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Multivessel Versus Culprit-Vessel Percutaneous Coronary Intervention in Cardiogenic Shock

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ABSTRACT

OBJECTIVES This study sought to compare outcomes of patients enrolled in the NCSI (National Cardiogenic Shock Initiative) trial who were treated using a revascularization strategy of percutaneous coronary intervention (PCI) of multivessel PCI (MV-PCI) versus culprit-vessel PCI (CV-PCI).

BACKGROUND In patients with multivessel disease who present with acute myocardial infarction and cardiogenic shock (AMICS), intervening on the nonculprit vessel is controversial. There are conflicting published reports and lack of evidence, particularly in patients treated with early mechanical circulatory support (MCS).

METHODS From July 2016 to December 2019, patients who presented with AMICS to 57 participating hospitals were included in this analysis. All patients were treated using a standard shock protocol emphasizing early MCS, revascularization, and invasive hemodynamic monitoring. Patients with multivessel coronary artery disease (MVCAD) were analyzed according to whether CV-PCI or MV-PCI was undertaken during the index procedure.

RESULTS Of 198 patients with MVCAD, 126 underwent MV-PCI (64%) and 72 underwent CV-PCI (36%). Demographics between the cohorts were similar with respect to age, sex, history of diabetes, prior PCI or coronary artery bypass grafting, and prior history of myocardial infarction. Patients who underwent MV-PCI had a trend toward more severe impairment of cardiac output and worse lactate clearance on presentation, and cardiac performance was significantly worse at 12 h. However, 24 h from PCI, the hemometabolic derangements were similar. Survival and rates of acute kidney injury were not significantly different between groups (69.8% MV-PCI vs. 65.3% CV-PCI; $p = 0.51$; and 29.9% vs. 34.2%; $p = 0.64$, respectively).

CONCLUSIONS In patients with MVCAD presenting with AMICS treated with early MCS, revascularization of nonculprit lesions was associated with similar hospital survival and acute kidney injury when compared with culprit-only PCI. Selective nonculprit PCI can be safety performed in AMICS in patients supported with mechanical circulatory support. (J Am Coll Cardiol Intv 2020;13:1171–8) © 2020 by the American College of Cardiology Foundation.

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ABBREVIATIONS AND ACRONYMS

ACC = American College of Cardiology

AHA = American Heart Association

AKI = acute kidney injury

AMI = acute myocardial infarction

AMICS = acute myocardial infarction cardiogenic shock

CAD = coronary artery disease

CPO = cardiac power output

CS = cardiogenic shock

CTO = chronic total occlusion

CV-PCI = culprit-vessel percutaneous coronary intervention

MCS = mechanical circulatory support

MVCAD = multivessel coronary artery disease

MV-PCI = multivessel percutaneous coronary intervention

PCI = percutaneous coronary intervention

RCT = randomized control trial

SCAI = Society for Cardiovascular Angiography and Interventions

STEMI = ST-segment elevation myocardial infarction

TIMI = Thrombolysis In Myocardial Infarction

The incidence of cardiogenic shock (CS) in acute myocardial infarction (AMI) ranges from 5% to 10% and is associated with a significantly elevated mortality (1,2). It has been reported that 50% to 80% of patients with ST-segment elevation myocardial infarction (STEMI) and CS have multivessel coronary artery disease (MVCAD) (3,4), which is also associated with increased mortality (5). There have been conflicting studies on the optimal revascularization strategy in patients with MVCAD presenting with AMI and CS (AMICS), and presently. there is no consensus for multivessel percutaneous coronary intervention (MV-PCI) versus culprit-vessel percutaneous coronary intervention (CV-PCI). The 2015 American College of Cardiology (ACC) and American Heart Association (AHA) Guidelines Focused Update support treatment of noninfarct arteries during STEMI in hemodynamically stable patients, changing a Class III recommendation from 2013 to a Class IIb recommendation (Level of Evidence: B-R) (6). In the setting of cardiogenic shock, the 2013 ACC/AHA guidelines (7) recommended considering percutaneous coronary intervention (PCI) of large noninfarct arteries in an attempt to improve hemodynamic stability. Similarly, the 2017 European Society of Cardiology guidelines recommend considering noninfarct artery PCI in patients with cardiogenic shock during the index PCI (8,9).

