with PCI or the loss of collateral flow to other coronary territories.[14] A recent study of multivessel revascularisation in patients with STEMI with cardiogenic shock and resuscitated cardiac arrest showed that multivessel revascularisation may improve 6-month survival, with a reduction in recurrent arrest and death due to shock.[15] In our study, a consistent survival benefit was observed in patients who had undergone multivessel revascularisation when multiple statistical analyses were performed.

There is no clear evidence regarding the reason for the survival advantage of multivessel revascularisation. However, pathological studies showed that multiple thrombi in patients with STEMI were seen in culprit lesions and in non-culprit lesions.[16] The survival benefit is directly related to myocardial ischaemia degree and the extent of LV dysfunction.[3] Non-culprit vessel lesion revascularisation in patients with MVD can decrease myocardial ischaemia by enhancing perfusion of the peri-infarct area, eventually improving LV function, perhaps partially explaining the multivessel revascularisation survival advantage.

Several studies showed that early revascularisation, LV function, coronary stenting, age and successful revascularisation (TIMI flow grade >2) were most important for improving clinical outcomes in patients with cardiogenic shock.[3 17 18] We here showed that intra-aortic balloon pump use, baseline serum creatinine and CIN independently predicted inhospital mortality and all-cause death during follow-up.

Mortality in patients with STEMI with cardiogenic shock is usually high, but is particularly high (up to 50%) in patients resuscitated from cardiac arrest.[19] Inhospital mortality in our cohort was relatively lower (13.5%) compared with 29% among 3087 shock patients from the NCDR,[10] which may be due to different patient clinical and angiographic characteristics and management patterns. A higher rate (62%) of right coronary artery as the infarct-related artery with a lower rate of overt pulmonary oedema compared with ~30% in the NCDR may also partly explain the lower mortality in our cohort. However, multivariate analysis did not confirm that these variables were associated with mortality risk.

Limitations

The present study was registry-based and limited by selection bias. Attending operators performed either culprit vessel only or multivessel revascularisation during primary PCI. Anatomical and procedural factors, such as lesion difficulty (including culprit and non-culprit vessels), expected procedure time, and operator's expertise, could have influenced the results. As this study was not randomised, unrecognised confounding variables may have influenced the results, although we used weighted Cox regression to minimise such confounding. The lack of detailed information regarding staged PCI for non-culprit lesions and its timing is another limitation, because they were not included as prespecified variables in the original registry. Chronic total occlusion in non-culprit vessels was not prespecified, and its influence on outcomes could not be evaluated. Finally, follow-up was short (median, 194 days), and the impact on outcomes over a longer period remains to be clarified.

Conclusions

This study showed that multivessel compared with culprit vessel revascularisation during primary PCI was associated with better outcomes in patients with STEMI with cardiogenic shock and MVD, supporting current revascularisation guidelines.

Key messages

What is already known on this subject?

Patients with acute ST-elevation myocardial infarction (STEMI) suffering from cardiogenic shock have high rates of morbidity and mortality during hospitalisation. Current guidelines recommend culprit vessel revascularisation during primary percutaneous coronary intervention in patients with STEMI, but multivessel revascularisation can be performed in patients with STEMI and persistent cardiogenic shock.

What might this study add?

Compared with culprit vessel revascularisation, multivessel revascularisation demonstrated a significantly lower risk of inhospital mortality and all-cause death, mainly because of fewer cardiac deaths. In addition, multivessel revascularisation significantly decreased the risk of the composite endpoint of all-cause death, recurrent myocardial infarction and any revascularisation during follow-up.

How might this impact on clinical practice?

The results of this study suggest that multivessel revascularisation should have a role in the revascularisation strategy for patients with STEMI with cardiogenic shock, based on the haemodynamic status, the degree of stenosis, procedural difficulty and the extent of myocardial ischaemia for non-culprit lesions.

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Patient consent Obtained.

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Heart

Culprit or multivessel revascularisation in ST-elevation myocardial infarction with cardiogenic shock

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