Coronary Heart Disease

Predictors of In-Hospital Mortality in Patients with Successful Primary Coronary Intervention for Acute ST-Elevation Myocardial Infarction Presenting as Cardiogenic Shock

Shao-Chi Yang,[1] Yi-Chih Wang,[1*] Juey-Jen Hwang,[1,2] Kwan-Lih Hsu,[1] Fu-Tien Chiang[1] and Chuen-Den Tseng[1]*

Background: Predictors of hospital mortality in patients with successful primary coronary angioplasty for ST-elevation myocardial infarction (STEMI) presenting as cardiogenic shock (CS) remained less specified.

Methods: From 1996 to 2007, we evaluated 64 patients (56 men, 65 ± 14 years) with STEMI and CS receiving successful intervention defined as residual stenosis < 40% by balloon angioplasty or < 20% by stenting with a TIMI grade 2 or 3 flow in the infarct-related artery.

Results: Among the 64 patients, 37 (58%) had anterior infarction, and 26 (41%) had inferior infarction. In-hospital death occurred to 21 (33%) patients with a mean hospital stay of 11 ± 18 days. Hospital mortality was associated with several pre- and peri-procedural parameters including initial higher white blood cell (16,629 ± 6492 vs. 13,093 ± 5387 1/μL) and serum creatinine (1.7 ± 0.6 vs. 1.2 ± 0.3 mg/dl) levels, anterior infarction (76 vs. 49%), less stenting (38 vs. 65%), and post-intervention TIMI grade 2 flow (57 vs. 14%) (all $p < 0.05$). After multivariate analysis, initial creatinine ≧ 1.4 mg/dl (odds ratio: 8.9; $p = 0.011$, 95% confidence interval: 1.7-47.7), and anterior infarction (odds ratio: 5.9; $p = 0.038$, 95% confidence interval: 1.1-31.3) were independent predictors of in-hospital death.

Conclusion: Anterior infarction or early renal dysfunction remained critical to the one-third mortality of successful intervention for patients with STEMI and CS.

Key Words: Acute myocardial infarction • Cardiogenic shock • Percutaneous coronary intervention

INTRODUCTION

The incidence of cardiogenic shock (CS) complicating acute ST-elevation myocardial infarction (STEMI) is around 6% to 20%,[1] and it remains the leading cause of in-hospital death of patients hospitalized for acute MI, with an early mortality rate of nearly 80% if untreated.[2,3] Some studies have suggested that emergency coronary revascularization could reduce the mortality of patients with cardiogenic skock.[4,5] The randomized Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial also demonstrated that early revascularization resulted in a nonsignificant reduction in 30-day mortality, from 55% to 46%, when compared with medical stabilization for patients with cardiogenic shock.[6,7]

Theoretically, success of primary coronary angio-

Received: February 28, 2009 Accepted: May 4, 2010

[1]Cardiovascular Division, Department of Internal Medicine, National Taiwan University Hospital, Taipei; [2]Department of Internal Medicine, National Taiwan University Hospital, Yun-Lin Branch, Yun-Lin, Taiwan.

Address correspondence and reprint requests to: Dr. Chuen-Den Tseng, Cardiovascular Division, Department of Internal Medicine, National Taiwan University Hospital, No. 7, Chung-Shan South Road, Taipei 100, Taiwan. Tel: 886-2-2312-3456 ext. 3951, 5035; Fax: 886-2-2393-4176; E-mail: cdtseng@ha.mc.ntu.edu.tw

[*]These two authors contributed equally to this work.

plasty would be expected to provide more favorable prognosis for patients with STEMI presenting as CS. However, few studies have specified the in-hospital course and clarified factors predisposing to worse outcome in such patients, even after successful intervention. In the study, among 123 patients who had documented acute STEMI accompanied by CS, we investigated the in-hospital prognosis of 64 patients who received successful primary percutaneous coronary intervention.

METHODS

Patients

Identified from the medical records during 1996 to 2007 in National Taiwan University Hospital (NTUH) and among 123 patients who presented to our emergency department with confirmed diagnosis of acute STEMI accompanied with cardiogenic shock, we included 64 patients who received successful primary coronary intervention.

