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Angiographic Findings and Clinical Correlates in Patients With Cardiogenic Shock Complicating Acute Myocardial Infarction: A Report from the SHOCK Trial Registry

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OBJECTIVES We sought to delineate the angiographic findings, clinical correlates and in-hospital outcomes in patients with cardiogenic shock (CS) complicating acute myocardial infarction.

BACKGROUND Patients with CS complicating acute myocardial infarction carry a grave prognosis. Detailed angiographic findings in a large, prospectively identified cohort of patients with CS are currently lacking.

METHODS We compared the clinical characteristics, angiographic findings, and in-hospital outcomes of 717 patients selected to undergo angiography and 442 not selected, overall and by shock etiology: left or right ventricular failure versus mechanical complications.

RESULTS Patients who underwent angiography had lower baseline risk and a better hemodynamic profile than those who did not. Overall, 15.5% of the patients had significant left main lesions on angiography, and 53.4% had three-vessel disease, with higher rates of both for those with ventricular failure, compared with patients who had mechanical complications. Among patients who underwent angiography, those with ventricular failure had significantly lower in-hospital mortality than patients with mechanical complications (45.2% vs. 57.0%; $p = 0.021$). Importantly, for patients with ventricular failure, in-hospital mortality also correlated with disease severity: 35.0% for no or single-vessel disease versus 50.8% for three-vessel disease. Furthermore, mortality was associated with the culprit lesion location (78.6% in left main lesion, 69.7% in saphenous vein graft lesions, 42.4% in circumflex lesions, 42.3% in left anterior descending lesions, and 37.4% in right coronary artery lesions), and Thrombolysis In Myocardial Infarction (TIMI) flow grade (46.5% in TIMI 0/1, 49.4% in TIMI 2 and 26% in TIMI 3).

CONCLUSIONS Patients who underwent angiographic study in the SHOCK Trial Registry had a more benign cardiac risk profile, more favorable hemodynamic findings and lower in-hospital mortality than those for whom angiograms were not obtained. Patients with CS caused by ventricular failure had more severe atherosclerosis, and a different distribution of culprit vessel involvement but lower in-hospital mortality, than those with mechanical complications. Overall in-hospital survival correlates with the extent of coronary artery obstructions, location of culprit lesion and baseline coronary TIMI flow grade. (J Am Coll Cardiol 2000;36: 1077–83) © 2000 by the American College of Cardiology

For the past two decades, the incidence of cardiogenic shock (CS) in acute myocardial infarction (AMI) has remained stable, involving 5% to 15% of patients hospitalized for AMI (1–4). Despite the introduction of specialized care units, the advent of potent pharmacological agents, and refinements in mechanical interventions, the prognosis of patients with CS complicating AMI remains poor (5–7). Importantly, conventional treatment with intravenous thrombolytics does not substantially improve in-hospital mortality in patients with AMI once CS is diagnosed (8,9).

Recent observational studies have shown that early, aggressive interventions may translate into improved early clinical outcomes in these patients (10–16). Furthermore, retrospective, nonrandomized studies consistently have reported an encouraging effect on mortality with revascularization strategies in this cohort (17–19). Consequently, knowledge of coronary angiographic findings may help formulate more rational therapeutic strategies to improve clinical outcomes in these patients. However, there are few data that describe the coronary anatomy, including the extent of coronary obstructions, location of culprit vessels,

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Abbreviations and Acronyms

AMI	= acute myocardial infarction
CAD	= coronary artery disease
CK(-MB)	= creatine phosphokinase (-MB)
CS	= cardiogenic shock
IABP	= intra-aortic balloon pump
LAD	= left anterior descending
LV	= left ventricular, left ventricle
MR	= mitral regurgitation
RV	= right ventricular, right ventricle
SHOCK	= SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK? (Trial)
TIMI	= Thrombolysis In Myocardial Infarction
VSR	= ventricular septal rupture

and status of coronary flow, in a large, prospectively collected database of patients with CS.

The "SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK?" (SHOCK) Trial was a multicenter study of emergency revascularization in patients with AMI complicated by CS. In an effort to ensure that all potentially eligible patients were screened for the study and to minimize enrollment bias, all patients with clinical or hemodynamic evidence of CS complicating AMI, but not enrolled in the randomized trial, were entered into a registry (6). The SHOCK Trial Registry provides a unique database to evaluate the cardiovascular anatomy, angiographic findings and clinical correlates in these patients.