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The CULPRIT-SHOCK (PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock) trial (10), a large multicenter randomized control trial (RCT), demonstrated improved survival at 30 days when using a CV-PCI strategy compared with complete revascularization during the index procedure. Improved survival was limited to the first 30 days, after which there was no significant difference in survival from 30 days to 1 year. CV-PCI was accompanied by an increased risk of repeat revascularization and hospitalization for heart failure within the first year of follow-up despite 17.4% of patients undergoing staged nonculprit lesion PCI within 30 days of the index PCI. Furthermore, a meta-analysis of observational studies and another meta-analysis including observational and randomized studies showed no difference in short- or long-term outcomes in CV- versus MV-PCI for STEMI CS (4,11). Recently, studies evaluating the use of mechanical circulatory support (MCS) have shown a decrease in AMICS mortality from 50% to 25% to 30% (12), along with decreasing rates of renal replacement therapy (13). To date, studies comparing revascularization strategies in patients with AMICS and MVCAD with the use of MCS have been limited. The NCSI (National Cardiogenic Shock Initiative; NCT03677180) trial is a single-arm, prospective, multicenter study to assess clinical outcomes associated with the use of a shock protocol emphasizing early MCS and PCI in patients presenting with AMICS. Therefore, we sought to compare outcomes of patients enrolled in the NCSI trial who were treated using a revascularization strategy of MV- versus CV-PCI.

METHODS

THE NCSI TRIAL. The NCSI trial encompasses 57 hospitals in the United States (Supplemental Appendix) who have adopted a mutually agreed-upon algorithm for the treatment of AMICS based on previously established best practices that include: 1) early identification and catheterization laboratory activation for patients presenting with AMICS; 2) early use of MCS (ideally before PCI); and 3) routine use of invasive hemodynamic monitoring with pulmonary artery catheters to guide management of MCS and use of inotropes. The diagnosis of AMI was confirmed by

institutional research grants/speaker fees/consulting honoraria from Abbott, Abiomed, Boston Scientific, Medtronic, LivaNova, MD Start, and Precardia. Dr. Riley has received consultant and speaker fees from Boston Scientific, Medtronic, and Asahi. Dr. Tehrani has received consultant/speaker fees from Abiomed and Medtronic. Dr. Truesdell has received consultant/speaker fees from Abiomed. Dr. Lasorda has received speaker fees from Abiomed. Dr. Bharadwaj has received speaker and proctor fees from Abiomed. Dr. Hanson has received speaker fees from Abiomed. Dr. O'Neill has received consultant fees from Abiomed, Boston Scientific, and Medtronic; speaker fees from Abiomed and Medtronic; and grant/research support from St. Jude Medical, Edwards Lifesciences, and Abiomed. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Interventions* author instructions page.

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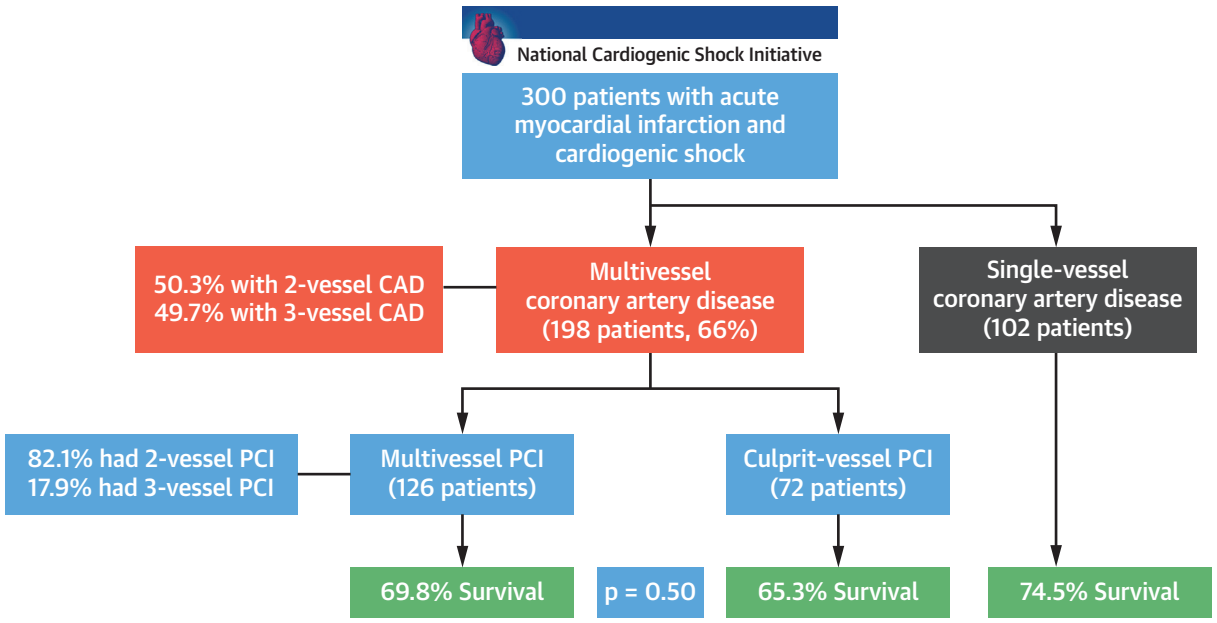
CENTRAL ILLUSTRATION **Flowchart With Patient Selection**