Definitions

Acute STEMI was defined as at least 30 minutes but < 12 hours of symptoms, and presence of ST-segment elevation ≧ 1 mm in at least 2 contiguous leads or presumed new bundle-branch block on the presenting 12-lead electrocardiogram. Right ventricular infarction was diagnosed based on the finding of ST-elevation in lead V4R. Cardiogenic shock was defined as systolic blood pressure < 90 mmHg for > 30 minutes accompanied with signs of end-organ hypoperfusion which was considered to be resulted from ventricular dysfunction and unresponsive to fluid replacement alone before usage of positive inotropic agents or mechanical support.

Primary coronary intervention

Following evaluation of feasibility by a cardiologist and obtaining informed consent, coronary angiography was performed initially via the femoral artery according to standard practice within 18 hours after onset of symptoms. Diseased coronary artery was defined as ≧ 50% diameter stenosis (DS), and the left main (LM) coronary artery was classified as having two-vessel disease if the left coronary was non-dominant and as having three-vessel disease if dominant. Multivessel disease was defined as ≧ 50% diameter stenosis in two or more major epicardial coronary arteries. The flow of the infarct-related artery (IRA) was categorized from grade 0 to grade 3 by the Thrombolysis in Myocardial Infarction (TIMI) grading system. After diagnostic coronary angiography, revascularization procedure was tailored to the individual patient and selected solely by the physicians. Procedural success was defined as a reduction to residual stenosis of < 40% by balloon angioplasty or successful stent deployment at the desired position with a residual stenosis < 20% followed by TIMI grade 2 or 3 flow in the IRA.

Concomitant medical therapy or mechanical devices

All patients were treated with aspirin 300 mg orally as the loading dosage and then 100 mg daily. Unfractionated heparin 5000 IU was given intravenously, and then infused continuously for 48 hours if there was no active contraindication. According to the clinical condition of each patient, prescriptions and dosages of inotropic agents, or implantation of intraaortic balloon pump (IABP) or extracorporeal membrane oxygenation (ECMO) machine were advised by attending physicians in order to optimize hemodynamics for maintenance of end-organ perfusion. Ticlopidine 250 mg twice per day or clopidogrel 300 mg loading dose, followed by 75 mg per day was administered after stent placement. In survivors with stabilized blood pressure, usage of cardiovascular medications such as angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, β-blockers, or diuretics were titrated and individualized.

Data collection and outcome evaluation

Detailed in-hospital data regarding serum creatinine level, white blood cell (WBC) count, reperfusion time, peak cardiac enzyme level and its timing, and in-hospital adverse events such as sepsis, atrial or ventricular arrythmias were obtained. Diagnosis of MI-related cardiac rupture or acute mitral regurgitation was confirmed by echocardiography. Major bleeding was defined as significant blood loss from any site not caused by trauma and requiring transfusion. Left ventricular ejection fraction (LVEF) was measured after intervention or before discharge by the area-length method using a Hewlett-Packard model Sonos 4500 or 5500 with a 2-4-MHz

transducer. The duration of hospital stay and causes of in-hospital death were recorded.

Statistical analyses

All values were expressed as mean ± standard deviation. Comparisons between parametric variables were performed using unpaired Student's *t* test, while comparisons between categorical variables were performed using the chi-square test with Yates' correction if needed. Multivariate logistic regression analysis was used to include factors with a p value less than 0.10. All analyses were performed with the SPSS 10.0 software package (SPSS Inc., Chicago, Illinois).

RESULTS

Baseline characteristics of patients with successful primary coronary intervention

Among the 64 patients (Table 1), there were 56 males and 8 females with a mean age of 65 ± 14 years. The clinical features were listed. Nearly 90% of patients experienced preinfarction angina, and the others presented with other symptoms such as shortness of breath or syncope. The mean time from onset of symptoms to arrival to emergency department was 185 ± 138 minutes.