METHODS

Patients. Thirty-six enrolling centers registered 1,190 patients with suspected CS complicating AMI who were either ineligible for the randomized SHOCK Trial or eligible and not randomized from April 1993 to August 1997. A detailed description of the SHOCK Trial Registry methodology is reported in this supplementary issue of the *Journal* (6). A local discharge diagnosis of AMI with CS (DRGs 410, 410.1 to 410.9, in conjunction with 785.51) constituted criteria for being registered. Seven hundred and thirty patients (61%) were registered in 24 U.S. centers; 256 (22%) in five Canadian centers; 76 (6%) in four Belgian centers; and 128 (11%) in Australia, New Zealand and Brazil. Reports were not available for 31 of the angiography patients, and this report is therefore based on a total of 1,159 SHOCK Trial Registry patients.

Data collection. Data were abstracted from medical records by SHOCK study coordinators, who were centrally trained to complete standard report forms. They captured patient and MI characteristics, hemodynamics, procedure use, and vital status at discharge. Cardiac catheterization reports from all investigation sites were sent to the Clinical Coordinating Center for abstraction and central completion of a standardized report form that included the extent of coronary artery obstructions, degree of lesion severity, culprit lesion location, Thrombolysis In Myocardial Infarction (TIMI) flow characteristics and ejection fraction. Not all of these variables were available on all patients; the sample size for each variable is noted in the tables.

Definitions. Predominant left ventricular (LV) failure was designated as the etiology of shock when none of the following was indicated: isolated right ventricular (RV) shock, mechanical cause (acute severe mitral regurgitation [MR] or ventricular septal rupture [VSR]), tamponade, prior severe valvular heart disease, excess beta or calcium-channel blockade, or shock resulting from a catheterization laboratory complication.

Re-infarction was defined as: 1) recurrent chest pain or ischemic symptoms ≥30 min and recurrent ST-segment elevation, new Q waves, or new left bundle branch block; 2) total creatine kinase (CK) at least twice the upper limit of normal and >25% or 200 U/mL over the previous value with an elevated CK-MB level; or 3) a rise in CK-MB above the upper limit of normal after it had reverted to the normal range.

The description of coronary blood flow varied from center to center. Thrombolysis In Myocardial Infarction flow grades 0 to 3 were recorded as stated. If no TIMI flow grade was specified, an assessment was made based on the information provided and the following pre-specified criteria: a statement that there was no antegrade flow or total occlusion or 100% stenosis were set to TIMI grade 0 to 1; sluggish flow or subtotal occlusion, including 95% to 99% stenosis, were set to TIMI grade 2; normal flow or patent or <95% stenoses were set to TIMI grade 3. This method may systematically categorize some patients with TIMI 3 flow as TIMI 2.

Statistical methods. The clinical characteristics, etiologies of CS and hemodynamic findings of the 717 patients with available angiographic results were compared with those of the 442 who did not undergo angiography (total, n = 1,159). In a subset analysis, the angiographic and clinical findings were compared between patients with LV or RV failure and those with a mechanical cause of shock. Angiographic correlations with clinical outcomes were examined in more detail in patients with pump (LV or RV) failure because they accounted for most of the patients (77.9%) who developed CS after AMI.

Groups were compared using the Fisher exact test for categorical variables, the Wilcoxon rank-sum test for ordinal and non-normally distributed continuous variables and the Student *t*-test for normally distributed continuous variables. When three ordered groups were being compared, variables were evaluated using the Mantel-Haenszel test for linear trend. All analyses were conducted using SAS (SAS Institute, Cary, North Carolina).

RESULTS

Patient characteristics (Table 1). Of the 1,190 patients in the SHOCK Trial Registry, 748 patients (63%) underwent angiography, and results were available for 717 patients.