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A total of 300 patients with acute myocardial infarction cardiogenic shock were enrolled in the study period, of which 198 patients had multivessel CAD, 126 underwent multivessel PCI, and 71 culprit-vessel PCI. Survival rates were 69.8% and 65.3%, respectively (p = 0.50). A total of 102 patients had only 1-vessel CAD, and their survival rate was 74.5%. CAD = coronary artery disease; PCI = percutaneous coronary intervention.

electrocardiographic changes indicative of presumed new ischemia (ST-T changes), detection of elevated cardiac biomarkers, or angiographic findings of an infarct-related artery on coronary angiogram in the presence of ischemic symptoms. Cardiogenic shock was defined as the presence of at least 2 of the following: 1) hypotension (systolic blood pressure <90 mm Hg, or inotropes/vasopressors to maintain systolic blood pressure >90 mm Hg); 2) signs of end-organ hypoperfusion (cool extremities, oliguria or anuria, or elevated lactate levels); and 3) hemodynamic criteria of cardiogenic shock (cardiac index <2.2 l/min/m^2 or cardiac power output [CPO] <0.6 W). All patients underwent MCS placement using an Impella device (Abiomed, Danvers, Massachusetts), and those who underwent intra-aortic balloon pump were not included in the trial. Operators were highly encouraged to follow the treatment algorithm.

PATIENTS. Patients were enrolled from July 2016 through December 2019. Data were collected retrospectively. Institutional review board approval was obtained at each of the participating sites. Consent was obtained from patients, patient surrogates, or capturing of deidentified data for patients who did not survive and would not require follow-up according to local institutional review board requirements.

PERCUTANEOUS CORONARY INTERVENTION. MVCAD was reported by each individual operator, considering a stenosis of >70% as significant; the culprit vessel was also defined by each operator, and revascularization for nonculprit vessels with Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 was not protocol-mandated and was left to the discretion of the operator. PCI of chronic total occlusions (CTOs) in the emergent setting was discouraged.

OUTCOMES. The primary outcome was hospital survival. Secondary outcomes included rates of acute kidney injury (AKI) (defined as an increase in creatinine of 1.5 times from baseline, excluding patients with end-stage renal disease), and length of stay.

STATISTICAL ANALYSIS. Continuous variables were described using mean ± SD if normally distributed or medians and interquartile ranges if non-normal in distribution. Categorical variables were described a frequencies and percentages. Unadjusted comparisons of continuous variables employed Student's *t*-test or Mann-Whitney *U* rank sum tests, as appropriate, whereas chi-square or Fisher exact tests were