Comparisons of clinical and peri-procedural characteristics

Overall, 37 (58%) patients had anterior infarction, 1 (2%) had lateral infarction, and 26 (41%) had inferior infarction, including right ventricular infarction in 18 of the 26 (69%) patients. Even after successful coronary intervention, there were still 21 patients (33%) who did not survive to discharge. Between patients with and without in-hospital mortality (Table 1), there was similarity in age, gender, preexisting systemic and cardiovascular diseases. Patients with hospital mortality had higher initial WBC count (16,629 ± 6492 vs. 13,093 ± 5387/μL, $p < 0.029$) and serum creatinine level (1.7 ± 0.6 vs. 1.2 ± 0.3 mg/dl, $p < 0.001$) than those in survivors. Regarding the intervention (Table 2), despite high prevalence (> 70%) of multi-vessel diseases existing in all of the patients, those with in-hospital death had IRA as LM/left anterior descending artery (LAD) preponderance (76 vs. 49%, $p = 0.038$), less performance of coronary stenting (38 vs. 65%, $p < 0.041$), and lower post-intervention TIMI flow (2.4 ± 0.5 vs. 2.9 ± 0.4, $p < 0.001$). However, the mean door-to-balloon time, pre-PCI TIMI flow, incidence of multi-vessel disease, and

Table 1. Clinical features of the 64 patients with successful coronary intervention and comparisons between patients with and without in-hospital death

	Total n = 64	In-Hospital death (+) n = 21	In-Hospital death (-) n = 43	*p*-value
Sex, male/female	56/8	16/5	40/3	.057
Age, yrs	65 ± 14	66 ± 15	65 ± 13	.859
Hypertension	31 (48%)	9 (43%)	22 (51%)	.540
DM	23 (36%)	7 (33%)	16 (37%)	.766
Smoking	33 (52%)	10 (48%)	23 (53%)	.665
Hyperlipidemia	15 (23%)	4 (19%)	11 (26%)	.570
Prior MI	5 (8%)	3 (14%)	2 (5%)	.183
Prior CVA	5 (8%)	0 (0%)	5 (12%)	.107
Prior PCI	2 (3%)	1 (5%)	1 (2%)	.606
Prior CABG	1 (2%)	1 (5%)	0 (0%)	.154
Preinfarction angina	56 (88%)	18 (86%)	38 (88%)	.767
Onset to arrival, min	185 ± 138	190 ± 141	182 ± 138	.816
Initial WBC, cell/μL	14272 ± 5965	16629 ± 6492	13093 ± 5387	.029
Creatinine, mg/dl	1.4 ± 0.5	1.7 ± 0.6	1.2 ± 0.3	< .001

CABG, coronary artery bypass graft; CVA, cerebrovascular accident; DM, diabetes mellitus; MI, myocardial infarction; PCI, percutaneous coronary intervention; WBC, white blood cell.

Table 2. Comparisons of periprocedural data between patients who had successful coronary intervention with and without in-hospital death

	In-Hospital death (+) n = 21	In-Hospital death (-) n = 43	*p*-value
Door-to-balloon, min	137 ± 108	169 ± 268	.602
Onset-to-balloon, min	328 ± 165	351 ± 357	.777
Infarct-related artery			
LM	5 (24%)	3 (7%)	.057
LAD proximal/mid	8/3 (38/14%)	17/1(40/2%)	.435
LM/LAD	16 (76%)	21 (49%)	.038
LCX	0 (0%)	1 (2%)	.489
RCA	5 (24%)	21 (49%)	.057
RV infarction	4 (19%)	14 (33%)	.266
Pre-PCI TIMI flow			.185
Grade 0	17 (81%)	29 (67%)	
Grade 1	3 (14%)	7 (16%)	
Grade 2	1 (5%)	7 (16%)	
Multivessel disease	17 (81%)	30 (70%)	.349
Pre-PCI stenosis	99 ± 3 (%)	97 ± 6 (%)	.404
Total Stenting	8 (38%)	28 (65%)	.041
Residual stenosis	6 ± 9 (%)	4 ± 9 (%)	.422
Post-PCI TIMI flow			< .001
Grade 2	12 (57%)	6 (14%)	
Grade 3	9 (43%)	37 (86%)	

CK, creatinine kinase; LAD, left anterior descending artery; LCX, left circumflex artery; LM, left main coronary artery; PCI, percutaneous coronary intervention; RCA, right coronary artery; RV, right ventricle; TIMI, thrombolysis in myocardial infarction.

pre- and post-PCI diameter stenoses were similar between the two groups.