Table 1. Baseline Characteristics of SHOCK Trial Registry Patients

	Overall (n = 1,159)	Angiography Performed		p Value
		Yes (n = 717)*	No (n = 442)	
Female gender	40.6%	39.9%	41.6%	0.558
Age (yrs)	68.7 ± 11.8	65.8 ± 11.2	73.8 ± 10.9	< 0.001
History of infarction	37.3%	32.7%	44.7%	< 0.001
History of hypertension	53.2%	50.6%	57.6%	0.024
Diabetes	32.6%	29.7%	37.4%	< 0.001
Smoking	50.2%	52.2%	46.7%	0.097
History of angioplasty	6.2%	7.6%	3.8%	< 0.001
History of bypass surgery	9.9%	9.4%	10.7%	0.467
History of congestive heart failure	20.1%	16.1%	26.5%	< 0.001
History of renal insufficiency	11.0%	8.4%	15.4%	< 0.001
History of peripheral vascular disease†	18.2%	16.0%	22.5%	0.033
History of elevated lipids‡	41.9%	45.0%	35.8%	0.034

*Reflects only patients with available angiographic results (717/748); †Data available for n = 738; ‡Data available for n = 573.

Hence, the size of the SHOCK Trial Registry cohort for these analyses is 1,159. Compared with the 442 patients who did not undergo angiography, patients with angiographic data had a more favorable risk profile; they were younger and had a lower incidence of prior MI, hypertension, diabetes, prior congestive heart failure, renal insufficiency and peripheral vascular disease.

Etiology of CS (Table 2). Mechanical complications of MI were responsible for the development of CS in 12.5% of the patients in this SHOCK Trial Registry cohort, whereas predominant RV or LV failure was diagnosed in 77.9%. Indeed, LV failure was the most frequent cause of CS (74.5%). Compared with patients who did not undergo angiography, the angiographic cohort had a higher rate of mechanical complications (14.6% vs. 9.1%, $p < 0.001$). The in-hospital mortality of patients who underwent angiography (47.3%) was markedly lower than those who did not undergo angiography (86.2%).

Hemodynamic findings (Table 3). The hemodynamic measurements closest to onset of CS were obtained for SHOCK Trial Registry patients, with a median time from onset of CS to right heart catheterization of 3.9 h and a range of from 1.1 h at the first quartile to 17.7 h at the third quartile. Patients who underwent angiography had more favorable hemodynamics findings with higher systolic and diastolic blood pressures, lower systolic pulmonary artery pressure, and higher ejection fraction and cardiac index values, compared with patients who were not selected for angiography.

Angiographic findings (Table 4). The majority of patients (72.4%) underwent angiography while they were in CS. Most patients (53.4%) had three-vessel disease, and there was a high frequency (15.5%) of significant (≥50%) left main lesions. The left anterior descending artery (LAD) was the culprit vessel in 41.3% of cases. Thrombolysis In Myocardial Infarction grade 0 or 1 flow in the infarct-related artery was noted in 67% of the 494 patients with data available. Ejection fraction (n = 252), which was most often obtained while the patient was receiving support measures, was severely compromised (34.2% ± 14.4%).

Patients with ventricular failure had a higher prevalence of triple-vessel disease (56.4% vs. 39.8%, $p = 0.029$), but there was no difference in the rate of severe left main disease (16.2% vs. 10.9%, $p = 0.161$) when compared with patients with CS shock due to mechanical failure. The culprit vessel was more often the LAD in the ventricular-failure cohort, whereas the circumflex artery was more often involved in patients with mechanical failure. In patients who received thrombolytic therapy and had TIMI flow data available, 60% of 116 patients had TIMI grade 0 or 1 flow post thrombolytic treatment, compared with 91% of 11 patients

Table 2. Etiology of CS and In-hospital Mortality

	Overall (n = 1,159)	Angiography Performed		p Value
		Yes (n = 717)*	No (n = 442)	
Predominant left ventricular failure	74.5%	73.6%	75.9%	0.375
Isolated right ventricular failure	3.4%	3.5%	3.2%	0.774
Mechanical complication†	12.5%	14.6%	9.1%	< 0.001
Cardiac tamponade or rupture	2.3%	2.5%	2.0%	0.606
Severe prior valvular disease	2.9%	2.2%	4.1%	0.070
Other	6.3%	5.9%	7.0%	0.431
In-hospital Mortality	62.1%	47.3%	86.2%	< 0.00001

*Reflects only patients with available angiographic results (717/748); †Ventricular septal defect or acute severe mitral regurgitation.