TABLE 1 Patient Demographics and Admission Characteristics

	Single-Vessel CAD (n =102)	All Multivessel CAD (N = 198)	Multivessel PCI (n = 126)	Culprit-Vessel PCI (n = 72)	p Value
Demographics					
Age, yrs	62.3 ± 13.3	64.2 ± 11.7	64.8 ± 11.8	63.3 ± 11.6	0.29
Males	30.4	79.8	81.8	76.4	0.37
Diabetes	32.4	55.3	55.4	55.1	0.97
End-stage renal disease	2.0	5.2	4.9	5.6	0.83
Chronic kidney disease	12.8	11.6	8.3	17.2	0.07
Heart failure, EF <50%	26.5	26.5	27.8	24.2	0.73
Prior coronary artery bypass graft	4.9	5.7	4.1	8.5	0.21
Prior percutaneous coronary intervention	22.6	25.4	23.7	28.2	0.50
Prior myocardial infarction	18.8	19.6	18.6	21.1	0.68
Cerebrovascular disease	6.9	11.1	11.7	10.1	0.75
Admission characteristics					
Patient transferred from another hospital	21.6	29.4	28.0	31.9	0.56
Support prior to transfer	28.6	17.5	17.1	18.2	0.44
Shock present on admission	62.4	69.4	71.2	66.2	0.47
Out-of-hospital arrest	29.3	44.6	44.4	44.8	0.76
In-hospital arrest	63.8	56.1	54.7	58.6	0.73
CPR at the time of Impella insertion	8.9	8.7	7.3	11.1	0.36
ST-segment elevation myocardial infarction	86.1	75.1	76.8	72.2	0.47
Non-ST-segment elevation myocardial infarction	13.9	24.9	23.2	27.8	
SCAI classification on admission					
Classic	62.8	58.6	58.5	56.9	0.21
Deteriorating	5.9	9.6	11.9	5.6	
Extremis	31.4	31.8	28.6	37.5	

Values are mean ± SD or %.

CAD = coronary artery disease; CPR = cardiopulmonary resuscitation; EF = ejection fraction; PCI = percutaneous coronary intervention; SCAI = Society of Cardiovascular Angiography and Interventions.

used where appropriate for categorical variables. Multivariate logistic regression was used to assess the independent association between CV- vs. MV-PCI and hospital survival. Variables used in the multivariate analysis included patient's baseline demographics and comorbidities. A backward elimination approach was used to select variables to include in the multivariate model, using a level of significance of $p < 0.20$. All statistical tests and/or confidence intervals were performed with a 2-sided p value = 0.05.

RESULTS

PATIENTS. The first 300 patients included in the NCSI database were evaluated for inclusion in this study (**Central Illustration**); 198 (66.0%) had MVCAD and were included in our analysis. Among the 198 patients with AMICS that had MVCAD, 126 patients (63.6%) underwent MV-PCI and 72 underwent CV-PCI. **Table 1** lists baseline demographics for patients enrolled in the NCSI trial, including the 2 groups compared in this study. The mean age for the entire group was 64.2 ± 11.7 years, 79.8% were male, 75.1% presented with STEMI, and 69.4% had CS on admission. When comparing baseline demographics between MVD cohorts, patients that underwent MV-PCI were older and more commonly male; they had a similar prevalence of diabetes and history of stroke, but lower prevalence of chronic kidney disease, prior coronary artery bypass grafting, prior PCI, and history of myocardial infarction compared with those that underwent CV-PCI. Cardiogenic shock was present on admission in 71.2% of patients in the MV-PCI cohort and in 66.2% of patients that underwent CV-PCI ($p = 0.47$); and 76.8% of patients in the MV-PCI cohort presented with STEMI, compared with 72.2% in the CV-PCI cohort ($p = 0.47$). Among patients in the MV-PCI cohort, 58.5% were Society for Cardiovascular Angiography and Interventions (SCAI) Stage C on presentation, 11.9% were SCAI Stage D, and 28.6% were SCAI Stage E. Similarly, in the CV-PCI cohort, 56.9% patients were SCAI Stage C, 5.6% Stage D, and 37.5% Stage E. No significant difference in the SCAI stages was observed between cohorts ($p = 0.21$).