Hospital course and causes of mortality

During the course from the emergency department to successful intervention in the catheterization laboratory, there were only 2 patients who received cardiopulmonary-cerebral resuscitation (CPCR) in the survivor group, compared to 14 patients with CPCR in the group of hospital death (5% vs. 67%, $p < 0.001$) (Table 3). Since the study patients had cardiogenic shock, 44 patients, including all of the 38 patients with left-side coronary artery infarction and 6 patients with right coronary artery infarction combined with multivessel disease, poor LV function at baseline, or acute renal failure, had variable degree of acute lung edema initially. However, there was no significant difference of the presentation of acute lung edema between the survivor and the in-hospital death groups. Overall, 37 patients were intubated within 3 days after admission due to CPCR or lung edema. Among the 37 patients, 18 patients were in the survivor group and 19 patients were in the hospital death group (42% vs. 90%, $p < 0.001$). There were 18 patients receiving renal replacement therapy in the acute stage ($\leqq$ 3 days after admission) in the study. Among the 18 patients, only patient was in the survivor group and the other 17 were in the hospital death group (2% vs. 81%, $p < 0.001$).

During hospitalization, patients with mortality had much higher peak level of creatinine kinase (12598 ± 9555 vs. 6997 ± 5979 U/L, $p < 0.001$). Among the peri-infarct cardiac arrhythmias, only a higher occurrence rate of ventricular tachycardia/fibrillation (81% vs. 44%, $p < 0.005$) was found to be associated with in-hospital mortality. Overall, of 47 patients (73%) received hemodynamic augmentation by IABP, and 7 patients (11%) received implantation of ECMO. Both devices were reasonably more frequently needed in patients with hospital mortality, mainly due to a higher degree of LV dysfunction (LVEF: 29 ± 13 vs. 44 ± 12%, $p < 0.001$). Sepsis

Table 3. Comparisons of admission course between patients who had successful coronary intervention with and without in-hospital death

	In-Hospital death (+) n = 21	In-Hospital death (-) n = 43	*p*-value
CPCR*	14 (67%)	2 (5%)	< .001
Acute lung edema§	17 (81%)	27 (63%)	.146
Intubation#	19 (90%)	18 (42%)	< .001
Dialysis#	17 (81%)	1 (2%)	< .001
Peak CK, U/L	12598 ± 9555	6997 ± 5979	.018
Peaking time, hrs	15.7 ± 9.3	15.1 ± 4.6	.752
VT/VF	17 (81%)	19 (44%)	.005
PAF	1 (5%)	6 (14%)	.276
CAVB	4 (19%)	7 (16%)	.787
IABP	20 (95%)	27 (62%)	.004
ECMO	6 (29%)	1 (2%)	.001
LV ejection fraction, %	29 ± 13	44 ± 12	.001
Sepsis	5 (24%)	1 (2%)	.005
Free wall rupture	1 (5%)	0 (0%)	.154
Major bleeding	2 (10%)	0 (0%)	.040
Hypoxic encephalopathy	0 (0%)	2 (5%)	.323
Stroke	0 (0%)	1 (2%)	.489
Mean hospital stay, day	11 ± 18	26 ± 43	.106
Causes of death			
Cardiogenic shock	16 (76%)		
Free wall rupture	1 (5%)		
Septic shock	4 (19%)		

CAVB, complete atrioventricular block; CPCR, cardio-pulmonary-cerebral resuscitation; ECMO, extracorporeal membrane oxygenation; IABP, intraaortic balloon pump; LV, left ventricular; PAF, paroxysmal atrial fibrillation; VT/VF, ventricular tachycardia/ventricular fibrillation.

* at emergency department or during catheterization.

§ initial presentation.

within 3 days after admission.

happened to 6 of the 64 patients (9%) during admission, and its occurrence (24 vs. 2%, $p < 0.005$) played a contributory role for in-hospital death (19%). Profound cardiogenic shock still remained the main cause (76%) of in-hospital death, and only one patient (2%) suffered from free wall rupture leading to subsequent mortality. Mortality of patients with acute STEMI and CS usually developed with a mean of less than 2 weeks (11 ± 18 days) from admission, and the survivors from the events experienced a much prolonged course of hospitalization (26 ± 43 days).

