Original article

Percutaneous coronary intervention in cardiogenic shock complicating acute ST-elevation myocardial infarction—a single centre experience

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KEYWORDS

Acute myocardial infarction
Cardiogenic shock
Primary angioplasty

ABSTRACT

Background: Mortality in acute myocardial infarction (AMI) complicated by cardiogenic shock (CS) approaches 70–80%, regardless of the type of pharmacological treatment. Early revascularisation improves survival in AMI with CS. Our aim is to assess the predictors of mid-term outcome after percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI) and CS.

Methods: Forty-one patients who underwent primary or rescue PCI for CS were analysed comparing their baseline, angiographic, PCI data, 30-day and 1-year survival.

Results: There were no significant differences between survivors and non-survivors in baseline characters, except for more number of transfer admissions ($P=0.0005$), and cardiopulmonary resuscitations ($P=0.015$) in the later group. The mean time between myocardial infarction (MI) onset to shock and MI onset to revascularisation were 12.8 ± 12.9 hours and 17.0 ± 16.8 hours, respectively. Patients with better pre-procedure thrombolysis in myocardial infarction (TIMI) flow in the infarct-related artery (IRA) had better survival ($P=0.0005$). Successful PCI was achieved in 48.8% of patients. The 30-day mortality was 56.1% and all were prior to hospital discharge. Patients with successful PCI had better short-term survival in comparison with patients with failed PCI (80% vs 9.6%). Eighteen patients who survived at 30 days were followed up for 12–72 months (mean 28.5 ± 5.4 months). Fifteen patients survived at 1 year after PCI and all were in good functional status.

Conclusion: Mortality remains high even with PCI. Achieving IRA patency with TIMI 3 flow is the main determinant of survival. Survival and functional status are good in patients who are discharged from hospital.

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Introduction

Cardiogenic shock (CS) is the leading cause of death in the setting of acute myocardial infarction (AMI). It complicates 5–10% of the patients with AMI. With conservative management, 70–80% of the patients with CS succumb to this illness.^{1,2} The incidence of shock remained constant over the past two decades with a declining trend in the recent years with increasing use of primary percutaneous coronary intervention (PCI) for AMI.^{3,4} Pathological studies have established that

myocardial dysfunction either from ischaemia or necrosis constitutes the major cause of CS.⁵ Restoration of perfusion to the ischaemic myocardium is the definitive way of improving survival in AMI with CS. Even though there was intensive medical management with inotropic support, thrombolysis and intra-aortic balloon pump (IABP) support has shown some improvement in survival, this modality is less effective in CS.^{6,7} Non-randomised studies have reported marked lowering of mortality with early revascularisation.^{8–12} In the randomised SHOCK (SHould we emergently revascularise Occluded Coronaries for cardiogenic shock?) trial, there were 132/1000 lives of patients saved, treated with early revascularisation as compared with medical therapy.¹³ This paper reviews our hospital PCI experience in AMI complicated by CS.

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Materials and methods

This study was conducted between January 2001 and June 2007. Consecutive patients with acute ST-elevation myocardial infarction (STEMI) who underwent primary or rescue PCI were prospectively enrolled in a database. Patients who had PCI for CS were selected for our study. This population included patients directly admitted to our coronary care unit and those referred from peripheral hospitals without PCI facilities.

To obtain a more homogeneous population, only patients with CS due to predominant left ventricular failure (LVF) were included in the analysis. Patients with mechanical complications such as severe mitral regurgitation (MR), ventricular septal rupture, free wall rupture with tamponade, isolated right ventricular infarction and CS resulting from excess beta or calcium channel blockade or as a complication of a cardiac catheterisation were excluded.

The diagnosis of AMI was made if at least two of the following three elements were present: chest pain; ST-segment elevation of at least 0.1 mV in limb leads or 0.2 mV in precordial leads or a new onset left bundle branch block; serum creatinine phosphokinase-myocardial band isoenzyme (CPK-MB) elevation above twice the upper limit of normal.