PROCEDURAL CHARACTERISTICS. Procedural characteristics for patients in each revascularization strategy group are listed in **Table 2**. Impella was placed before PCI in the majority of patients in both

TABLE 2 Procedural Characteristics

	All Multivessel CAD (N = 198)	Multivessel PCI (n = 126)	Culprit-Vessel PCI (n = 72)	p Value
Impella insertion				
Pre-PCI	71.4	72.0	70.4	0.90
Intra-procedural	20.9	20.0	22.5	
Post-PCI	7.7	8.0	7.0	
RHC insertion				
Pre-Impella	30.6	29.8	31.9	0.42
Post-Impella	61.7	60.5	63.8	
RHC not performed	7.7	9.7	4.4	
Initial device used				
Impella 2.5	3.5	4.0	2.8	0.66
Impella CP	93.9	95.2	91.7	0.31
Impella access				
Femoral	98.5	98.4	98.6	0.90
Axillary	1.5	1.6	1.4	
PCI access				
Radial	20.7	19.1	23.6	0.45
Femoral	78.3	79.4	76.4	0.63
Thrombectomy used	23.6	26.6	18.3	0.19
Atherectomy used	6.7	8.9	2.9	0.10
Culprit vessel				
Left main	9.8	15.3	0.0	0.02
Left anterior descending	34.8	27.8	47.5	
Left circumflex	21.4	23.6	17.5	
Right coronary artery	30.4	29.2	32.5	
Ramus	2.7	4.2	0.0	
Number of diseased vessels				
2 vessels	50.3	50.8	49.3	0.42
3 vessels	49.7	49.2	50.7	
Number of vessels treated				
1 vessel	36.9	0.0	100.0	<0.001
2 vessels	51.8	82.1	0.0	
3 vessels	11.3	17.9	0.0	
Number of stents placed, mean	2.0	2.3	1.4	<0.001
Door-to-balloon time, min*	105	100	114	0.60
Door-to-support time, min*	106	99	119	0.18
TIMI flow grade pre-PCI, culprit vessel				
0	61.5	64.6	56.5	0.63
1	17.0	15.0	20.3	
2	12.1	10.6	14.5	
3	9.3	9.7	8.7	
TIMI flow grade post-PCI, culprit vessel				
0	1.6	1.7	1.4	0.28
1	1.1	0.8	1.4	
2	3.2	5.0	0.0	
3	94.2	92.4	97.2	
Contrast volume, ml	195 ± 91	205 ± 100	177 ± 66	0.22
Number of vasopressors before PCI				
0	33.5	33.6	33.3	0.25
1	47.7	46.0	50.8	
≥2	18.7	20.3	15.9	
Number of vasopressors post-PCI				
0	42.9	44.4	40.3	0.36
1	36.9	33.3	43.1	
≥2	20.2	22.2	16.7	

Values are % or mean ± SD, except as noted. *For patients presenting with ST-segment elevation myocardial infarction (mean minutes).
RHC = right heart catheterization; TIMI = Thrombolysis In Myocardial Infarction; other abbreviations as in Table 1.

TABLE 3 Hemodynamic and Laboratory Data Within the First 24 H

	Pre-MCS			12 H			24 H		
	MV-PCI	CV-PCI	p Value	MV-PCI	CV-PCI	p Value	MV-PCI	CV-PCI	p Value
HR, beats/min	99	95	0.48	91	88	0.24	93	87	0.06
SBP, mm Hg	95	98	0.23	103	110	0.02	102	108	0.11
DBP, mm Hg	62	62	0.64	73	76	0.12	66	70	0.06
PA sat, %	53	59	0.12	56	64	0.06	58	63	0.47
Cardiac output	3.8	4.0	0.48	4.2	4.8	0.08	4.9	5.2	0.56
LVEDP, mm Hg	30	30	0.85	-	-	-	-	-	-
CPO, W	0.64	0.71	0.86	0.79	0.91	0.03	0.9	0.9	0.14
Creatinine, mg/dl	1.72	1.90	0.77	1.98	1.96	0.11	1.9	2.4	0.99
Lactate, mg/dl	5.6	5.0	0.93	4.8	3.5	0.03	3.3	2.5	0.13

CPO = cardiac power output; CV-PCI = culprit-vessel percutaneous coronary intervention; DBP = diastolic blood pressure; LVEDP = left ventricular end-diastolic pressure; MCS = mechanical circulatory support; MV-PCI = multivessel percutaneous coronary intervention; PA sat = pulmonary artery saturation; SDP = systolic blood pressure.

groups (72.0% vs. 70.4%; p = 0.90). Right heart catheterization was performed in 90.3% of patients undergoing MV-PCI and 95.7% in those undergoing CV-PCI. There was a similar number of diseased vessels in both groups. There was no statistical difference in the contrast volume administered between groups (205 ± 100 ml vs. 177 ± 66 ml; p = 0.22). Furthermore, the use of vasopressors was reduced from 66% of patients requiring at least 1 vasopressor before PCI to 57% of patients requiring vasopressors post-PCI. It is important to highlight that both groups achieved >90% TIMI flow grade 3 post-revascularization.