Table 3. Hemodynamic Findings Measured Close to Shock Onset While Patients Often on Mechanical or Pharmacological Support

	Overall (n = 1,159)	Angiography Performed		p Value
		Yes (n = 717)*	No (n = 442)	
Heart rate (beats/min)	95.7 ± 26.2 (n = 1,121)	96.7 ± 24.9 (n = 675)	93.8 ± 27.3 (n = 417)	0.091
Systolic blood pressure (mm Hg)	87.7 ± 22.3 (n = 1,124)	89.7 ± 21.6 (n = 679)	84.4 ± 22.8 (n = 417)	< 0.001
Diastolic blood pressure (mm Hg)	52.3 ± 17.0 (n = 976)	53.5 ± 16.5 (n = 613)	50.0 ± 17.7 (n = 338)	0.007
PCWP (mm Hg)	23.4 ± 8.4 (n = 739)	23.3 ± 8.5 (n = 536)	23.6 ± 8.5 (n = 185)	0.512
Pulmonary artery systolic pressure (mm Hg)	41.2 ± 12.8 (n = 482)	40.3 ± 12.9 (n = 371)	44.8 ± 12.3 (n = 96)	< 0.001
Left ventricular ejection fraction (%)†	32.6 ± 13.8 (n = 468)	33.5 ± 13.8 (n = 357)	28.8 ± 12.5 (n = 96)	0.003
Cardiac index (L/min/m^2)	2.1 ± 0.8 (n = 562)	2.1 ± 0.8 (n = 398)	1.9 ± 0.6 (n = 149)	0.017

*Reflects only patients with available angiographic results (717/748). Right heart catheterization was performed in 79% of patients with angiogram; †Measured at any time during the index hospitalization.

whose angiograms were obtained prior to thrombolysis (p = 0.051).

The extent of coronary artery stenoses was slightly less severe in the SHOCK Trial Registry, compared with patients who met strict criteria and were enrolled in the randomized SHOCK Trial with LV failure (24): 0/1-vessel disease, 22.4% vs. 13%; two-vessel disease, 20.7% vs. 22.6%; three-vessel disease, 57% vs. 64.4%; p = 0.006, and left main, 16.1% vs. 21%; p = 0.122 for 518 SHOCK Trial Registry patients with predominant LV failure (excluding patients with RV failure) and 239 SHOCK Trial patients, respectively.

Outcomes in relation to angiographic findings in patients with ventricular failure. Patients with ventricular failure had significantly lower in-hospital mortality than those with a mechanical cause of shock (45.2% vs. 57%, p = 0.021, Table 4). Their mortality increased as the severity of disease increased, from 35% in patients with pump failure and no or single-vessel disease at angiography to 50.8% in such patients with three-vessel disease (p = 0.002, Table 5). The infarct-related artery was also associated with survival in patients with pump failure; if the culprit vessel was the left main or a saphenous vein graft, the associated mortality was dismally high (78.6% and 69.7%, respectively; Table 6). Remarkably, any subnormal TIMI flow in the culprit vessel was associated with a poorer prognosis; only the presence of TIMI grade 3 flow was associated with better survival in this cohort. Percutaneous interventions were the most prevalent for patients with single-vessel disease (75.6%), but bypass surgery was more common in patients with more extensive disease (Table 5). Recurrent ischemia and re-infarction occurring between the initial MI that brought the patient to the hospital and the onset of shock was noted in 26.9% of patients with pump failure. This might result from a combination of the unstable hemodynamic status and the extensive obstructive coronary artery disease (CAD) in these