Multivariate analysis of baseline and procedural predictors for in-hospital death

When arbitrarily set 1.4 mg/dl as the cut-off point for initial serum creatinine, and also enrolled age, gender, WBC count, LM/LAD as IRA, and the existence of post-intervention TIMI III flow for multivariate analysis, we found that initial serum creatinine ≧ 1.4 mg/dl (odds ratio: 8.9; $p < 0.011$, 95% confidence interval: 1.7-47.7), LM/LAD as IRA (odds ratio: 5.9; $p < 0.038$, 95% confidence interval: 1.1-31.3) remained as independent predictors of in-hospital death in patients who received successful coronary intervention for STEMI complicated by CS.

Multivariate analysis of hospital course predictors for in-hospital death

When we took possible associated factors of in-hospital mortality from Table 3 such as CPCR, intubation,

dialysis, peal CK level, VT/VF, LV ejection fraction, and sepsis into consideration for multivariate analysis, only the occurrence of dialysis during admission remained independent to be associated with in-hospital death (OR = 91, $p < 0.001$).

DISCUSSION

From the more than 10-year experience of managing acute STEMI complicated by CS at a single high-volume center, we demonstrated that successful primary percutaneous coronary intervention was still accompanied with one third of in-hospital mortality, occurring mostly within 2 weeks. Infarction involving LM/LAD territory or initial presentation of renal insufficiency carried worse prognosis for this group of patients.

Primary angioplasty has been increasingly advocated as an approach that should supersede thrombolysis for the treatment of cardiogenic shock complicating acute STEMI since the end of 1980s.[8,9] A pooled report[10] showed that patients with successful coronary angioplasty had a lower mortality (37%) than with either unsuccessful procedure (79%) or only conservative treatment (83%). With a similar success rate (64/71, 90%) of intervention in the present study as prior studies,[11] we demonstrated a similar in-hospital mortality of 33% after successful angioplasty in patients with acute STEMI complicated by early CS.

From previous studies, clinical features such as increasing age or renal dysfunction, ECG or echocardiographic parameters such as sum of ST depression or depressed LV function, procedural factors such as LM-LAD occlusion or use of nonstent devices were ever shown to be significantly associated with in-hospital mortality in univariate analyses in patients receiving emergent percutaneous angioplasty for acute STEMI presenting as CS.[12-14] In our specified group who had successful intervention, with partial similarity as before, we found several clinical and procedural characteristics predicting in-hospital mortality, including higher presenting WBC or creatinine level, no use of stents, and post-PCI grade II TIMI flow. Inflammatory markers such elevated baseline WBC counts in patients with STEMI have been associated with a poor prognosis.[15,16] Further in the present study, we also showed an association between higher WBC and hospital mortality even in STEMI patients with CS after successful reperfusion. The use of coronary stents could provide better angiographic results than those obtained by balloon angioplasty alone, improve the rate of post-PCI TIMI flow, and reduce in-hospital recurrent ischemic events due to abrupt closure of the dilated artery.[17] Less coronary stenting and resultant more post-angioplasty TIMI II flow was also demonstrated to have partial contribute to the in-hospital death in the present study patients.

Initial higher creatinine level and LM/LAD as IRA were the two independent predictors of mortality in the current study. Renal dysfunction and more impaired LV function were proved to increase mortality risk during STEMI.[18] With regard to renal dysfunction, even mild renal impairment is associated with more adverse cardiac events in patients with STEMI treated medically with thrombolysis[19] and with primary angioplasty.[20] There are many factors that have been reported to be associated with poor prognosis in patients with STEMI and cardiogenic shock. In the present study, our purpose was to see if any early clinical or procedural factors, which were present within several hours after arriving at the hospital, could serve as prognosticators for the in-hospital course. Naturally, renal dysfunction can be confounded by several factors, such as a poorer baseline renal function, more profound cardiac dysfunction, and more severe or prolonged shock status before admission, etc. Since initial higher serum creatinine level was the combined consequences of multiple worse clinical factors rather than the cause of in-hospital mortality, it might imply a poor prognosis during hospitalization potentially. Our data (Table 3) showed the fact that more than 80% of patients with in-hospital death had renal failure progressing into dialysis dependence within 3 days after admission compared to only 2% in survivors, which also addressed the role of early-presenting renal dysfunction on poorer outcome in patients presenting with STEMI and cardiogenic shock. Further, we included all possible clinical and procedural factors, which were similar to the prognosticators mentioned in previous studies, into multivariate analysis and showed persistent significance of the association between initial Cr level and in-hospital death even after successful intervention.