The CS was defined as a compatible clinical presentation associated with a systolic blood pressure of 90 mmHg for at least 30 minutes despite inotropic and volume support as needed.

After admission to the coronary care unit, patients received inotropic and ventilator support as needed. They, then, underwent coronary angiography and angioplasty of the infarct-related artery (IRA) after IABP insertion. Patients were pre-loaded with 325 mg of aspirin and 50–70 units/kg of heparin. Glycoprotein (GP) IIb–IIIa inhibitor was administered as an adjunct in most of the patients. Periprocedural activated clotting time was maintained >200 seconds (HemoTec device). All suitable lesions in the IRA were stented. Multi-vessel PCI was done, when persistent haemodynamic instability was assumed to be related to the critical lesions in the non-IRAs.

Successful PCI was defined as a residual diameter stenosis of <50% and a thrombolysis in myocardial infarction (TIMI) 3 flow in the culprit vessel after the procedure.

All surviving patients were followed up clinically after hospital discharge for a mean of 28.5 ± 3.4 months.

Statistical methods

Data were collected prospectively. SPSS version 15.0 (SPSS Inc, Chicago, Illinois, USA) software was used for analysis. Continuous variables were summarised as mean $\pm$ standard deviation (SD) and categorical variables as percentages. To compare mortality across single variables, the χ^2 test (262 contingency tables) was used. A *P* value <0.05 was considered statistically significant. Cumulative survival was calculated by the Kaplan–Meier method.

Results

Between January 2001 and June 2007, 41 patients underwent primary or rescue PCI for AMI complicated by CS in our institution.

Baseline data

There were 31 (75.6%) men and 10 (24.4%) women. Their mean age was 58.2 ± 10.4 years. The baseline characteristics of these patients are shown in Table 1.

Anterior wall myocardial infarction (AWMI) occurred in 25 (61%) patients. Remaining patients 16 (39%) had non-anterior wall myocardial (inferior wall and posterior wall) infarction.

Mechanical ventilator support was given in 29 (70.8%) patients. All the patients received catecholamine support and IABP. Ten patients (24.4%) required temporary transvenous pacing. The MR (<Grade II) was present in 10 (24.4%) patients. Post PCI, two patients developed severe MR, two had ventricular septal rupture and free wall rupture occurred in one patient. All these patients underwent emergency coronary artery bypass surgery. Renal dysfunction requiring peritoneal dialysis occurred in 12 (29.2%) patients. Multi-organ dysfunction developed in 19 (46.3%) patients. Two patients had re-infarction.

Angiographic data (Table 2)

Coronary angiography showed single vessel disease in 15 (36.6%) patients. Double vessel disease was present in 12 (29.3%) and triple vessel disease in 11 (26.8%) patients. Left main coronary artery (LMCA) disease was present in 3 (7.3%) patients.

The IRA was the left anterior descending in 27 (65.79%), the circumflex in 7 (17.1%) and the right coronary artery in 7 (17.1%). Before the intervention, an occluded IRA (TIMI 0/1 flow) was observed in 35 (85.4%), a TIMI 2 flow in 6 (14.6%) and none of the patients had TIMI 3 flow.

Percutaneous coronary intervention data (Table 3)

Most of the patients underwent percutaneous transluminal coronary angioplasty (PTCA) to the IRA. Three patients had multivessel PCI. The LMCA stenting was done in one patient after right coronary artery distribution (RCA) stenting for persistent shock. Two patients underwent left anterior descending (LAD) stenting (90% stenosis) after RCA stenting.

The intervention resulted in TIMI 3 flow in 20 (48.8%) and TIMI 2 flow in 16 (39%) patients, while 5 (12.2%) had TIMI 1 flow in the IRA. A PCI success was achieved in 20 (48.8%) patients.

A stent was implanted in 37 (90.2%) patients. Totally, 43 stents were implanted, 26 (60.4%) bare metal stents and 17 (39.6%) drug-eluting stents. The GP IIb/IIIa inhibitor was

Table 1

Cardiogenic shock complicating acute myocardial infarction: baseline characteristics.