Table 4. Registry Angiographic Findings, Overall and by Shock Etiology

	All* (n = 717)	Etiology of Shock		p Value
		Mechanical (n = 114)	Pump Failure (n = 549)	
Left main disease ≥50% (n = 691)	15.5%	10.9%	16.2%	0.161
Number diseased vessels (n = 708)				0.029
0 or 1	22.7%	24.8%	22.7%	
2	23.9%	35.4%	20.9%	
3	53.4%	39.8%	56.4%	
Culprit vessel (n = 552)				0.053
LAD	41.3%	33.3%	42.7%	
Left circumflex	15.8%	26.9%	13.5%	
Right coronary	29.5%	30.8%	29.9%	
Left main	5.6%	3.9%	6.4%	
Saphenous vein graft	7.8%	5.1%	7.5%	
Quantitative findings				
Stenosis ≥50% (n = 525)	99.6%	100.0%	99.5%	0.545
TIMI grade 0/1 flow (n = 494)	66.8%	69.3%	67.2%	0.716
Ejection fraction (%) (n = 252)†	34.2 ± 14.4	35.7 ± 13.3	33.2 ± 14.3	0.240
In-hospital mortality	47.3%	57.0%	45.2%	0.021

*Includes those with mechanical failure, those with left or right ventricular failure, and patients with other causes of shock; †By left ventriculogram.
LAD = left anterior descending.

Table 5. In-hospital Mortality and Intervention Rates by Angiographic Disease Severity in Patients with LV or RV Failure

	0/1-Vessel Disease (n = 123)	2-Vessel Disease (n = 113)	3-Vessel Disease (n = 305)	p Value*
Mortality	35.0%	39.8%	50.8%	0.002
Percutaneous coronary intervention†	75.6%	63.7%	43.3%	0.001
Angioplasty only	72.4%	61.1%	38.0%	0.001
Bypass surgery	6.5%	15.9%	33.8%	0.001

*p value from Mantel-Haenszel test for linear trend; †Includes 23 patients with bypass surgery post angioplasty.

critically ill patients. Importantly, no angiographic variables were significantly associated with re-infarction.

DISCUSSION

The extensive CAD demonstrated in the SHOCK Trial Registry is consistent with our understanding of the pathogenesis of pump failure, which is extensive myocardial ischemia and necrosis. Furthermore, it highlights the challenges in accomplishing restoration of normal coronary perfusion. Cardiogenic shock remains the leading cause of mortality in patients admitted for AMI (20,21), despite the substantial improvements in survival that have occurred since the introduction of thrombolytic treatment. More recently, the combination of intra-aorta balloon pump (IABP) and successful thrombolysis or revascularization (percutaneous or surgical) has been shown to enhance clinical outcomes in observational studies (22,23). The randomized SHOCK Trial demonstrated that early revascularization improved six-month survival (24). Therefore, knowledge of angiographic findings may provide insight into the pathogenesis of CS and suggest more rational treatment approaches in these critically ill patients.

Angiographic findings in patients with CS. Patients selected for angiography had a better risk profile than those who did not undergo angiography. This partially accounts for their better survival, as reported (6). In the current analysis, the extent of CAD was significantly and inversely related to in-hospital survival in patients with shock due to LV or RV failure. This correlates well with the report by Wacker et al. (25), which noted a high prevalence of severe triple-vessel disease (75% of cases) and diffuse LAD obstructions (84%) at autopsy in patients who died from CS with AMI.

In recent multicenter trials for treatment of AMI, multivessel CAD was noted in 47% to 59.4% of the patients enrolled; left main culprit lesions were present in 0.8% to 1.7%; and vein-graft lesions constituted 1.7% to 3.9% of the culprit lesions (26–33). Compared with these patients,

Table 6. In-hospital Event Rates by Angiographic Findings in Patients with LV or RV Failure

	Mortality		Recurrent Ischemia and Re-infarction*	
Overall event rate (n = 549, 409)	45.2%		26.9%	
Number of diseased vessels		p = 0.002		p = 0.300
0 or 1	35.0%		24.4%	
2	39.8%		23.3%	
3	50.8%		29.3%	
Left main disease present	62.8%		25.4%	
		p = 0.001		p = 0.734
Left main disease absent	49.9%		27.3%	
Infarct-related vessel		p < 0.001		p = 0.295
Right coronary	37.4%		23.7%	
Circumflex	42.4%		33.3%	
Left anterior descending	42.3%		26.2%	
Left main	78.6%		19.1%	
Saphenous vein graft	69.7%		42.3%	
TIMI flow in culprit vessel		p = 0.035		p = 0.064
0 or 1	46.5%		22.9%	
2	49.4%		33.9%	
3	26.0%		34.4%	
Ejection fraction (%)†	Dead: 32.0 ± 16.2		ReMI: 36.9 ± 13.4	
		p = 0.245		p = 0.163
	Alive: 33.9 ± 13.1		No ReMI: 31.2 ± 14.4	