With respect to LV dysfunction, LVEF is an important factor related to prognosis in patients with AMI in many prior studies. However, owing to the retrospective nature of the current study, we had missing data in LVEF in 27 patients. Usually in STEMI, we performed complete echo study after coronary intervention, and thus this would result in LVEF not being available if patients died early. In addition, the long enrollment period also contributed partially to the loss of data a long time ago. Nevertheless, due to the significant amount of missing data and as a "post-PCI" parameter essentially, we did not take LVEF as an initial clinical or procedural factor for multivariate analysis although it also showed significant association with prognosis in the present study. On the other hand, the preponderance of LM/LAD infarction, which could lead to a higher peak CK level, poorer LVEF, and more use of invasive devices demonstrated in the present study, also had a considerable impact on hospital death even in those undergoing successful reperfusion for STEMI accompanied by CS.

STUDY LIMITATIONS

Interpretation from the above results has limitations. Patients with multiple comorbidities or much more critical conditions were likely to be excluded from intensive revascularization, and this kind of selection bias may lead to overestimating the favorable outcome attributed to revascularization. Due to more intravenous catheters or augmented devices being used for the more complicated conditions of our study population, the relatively longer door-to-balloon time in the present study than that recommended in current guideline for STEMI could also have interfered with the results. Because some imaging information many years ago was lost, the description of coronary anatomy was oversimplified in the study without use of the SYNTAX score[21] developed in 2005, which can provide a more comprehensive angiographic scoring system and a promising tool for risk and outcome stratification in patients undergoing contemporary coronary intervention for extensive coronary artery disease, such as the majority with multivessel disease in our study. We took both genders together and used the same cut-off value of serum creatinine for analysis, which might overlook the gender difference. However, the small-size population and only 8 women in our study disabled us from further separating the male from female gender with different creatinine levels for statistical analysis. Further, the effects of device evolution or technical improvement during the relatively long collection period couldn't be appropriately taken into consideration for outcome evaluation. For example, coronary stenting was only performed in less than 60% of all patients and much lower (38%) in those with in-hospital death in the study. Thus the results and findings must be interpretated as descriptive as opposed to demonstrating effectiveness. Nevertheless, the patients included in this study represented a high-risk population who had successful coronary reperfusion and expected favorable prognosis, and the data are likely to be representive of outcomes in this single medical center with high-volume catheterization laboratory and experienced operators.

CONCLUSION

In the era of primary coronary intervention, one third of mortality still occur after the success of intervention for patients with STEMI presenting as CS. Multiple clinical and procedural factors, especially the presence of initial renal dysfunction and LM/LAD infarction, played critical roles on the in-hospital deaths. Whether newer pharmacotherapy such as antiplatelet agents, device therapy such as embolic protecting filters or thrombectomy catheters, or the strategy of complete revascularization for the high prevalence of multi-vessel involvement in such group of patients can improve the prognosis for patients with STEMI and CS, especially those accompanied with anterior infarction or early renal dysfunction, warrants further investigation.

ACKNOWLEDGEMENTS

We would like to thank the Intervention Team for AMI, especially Dr. Lung-Chun Lin, Dr. Tzung-Dau Wang, Dr. Chia-Lun Chao, Dr. Hsien-Li Kao, Dr. Chii-Ming Lee, and Dr. Chiau-Suong Liau, for performing coronary intervention and management in the patients of this study.