Patient hemodynamics are shown in Table 3. Notably, the mean heart rate, blood pressure, cardiac output, CPO, pulmonary artery oxygen saturation, and left ventricular end-diastolic pressure were similar in both groups before initiation of MCS.

OUTCOMES. Primary and secondary outcomes are listed in Table 4. Survival was not significantly different between groups (69.8% for the MV-PCI cohort vs. 65.3% for the CV-PCI cohort; p = 0.51). Similarly, the rate of AKI was similar (29.9% vs. 34.2%; p = 0.64). The median hospital duration was 10 days (interquartile range: 5 to 16 days) for those undergoing MV-PCI and 8 days (interquartile range: 4 to 14 days) for patients with CV-PCI (p = 0.50). Among patients with 3-vessel coronary artery disease (CAD) and MV-PCI, those that underwent 2-vessel PCI (n = 35) had a 65.7% survival and those with 3-vessel PCI (n = 22) had a 72.7% survival.

TABLE 4 Primary and Secondary Outcomes

	Single-Vessel CAD (n = 102)	All Multivessel CAD (N = 198)	Multivessel PCI (n = 126)	Culprit-Vessel PCI (n = 72)	p Value
Survived hospitalization	74.5	68.2	69.8	65.3	0.51
Acute kidney injury*	20.9	31.4	29.9	34.2	0.64
Length of stay	8 (4–13)	9 (4–16)	10 (5–16)	8 (4 - 14)	0.50

Values are % or median (interquartile range). *Acute kidney injury was defined as an increase in creatinine ≥1.5 times from baseline; patients with end-stage renal disease were excluded.
Abbreviations as in Table 1.

DISCUSSION

The optimal revascularization strategy for patients who present with MVCAD and AMICS is unclear, and practice varies among centers in the United States. In this single-arm observational study from a large national database on AMICS, we analyzed revascularization strategies among 198 patients with MVCAD presenting with AMI complicated by CS. Key findings include: 1) in the setting of early MCS use, no difference in hospital survival was observed between patients undergoing MV-PCI versus CV-PCI; 2) no difference in survival was observed in any relevant patient subgroups; and 3) AKI rates were similar between patients who underwent MV-PCI versus CV-PCI.

Recent RCTs, such as the PRAMI (Preventive Angioplasty in Myocardial Infarction) (14), the COMPARE-ACUTE (Comparison Between FFR Guided Revascularization Versus Conventional Strategy in Acute STEMI Patients With MVD) (15), and the COMPLETE (Complete vs Culprit-only Revascularization to Treat Multi-vessel Disease After Early PCI for STEMI) (16) trials studied patients presenting with STEMI without CS and showed benefit from MV-PCI. The COMPLETE trial, the only trial powered for hard outcomes to date, demonstrated lower cardiovascular death or myocardial infarction at 3 years with non-culprit PCI. However, patients who presented with CS

were excluded from these studies. The CULPRIT-SHOCK trial (10) included patients with AMICS and demonstrated a lower 30-day mortality when patients with MVCAD were treated with CV-PCI as compared with MV-PCI during the index procedure. It is important to note limitations of the CULPRIT-SHOCK trial, in which only 28% of patients received a MCS device, including 12% of the total cohort who were supported with Impella. It is also important to note that 24% of the MV-PCI group had CTO PCI, which increased the risk of complications and contrast use, and that only one-third of patients had successful CTO PCI. In fact, a subgroup analysis of patients without CTO PCI showed no significant difference in the primary endpoint between MV-PCI and CV-PCI (56.4% vs. 49.1%, odds ratio: 0.87; 95% confidence interval: 0.74 to 1.02) (10). It is unclear whether providing MCS to allow for further revascularization or limiting revascularization to non-CTO MV-PCI would have improved outcomes.