*Collected on new forms only—409 of 549 patients. Re-infarction was defined as occurring between the initial MI causing hospitalization and onset of shock; †By left ventriculography (data available for 179 patients).

those in the SHOCK Trial Registry had more multivessel disease (77.3%) and more frequent culprit left-main (5.6%) and vein-graft lesions (7.8%). When the infarct-related artery was the left main or a vein graft, the prognosis was particularly poor in those with shock brought on by pump failure.

The presence of TIMI grade 3 flow in the infarct artery also was associated with improved survival. Perhaps normal TIMI flow reflects not only the severity of the culprit lesion but also the integrity of the microvasculature and viability of the myocardium normally supplied by the culprit vessel. The high frequency of left-main and three-vessel disease, in conjunction with the impaired LV function in patients with CS, justifies routine angiography for most of these patients, based on long-term survival benefits shown with revascularization for these anatomic findings. Furthermore, the SHOCK Trial demonstrated improved six-month survival with early revascularization, compared with initial medical stabilization (24).

Although the majority of the eligible patients received thrombolysis, nearly 70% of the patients who underwent angiogram had only TIMI grade 0 or 1 flow. This finding confirms the low reperfusion rate in patients with CS who receive thrombolysis (34). This is particularly disconcerting, considering the strong association of normal TIMI flow with survival in our study. Whether more potent thrombolytic agents or the combination of thrombolytics with platelet glycoprotein IIb/IIIa antagonists would enhance reperfusion and thus outcomes in these patients remains to be determined.

Outcome in relation to shock etiology and angiographic findings. Patients with ventricular failure had significantly better in-hospital survival than those with mechanical etiology of shock, despite having higher rates of left-main and vein-graft involvement, and more multivessel disease, than those with mechanical failure. Of note, improved survival in patients with ventricular failure was observed only in the cohort selected for angiography; outcomes for such patients with severe MR were similar, and those patients with VSR were worse (6,35,36).

Patients with LV or RV failure. Patients admitted with shock brought on by pump failure had a high mortality rate (45.2%), but this is lower than rates from historical reports (2–5). This can be explained partly by the selection for angiography and by the aggressive treatment that our patients received (6). Indeed, over 70% of those who underwent angiography in the SHOCK Trial Registry received IABP and revascularization.

Study limitations. By contrast to the randomized SHOCK Trial, the angiograms obtained in the SHOCK Trial Registry patients were not analyzed in a core laboratory, but were reviewed during central completion of standardized report forms based on local laboratory reports. Although our data demonstrated that TIMI flow grade has important prognostic value in patients with CS, the absence of a central core lab analysis and overall hemodynamic status of these critically ill patients at the time of catheterization may have affected the overall TIMI flow findings. Patients enrolled in the SHOCK Trial Registry were those who failed to meet all the prespecified stringent enrollment criteria or refused participation in the multicenter randomized trial, and the decision to obtain angiography in these patients was largely dictated by the patients' personal physicians. These confounding variables clearly would affect the angiographic findings in this analysis. Indeed, patients in the SHOCK Trial Registry seem to have less extensive coronary artery obstructions, compared with those enrolled in the randomized trial with its more stringent enrollment criteria. On the other hand, the less stringent enrollment criteria in the SHOCK Trial Registry patients also makes the angiographic findings closer to the "real-world" findings derived from everyday practices in catheterization laboratories.

Conclusions. Patients selected by their physicians for angiography in the SHOCK Trial Registry had a lower-risk profile than those who did not undergo angiography. Overall, patients in the SHOCK Trial Registry had a high prevalence of left-main and diffuse triple-vessel disease. Patients with different etiologies for CS have different clinical characteristics and angiographic findings. Patients with a mechanical cause of shock had a lower rate of three-vessel disease. Despite these findings, these patients had higher mortality than did those with shock due to ventricular failure.