Characteristics	All PCI patients (n=41)	Survivors (n=18)	Non-survivors (n=23)	P value
Age (yr)	58.2±10.4	55.3±10.7	60.4±9.9	0.300
Male gender (%)	31 (75.6)	16 (88.9)	15 (65.2)	0.081
Diabetes (%)	22 (53.7)	10 (55.6)	12 (52.2)	0.540
Hypertension (%)	15 (36.6)	8 (44.4)	7 (30.4)	0.275
Smoking (%)	16 (39)	9 (50)	7 (30.4)	0.171
Dyslipidaemia (%)	9 (22)	4 (22.2)	5 (21.7)	0.630
Family history (%)	16 (39)	8 (44.4)	8 (34.8)	0.379
Previous MI (%)	8 (19.5)	3 (16.7)	5 (21.7)	0.500
Previous PCI (%)	5 (12.2)	2 (11.1)	3 (13)	0.581
Previous CABG (%)	1 (2.4)	1 (5.6)	0	0.252
Lowest SBP (mmHg)	68.8±11.8	69.9±10.5	68±12.9	0.254
HR (bpm)	112.5±3.5	118.4±27.2	107.9±32.7	0.533
Creatine kinase total	4704.9±3215.3	5206.28±3274.2	4312.3±3185.3	0.426
EF (%)	29.4±7.7	29.8±5.6	29.1±9.1	0.475
Mean time from MI to Shock (hr)	12.2±12.9	12.6±15	11.8±11.4	0.348
Mean time from MI to PCI (hr)	17±16.8	17.7±18.7	16.4±15.6	0.240
CPR (%)	18 (43.9)	4 (22.2)	14 (60.9)	0.015*
Transfer admission (%)	24 (58.5)	4 (22.2)	20 (87)	0.0005*
Thrombolysis eligibility (%)	31 (75.6)	13 (72.2)	18 (78.2)	0.655
Thrombolytic administered (%)	15 (36.6)	5 (27.8)	10 (43.4)	0.300
GP IIb/IIIa inhibitor (%)	35 (85.4)	15 (83.3)	20 (87)	0.542
Inotropes/vasopressors (%)	41 (100)	18 (100)	23 (100)	–
IABP (%)	41 (100)	18 (100)	23 (100)	–
IABP duration (hr)	51.5±43	57.4±40.4	46.9±45.3	0.260
Stent in IRA (%)	37 (90.2)	17 (94.4)	20 (87)	0.694
Mechanical ventilation (%)	35 (70.8)	12 (66.7)	23 (100)	0.745

*Statistically significant. CABG: coronary artery bypass grafting, CPR: cardio-pulmonary resuscitation, EF: ejection fraction, GP: glycoprotein, HR: heart rate, IABP: intra aortic balloon pump, IRA: infarct-related artery, MI: myocardial infarction, PCI: percutaneous coronary intervention, SBP: systolic blood pressure.

Table 2

Cardiogenic shock complicating acute myocardial infarction: angiographic characteristics.

Characteristics	All PCI patients (%)	Survivors (%)	Non-survivors (%)	P value
Diseased vessels				0.505
SVD	15 (36.6)	7 (38.9)	8 (34.8)	
DVD	12 (29.3)	6 (33.3)	6 (26.1)	
TVD	11 (26.8)	4 (22.2)	7 (30.4)	
LMD	3 (7.3)	1 (5.6)	2 (8.7)	
Culprit vessel				0.557
LAD	27 (65.8)	12 (66.7)	15 (65.2)	
LCX	7 (17.1)	4 (22.2)	3 (13.1)	
RCA	7 (17.1)	2 (11.1)	5 (21.7)	
Pre-PCI TIMI flow				0.001*
0	12 (29.3)	0	12 (52.2)	
1	23 (56.1)	13 (72.2)	10 (43.5)	
2	6 (14.6)	5 (27.8)	1 (4.3)	
3	0	0	0	

*Statistically significant. DVD: double vessel disease, LAD: left anterior descending artery, LCX: left circumflex artery, LMD: left main disease, PCI: percutaneous coronary intervention, RCA: right coronary artery, SVD: single vessel disease, TIMI: thrombolysis in myocardial infarction, TVD: triple vessel disease.

used in 35 (85.4%) patients; eptifibatide in 20 (48.8%), abciximab in 6 (14.6%) and tirofiban in 9 (21.9%).