In the MV-PCI cohort, we observed more severe impairment of cardiac function at presentation that became worse at 12 h, with a CPO of 0.79 W in the MV-PCI cohort versus 0.91 W in the CV-PCI cohort ($p = 0.03$). Similarly, lactic acid was higher at 12 h for the MV-PCI group (4.8 mg/dl vs. 3.5 md/dl; $p = 0.03$). However, at 24 h, both CPO and lactic acid were not different between groups. One explanation for this finding is that MV-PCI may delay recovery of hemodynamics. Prior studies have demonstrated that transient changes in contractility and hemodynamic shifts occur with balloon inflations and PCI (17). These transient changes are likely more pronounced in states of CS. This may also explain why in our study in which all patient received MCS, patients were more tolerant of this transient worsening of myocardial function without effecting in-hospital mortality.

A meta-analysis by Bertaina et al. (11), which included 11 observational studies and 1 RCT (the CULPRIT-SHOCK trial), demonstrated that MV-PCI was not associated with increased mortality when compared with CV-PCI, but did show higher rates of AKI. This could potentially be explained by the higher contrast volume used in MV-PCI. In our study, no significant difference was seen between the rates of AKI regardless of the revascularization strategy and the amount of contrast use. In patients with AMICS, strategies to prevent contrast-induced nephropathy are sometimes limited by time and the patient's volume status; however, the use of MCS could potentially explain the lower rates of AKI in our study and the fact that there was no association between the amount of contrast and the rates of AKI in our study group.

At present, both ACC/AHA (6) and European Society of Cardiology (8) guidelines recommend that in AMICS, a complete revascularization strategy should be considered, especially if there is severe stenosis of a non-infarct-related vessel. Lee et al. (18) compared 260 patients with MV-PCI versus 399 patients with CV-PCI who presented with STEMI and CS, and found a lower risk of all-cause-mortality at 1 year with MV-PCI. In that study, 26% of patients had a mechanical support device, and 33% of patients had 3-vessel CAD. In our study, 50% of patients had 3-vessel disease, and those who underwent 3-vessel PCI had a lower mortality (27%) than patients who underwent 2-vessel PCI (34%) and 1-vessel PCI (34%). The main difference between prior studies and this study is that all patients were supported with MCS, which allows the myocardium to rest, and in combination with MV-PCI, can reduce potential ischemia in the nonculprit vascular territories (19).

Intervening in nonculprit vessels in patients with AMICS should be carefully evaluated. Operators must take into account several factors, including TIMI flow grade, presence of a CTO, the size and distribution of the vessel, underlying hemodynamics, and the patient's hemodynamic reserve to guide the decision of performing MV-PCI.

STUDY LIMITATIONS. This is a subgroup analysis of a single-arm observational study in which all patients received MCS, and the sample size could be underpowered to detect any significant differences between groups. The ultimate decision to perform MV-PCI was solely at the operator's discretion, possibly introducing selection bias. Nevertheless, the NCSI trial is one of the largest registries of patients with AMICS undergoing Impella-supported PCI, and multivessel Impella-assisted PCI in the setting of AMICS has not been studied previously.

CONCLUSIONS

In patients with MVCAD presenting with AMICS who were treated with early MCS, revascularization of nonculprit lesions was associated with similar hospital survival and rates of AKI when compared with culprit-only PCI. Selective nonculprit PCI can be safely performed in AMICS in patients supported with MCS. Further studies are needed to assess long-term outcomes of this treatment strategy.

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PERSPECTIVES

WHAT IS KNOWN? Complete revascularization has been shown to be beneficial in the setting of acute coronary syndrome; however, there is conflicting evidence about nonculprit vessel intervention in the setting of cardiogenic shock, particularly with the use of mechanical circulatory support.

WHAT IS NEW? In patients with acute myocardial infarction complicated by cardiogenic shock, revascularization of non-culprit lesions is associated with similar hospital survival and rates of acute kidney injury when compared to culprit-only percutaneous coronary intervention.

WHAT IS NEXT? Further studies are needed to assess long-term outcomes of this treatment strategy.

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KEY WORDS acute myocardial infarction, cardiogenic shock, culprit, multivessel

APPENDIX For a list of the National Cardiogenic Shock Initiative investigators and collaborators and the participating sites, please see the online version of this paper.