For patients with CS brought on by pump failure, in addition to the extent of coronary artery obstructions, infarct-related vessel and baseline TIMI flow were also found to have significant prognostic value for these patients. Importantly, if either the left main or a vein graft was the culprit vessel, the prognosis was particularly poor, with in-hospital mortality of ~70%. On the other hand, patients with initial TIMI grade 3 flow in the infarct vessel had substantially better in-hospital survival than those with TIMI grade 0 or 1 flow. Thus, timely angiographic studies would be helpful in the management and prognostication of these patients.

Although survival in patients with CS complicating AMI remains poor, an aggressive treatment approach (including a combination of more potent thrombolytic agents, platelet glycoprotein IIb/IIIa inhibitors, early placement of an IABP and emergency percutaneous or surgical reperfusion strategies) may further improve TIMI flow and hence clinical outcomes in these complex, critically ill patients.

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REFERENCES

1. Goldberg RJ, Samad NA, Yarzebski J, et al. Temporal trends in cardiogenic shock complicating acute myocardial infarction. N Engl J Med 1999;340:1162–8.
2. Goldberg RJ, Gore JM, Alpert JS, et al. Cardiogenic shock after acute myocardial infarction: incidence and mortality from a community-wide perspective, 1975 to 1988. N Engl J Med 1991;325:1117–22.
3. Scheidt S, Ascheim R, Killip T III. Shock after acute myocardial infarction: a clinical and hemodynamic profile. Am J Cardiol 1970;26: 556–64.
4. Leor J, Goldbourt U, Reicher-Reiss H, Kaplinsky E, Behar S. Cardiogenic shock complicating acute myocardial infarction in patients without heart failure on admission: incidence, risk factors, and outcome. Am J Med 1993;94:265–73.
5. Killip T III, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two year experience with 250 patients. Am J Cardiol 1967;20:457–64.
6. Hochman JS, Buller CE, Sleeper LA, et al., for the SHOCK Investigators. Cardiogenic shock complicating acute myocardial infarction—etiologies, management and outcome: a report from the SHOCK Trial Registry. J Am Coll Cardiol 2000;36:1063–70.
7. Califf RM, Bengtson JR. Cardiogenic shock. N Engl J Med 1994;330: 1724–30.
8. Gruppo Italiano per lo Studio della Streptokinasi nell'Infarto Miocardico. Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986;1:397–401.
9. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. Lancet 1994;343:311–22.
10. Lee L, Bates ER, Pitt B, et al. Percutaneous transluminal coronary angioplasty improves survival in acute myocardial infarction complicated by cardiogenic shock. Circulation 1988;78:1345–51.
11. Verna E, Repetto S, Boscarini M, et al. Emergency coronary angioplasty in patients with severe left ventricular dysfunction or cardiogenic shock after acute myocardial infarction. Eur Heart J 1989;10:958–66.
12. Lee L, Erbel R, Brown TM, et al. Multicenter registry of angioplasty therapy of cardiogenic shock: initial and long-term survival. J Am Coll Cardiol 1991;17:599–603.
13. Hibbard MD, Holmes DR, Bailey KR, et al. Percutaneous transluminal coronary angioplasty in patients with cardiogenic shock. J Am Coll Cardiol 1992;19:639–46.
14. Champagnac D, Claudel J, Chevalier PH, et al. Primary cardiogenic shock during acute myocardial infarction: results of emergency cardiac transplant. Eur Heart J 1993;14:925–9.
15. Gacioch GM, Ellis SG, Lee L, et al. Cardiogenic shock complicating acute myocardial infarction: the use of coronary angioplasty and the integration of new support devices into patient management. J Am Coll Cardiol 1992;19:647–53.
16. Webb JG, Carere RG, Hilton JD, et al. Usefulness of coronary stenting for cardiogenic shock. Am J Cardiol 1997;79:81–4.
17. Moosvi AR, Khaja F, Villanueva L, et al. Early revascularization improves survival in cardiogenic shock complicating acute myocardial infarction. J Am Coll Cardiol 1992;19:907–14.
