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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 wave patient of the patient of

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

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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 enroled 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 90mmHg 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. Multivessel 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±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 nonanterior 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

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