Our data as compared to the SHOCK trial data is detailed in Table 4.

Table 3

Cardiogenic shock complicating acute myocardial infarction: percutaneous coronary intervention details.

Characteristics	All PCI patients (%)	Survivors (%)	Non-survivors (%)	P value
Post-PCI TIMI flow				0.0005*
0	0	0	0	
1	5 (12.2)	0	5 (21.7)	
2	16 (39)	2 (11.1)	14 (60.9)	
3	20 (48.8)	16 (88.9)	4 (17.4)	
PCI success				0.0005*
Successful	20 (48.8)	16 (88.9)	4 (17.4)	
Unsuccessful	21 (51.2)	2 (11.1)	19 (82.6)	

*Statistically significant. PCI: percutaneous coronary intervention, TIMI: thrombolysis in myocardial infarction.

Survival

The 30-day mortality rate was 56.1%. Of all the parameters analysed, only three variables were significantly different between survivors and non-survivors. Patients who were transferred from peripheral centres to our institution after shock onset ($P=0.0005$) and those who underwent cardiopulmonary resuscitation prior to angioplasty ($P=0.015$) fared less well in our series. The 30-day survival rate was better in patients with higher pre- and post-TIMI flow grades ($P=0.0005$).

Table 4

Comparison of percutaneous coronary intervention in SHOCK trial data and our data.

Characteristics	SHOCK trial data	Our data
No. of patients	82	41
Age (yr)	65±10	58.2±10.4
Diabetes mellitus (%)	25	53.7
Mean time from MI to shock (hr)	NA	12.2±12.9
Mean time from MI to revascularisation (hr)	NA	17±16.8
Thrombolysis (%)	49	36.6
Multivessel disease (%)	81	65
IABP (%)	89	100
Stents (%)	34	90.2
GP IIb/IIIa inhibitors (%)	32	85
TIMI-3 flow (%)	61	48.8
30-day survival (%)	54	45
1 year survival (%)	51	36

GP: glycoprotein, IABP: intra-aortic balloon pump, MI: myocardial infarction, NA: not available, TIMI: thrombolysis in myocardial infarction.

Figure 1 Post procedure TIMI flow and mortality. TIMI: thrombolysis in myocardial infarction.

(Figure 1). With successful PCI, the survival was 80%. In contrast, with failed PCI, the survival was only 9.6%. Of the five patients who underwent emergency surgery, four died during the postoperative period.

Follow-up data

Eighteen patients survived at 30 days and were followed up for 12–72 months (mean 28.5±5.4 months). Elective repeat PCI was done in two patients for diseased non-IRAs. Two patients underwent implantable cardioverter defibrillator (ICD) implantation for ventricular tachycardia and one patient had cardiac resynchronisation therapy. Three patients died during first year follow-up, one from heart failure and other two from ventricular arrhythmias (Figure 2). Another three patients died after 1 year follow-up. Remaining 12 patients are on follow-up till date. Most of these patients are in New York Heart Association (NYHA) I/II functional status.

Figure 2 Kaplan–Meier 1-year survival of patients with cardiogenic shock complicating acute myocardial infarction.