REFERENCES

1. Califf RM, Bengtson JR. Cardiogenic shock. *N Engl J Med* 1994;330:1724-30.
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. Holmes DRJ, Bates ER, Kleiman NS, et al. Contemporary reperfusion therapy for cardiogenic shock: the GUSTO-I trial experience. The GUSTO-I investigators. Global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries. *J Am Coll Cardio*l 1995;26:668-74.
4. Apolito RA, Greenberg MA, Menegus MA, et al. Impact of the New York State Cardiac Surgery and Percutaneous Coronary Intervention Reporting System on the management of patients with acute myocardial infarction complicated by cardiogenic shock. *Am Heart J* 2008;155:267-73
5. Fang J, Mensah GA, Alderman MH, Croft CB. Trends in acute myocardial infarction complicated by cardiogenic shock, 1979-2003, United States. *Am Heart J* 2006;152:1035-41.
6. Hochman JS, Sleeper LA, White HD, et al. One-year survival following early revascularization for cardiogenic shock. *JAMA* 2001;285:190-2.
7. Sleeper LA, Ramanathan K, Picard MH, et al. Functional capacity and quality of life following emergency revascularization for cardiogenic shock complicating acute myocardial infarction. *J Am Coll Cardiol* 2005;46:266-73.
8. Hibbard MD, Holmes DR Jr, Bailey KR. Percutaneous transluminal coronary angioplasty in patients with cardiogenic shock. *J Am Coll Cardiol* 1992;19:639-46.
9. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization and long-termsurvival in cardiogenic shock complicating acute myocardial infarction. *JAMA* 2006;295:2511-5.
10. Barry WL, Sarembock IJ. Cardiogenic shock: therapy and prevention. *Clin Cardiol* 1998;21:72-80.
11. Antoniucci D, Valenti R, Santoro GM. Systematic direct angioplasty and stent-supported direct angioplasty therapy for cardiogenic shock complicating acute myocardial infarction: in-hospital and long-term survival. *J Am Coll Cardiol* 1998;31:294-300.
12. White HD, Palmeri ST, Sleeper LA, et al. Electrocardiographic findings in cardiogenic shock, risk prediction, and the effects of emergency revascularization: results from the SHOCK trial. *Am Heart J* 2004;148:810-7.
13. Klein LW, Shaw RE, Krone RJ, et al. Mortality after Emergent percutaneous coronary intervention in cardiogenic shock secondary to acute myocardial infarction and usefulness of a mortality prediction model. *Am J Cardiol* 2005;96:35-41.
14. Sutton AGC, Finn P, Hall JA, et al. Predictors of outcome after percutaneous treatment for cardiogenic shock. *Heart* 2005;91: 339-44.
15. Sabatine MS, Morrow DA, Cannon CP, et al. Relationship between baseline white blood cell count and degree of coronary artery disease and mortality in patients with acute coronary syndromes: a TACTICS-TIMI 18 (Treat Angina with Aggrastat and determine Cost of Therapy with an Invasive or Conservative Strategy – Thrombolysis in Myocardial Infarction 18 trial) substudy. *J Am Coll Cardiol* 2002;40:1761-8.
16. Smit JJ, Ottervanger JP, Slingerland RJ, et al. Comparison of usefulness of C-reactive protein versus white blood cell count to predict outcome after primary percutaneous coronary intervention for ST elevation myocardial infarction. *Am J Cardiol* 2008;101:446-51.
17. Suryapranata H, van't Hof AW, Hoorntje JC. Randomized comparison of coronary stenting with balloon angioplasty in selected patients with acute myocardial infarction. *Circulation* 1998;73: 2502-5.
18. Marenzi G, Moltrasio M, Assanelli E, et al. Impact of cardiac and renal dysfunction on inhospital morbidity and mortality of patients with acute myocardial infarction undergoing primary angioplasty. *Am Heart J* 2007;153:755-62.
19. Gibson CM, Pinto DS, Murphy SA, et al. Association of creatinine and creatinine clearance on presentation in acute myocardial infarction with subsequent mortality. *J Am Coll Cardiol* 2003;42:1535-43.
20. Yamaguchi J, Kasanuki H, Ishii Y, et al. Prognostic significance of serum creatinine concentration for in-hospital mortality in patients with acute myocardial infarction who underwent successful primary percutaneous coronary intervention (from the Heart Institute of Japan Acute Myocardial Infarction [HIJAMI] registry). *Am J Cardiol* 2004;93:1526-8.
21. Sianos G, Morel MA, Kappetein AP, et al. The Syntax score: an angiographic tool grading the complexity of coronary artery disease. *Eurointervention* 2005;1:219-227.