18. Berger PB, Holmes DR, Jr, Stebbins AL, et al., for the GUSTO-I Investigators. Impact of an aggressive invasive catheterization and revascularization strategy on mortality in patients with cardiogenic shock in the Global Utilization of Streptokinase and Tissue plasminogen activator for Occluded coronary arteries (GUSTO-I) trial. An observational study. Circulation 1997;96:122–7.
19. Holmes DR, Jr, Bates ER, Kleiman NS, et al., for the GUSTO-I Investigators. Contemporary reperfusion therapy for cardiogenic shock: the GUSTO-I trial experience. J Am Coll Cardiol 1995;26: 668–74.
20. Killip T. Cardiogenic shock complicating myocardial infarction. J Am Coll Cardiol 1989;14:47–8.
21. Becker RC, Gore JM, Lambrew C, et al. A composite view of cardiac rupture in the United States National Registry of Myocardial Infarction. J Am Coll Cardiol 1996;27:1321–6.
22. Anderson RD, Ohman EM, Holmes DR, Jr, et al., for the GUSTO-I Investigators. Use of intraaortic balloon counterpulsation in patients presenting with cardiogenic shock: observations from the GUSTO-I study. J Am Coll Cardiol 1997;30:708–15.
23. Kovack PJ, Rasak MA, Bates ER, et al. Thrombolysis plus aortic counterpulsation: improved survival in patients who present to community hospitals with cardiogenic shock. J Am Coll Cardiol 1997;29: 1454–8.
24. Hochman JS, Sleeper LA, Webb JG, et al., for the SHOCK Investigators. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. N Engl J Med 1999;341:625–34.
25. Wacker FJ, Lie KI, Becker AE, et al. Coronary artery disease in patients dying from cardiogenic shock or congestive heart failure in the setting of acute myocardial infarction. Br Heart J 1976;38:906–10.
26. Zijlstra F, de Boer MJ, Hoorntje JCA, et al. A comparison of immediate coronary angioplasty with intravenous streptokinase in acute myocardial infarction. N Engl J Med 1993;328:680–4.
27. Zimarino M, Corcos T, Favereau X, et al. Predictors of short term clinical and angiographic outcome after coronary angioplasty for acute myocardial infarction. Cathet Cardiovasc Diagn 1995;36:203–8.
28. Antoniucci D, Valenti R, Buonamici P, et al. Direct angioplasty and stenting of the infarct-related artery in acute myocardial infarction. Am J Cardiol 1996;78:568–71.
29. Zijlstra F, Beukema WP, van't Hof AWJ, et al. Randomized comparison of primary coronary angioplasty with thrombolytic therapy in low risk patients with acute myocardial infarction. J Am Coll Cardiol 1997;29:908–12.
30. Stone GW, Marsalese D, Brodie BR, et al. A prospective, randomized evaluation of prophylactic intra-aortic balloon counterpulsation in high risk patients with acute myocardial infarction treated with primary angioplasty. J Am Coll Cardiol 1997;29:1459–67.
31. Stone GW, Brodie BR, Griffin JJ, et al. Prospective, multicenter study of the safety and feasibility of primary stenting in acute myocardial infarction: in-hospital and 30-day results of PAMI Stent Pilot Trial. J Am Coll Cardiol 1998;31:23–30.
32. Brener SJ, Barr LA, Burchenal JEB, et al. Randomized, placebo-controlled trial of platelet glycoprotein IIb/IIIa blockade with primary angioplasty for acute myocardial infarction. Circulation 1998;98:734–41.
33. Hands ME, Rutherford JD, Muller JE, et al., and the MILIS Study Group. The in-hospital development of cardiogenic shock after myocardial infarction: incidence, predictors of occurrence, outcome and prognostic factors. J Am Coll Cardiol 1989;14:40–6.
34. Kennedy JW, Ritchie JL, Davis KB, Fritz JK. Western Washington randomized trial of intracoronary streptokinase in acute myocardial infarction. N Engl J Med 1983;309:1477–82.
35. Thompson CR, Buller CE, Sleeper LA, et al., for the SHOCK Investigators. Cardiogenic shock due to acute severe mitral regurgitation complicating acute myocardial infarction: a report from the SHOCK Trial Registry. J Am Coll Cardiol 2000;36:1104–9.
36. Menon V, Webb JG, Hillis LD, et al., for the SHOCK Investigators. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. J Am Coll Cardiol 2000;36:1110–6.