Discussion

The CS in the setting of AMI occurs most commonly due to abrupt dysfunction of >50% of the ventricular myocardium.⁵ This results either from extensive myocardial infarction (MI) per se, or from a less extensive infarction in the presence of a previous infarction or severe triple vessel disease. Myocardial infarction or ischaemia initiates cardiac dysfunction in patients with CS. The resulting myocardial dysfunction further worsens ischaemia creating a downward spiral. This vicious cycle if not quickly interrupted, may result in irreversible myocardial dysfunction. Thus, mortality in established shock without intervention is very high.^{14–16} The outcome of CS is closely related to the patency of the IRA. Even though thrombolytic therapy reduced the incidence of CS in the GISSI trial, the benefit of thrombolytic therapy in the presence of CS was disappointing.¹⁷ The lack of benefit might be due to reduced lysis of thrombi in patients with low perfusion pressures.¹⁸

Many non-randomised studies have shown improvement in survival with early revascularisation.^{8–12} However, there are only two randomised trials that compared medical therapy with early revascularisation and not much data is available on PCI in the setting of CS.

Swiss multicentre angioplasty for SHOCK (SMASH) trial, the first randomised trial to compare intensive medical treatment and invasive treatment showed a non-significant mortality difference (69% vs 78%; relative risk 0.88, 95% confidence interval, 0.6–1.2).¹⁹ This trial was terminated prematurely due to inadequate enrollment. In the SHOCK trial, 302 patients were randomly assigned to initial medical stabilisation ($n=150$) or to early revascularisation ($n=152$) within 6 hours of randomisation. Patients underwent revascularisation if CS began within 36 hours of the onset of MI. Mortality at 30 days was 46.7% in the revascularisation arm and 56% in the medical stabilisation arm which was not statistically significant. Nevertheless, at 6 months, the mortality was significantly lower in the revascularisation group (50.3% vs 63.1%, $P=0.03$), resulting in 128 lives saved per 1000 patients

treated. At the end of 1 year, there was a 13.2% reduction in mortality in the revascularisation arm, representing 132 lives saved per 1000 patients treated.¹³

In the PCI arm of SHOCK trial, the overall survival in the 82 patients who underwent angioplasty was 54% ($n=44$) at 30 days and 50% ($n=41$) at 1 year.²⁰ In the large primary PCI registry of the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte (ALKK), 1333 patients with CS underwent PCI. Total in-hospital mortality was 46.1%.²¹ Sutton et al. has reported in-hospital mortality of 51% in 113 patients who underwent PCI for CS.²²

Various clinical and procedural characteristics have been shown to be associated with poor outcomes in different studies. In the SHOCK trial¹³ and other observational studies, advanced age with its associated co-morbidities, was an important predictor of mortality in patients undergoing PCI for CS. Mean age in our patients was 58.2 ± 10.4 years, slightly younger than other studies. However, age was not a predictor of mortality in our population.

Timings of shock from chest pain onset and time to revascularisation were also important determinants of outcome in major trials and registries. In SHOCK Trial Registry, patients who developed early shock (<24 hours) fared less well than those with late onset shock (after 24 hours).²³ The mean time from MI to shock in our study was 12.8 ± 12.9 hours. Of the 41 patients, 20 (48%) developed shock within 6 hours of MI and 31 (76%) within 24 hours. Late shock occurred only in 10 (24%) patients. There was no relationship between timing of shock and mortality. In the randomised SHOCK trial and other registries,^{13,21,22,24} there was a trend towards improved survival in those who had early revascularisation (<6 hours). However, SHOCK trial¹³ also has shown survival benefit for as long as 48 hours after MI and 18 hours after shock onset. The mean time from MI onset to revascularisation in our study was 17 ± 16.8 hours. It is relatively longer compared to other studies. Most of our patients were transferred from other hospitals, which could explain the delay in PCI. This delay might have contributed to the higher mortality among our patients. We did not find any difference in mortality between those receiving revascularisation within 12 hours of MI and those revascularised later. As with SHOCK trial data, our study also suggests that late revascularisation (outside the traditional 12-hour window) is also useful in patients with CS.

In the SHOCK trial,¹³ 60% of the patients were transferred from centres without PCI facilities. This variable did not influence survival following PCI in the SHOCK study. Transfer admission was an important predictor of mortality in our study. Twenty-four (58%) patients were transferred from peripheral centres without PCI facilities. Most of the transferred patients (80%) succumbed to CS in spite of PCI. These patients were haemodynamically more unstable than patients directly admitted in our institution. Infrequent use of IABP, mechanical ventilation, inotropic support and long delay from MI to revascularisation in the transfer admissions might have contributed to higher mortality in this group of patients.

In the SHOCK Trial Registry, administration of thrombolytic therapy before PCI did not affect survival in patients undergoing PCI for CS.²⁵ However, in a series of 171 patients who

underwent PCI for CS, rescue angioplasty was associated with lower final TIMI 3 flow and higher 1-year mortality. Even patients with a successful rescue PCI procedure had higher 1-year mortality than those with a successful primary PCI.²⁶ In our series, 15 (36.6%) patients underwent rescue angioplasty. As in the SHOCK Trial Registry patients, rescue angioplasty did not significantly influence survival in our patients.

In SHOCK trial and other observational studies, procedural success with establishment of TIMI 3 flow in the IRA was the strongest predictor of survival.^{8–12,20} We could achieve TIMI 3 flow in 48.8% of the patients only, though we had established IRA patency (<50% stenosis) in 87.8% of the patients. The 30-day mortality in 18 patients who had procedural success was only 20% in contrast to 90.4% in failed PCI.

It has been shown in the primary PCI studies that usage of stent and other adjuncts was associated with improved IRA patency rate and TIMI flow. Even though there was no large randomised data in the setting of CS, subgroup of patients in SHOCK trial, ALKK registry and small observational studies achieved better procedural success with stenting.^{20,21,27–31} We used stent in the IRA whenever the lesion was suitable for stenting (>2.5 mm). In our study, 37 (90.2%) patients underwent stenting in the IRA. In the REO SHOCK study³² and other observational studies,^{30,31} use of GP IIb–IIIa inhibitors was associated with improved TIMI 3 flow in the IRA in patients undergoing PCI for CS. We have used GP IIb–IIIa inhibitors in 35 (85.4%) of our patients. However, neither stenting nor GP IIb–IIIa inhibitors influenced the outcome in our series. Mechanical ventricular support as a bridge to recovery might improve outcome in CS. Even though IABP is the commonly employed assist device, the results in the setting of CS are conflicting. As a policy, we used IABP in all CS patients and hence we did not assess influence of IABP on mortality. Ongoing studies with IABP and other assist devices might shed more light on this issue.³³ Although, most of our patients received IABP support, GP IIb–IIIa inhibitors and stenting, PCI success was less in our series than in SHOCK study and other registries. This could be due to long delay between shock onset and PCI.

Data for long-term outcome in CS is now available. In the SHOCK trial cohort, the 6 year survival rates for the hospital survivors were 62.4% vs 44.4% in the early revascularisation and initial medical stabilisation groups respectively.³⁴ In the GUSTO-I trial, 55% hospital survivors were alive at 11 years. Furthermore, annual mortality rates after 1 year (2–4%) were similar for those with and without shock. Most of these patients were in good functional status.³⁵ Patients who survived the initial crisis did well in our series also. This also emphasises the importance of aggressive revascularisation in patients with CS.

Limitations

Small sample size was the main limitation of the study. The CS was not confirmed invasively in all patients. Only 1-year follow-up was available in all patients. Mechanical causes of CS were excluded from analysis. Shock due to non-STEMI was not included in our study.

Conclusion

The CS remains the leading cause of death in AMI. By preserving ischaemic myocardium, PCI reduces the in-hospital mortality which was maintained in the long-term. Establishment of TIMI 3 flow is of paramount importance. In AMI with CS, even with best available treatment, survival is <50% in our study. This could be due to longer delay between shock onset and PCI. Transfer admissions were the main contributors to this delay. Early risk stratification, better referral systems, optimal use of adjunctive therapies and assist devices could improve the survival in future.

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