

Original paper

Cardiogenic Shock Complicating Acute Myocardial Infarction; Aetiology, Treatment and Prognosis

Shokry F. Nassir^{^*}

[^]Babylon University, College of Medicine, Al-Hilla, Iraq.

Abstract

Background: Cardiogenic shock is the leading cause of death in patients hospitalized for acute myocardial infarction. Treatment strategies using intra-aortic balloon counter pulsation and emergency revascularization by percutaneous coronary interventions or coronary bypass surgery have been shown to improve outcomes.

Aim: to provide an overview of patients with cardiogenic shock complicating acute myocardial infarction admitted to Ibn Al-Bitar center for cardiac surgery in Baghdad.

Methods: This is a prospective observational study conducted at Ibn Al-Bitar center for cardiac surgery in Baghdad. The criteria of the British cardiovascular intervention society were used to identify patients who were admitted to the hospital within one year period with the main focus of the study was on patients with cardiogenic shock complicating acute myocardial infarction. Full clinical assessment was done at bedside. Twelve leads electrocardiography and basic biochemical tests were obtained and echocardiography was done for every patient.

Results: During a period of one year from March 2010 through March 2011 One hundred seventeen patients with cardiogenic shock were identified. Acute myocardial infarction accounted for shock in 78(66.7%). In this patients population there were good percentages of history of myocardial infarction, hypertension, diabetes and smoking. Anterior ST-elevation myocardial infarction was found in two-thirds of them. Predominant left ventricular failure caused cardiogenic shock in 54(69.2%), while ventricular septal rupture, acute mitral regurgitation and right ventricular failure accounted for the rest. The overall mortality rate of cardiogenic shock was 64 (82.1%). Intra-aortic balloon pumping (IABP) was placed in 28 (35.9%) and Thrombolytic therapy was used in 19 patients (24.4%), while Both IABP and thrombolytic therapy were used in 8 (10.3%). Coronary angiography was done in 18 (23.07%). Percutaneous coronary intervention was used in 10 (12.8%), while coronary artery bypass grafting was done in 3(3.8%) of the patients.

Conclusions: This study shows a high in-hospital mortality for patients with cardiogenic shock complicating acute myocardial infarction and despite the proven benefit of revascularization procedures there were only few patients had undergone these

Procedures. The utilization rates of thrombolytic therapy and intra-aortic balloon pump were also low.

Key words: cardiogenic shock, acute myocardial infarction

Introduction

The cardiogenic shock syndrome had been well-defined as failure of the cardiac muscle--due to loss of its function of pumping-- to provide adequate blood stream to tissue to give the metabolic

requirements at rest. Hence, the wholesome definition of the syndrome of cardiac shock comprises a low heart output and signs of hypoxic tissues accompanied with sufficient intravascular volume¹.

In the presence of hemodynamic monitoring, the diagnosis is specified by

the combined presence of reduced systolic arterial pressure (< 90 mmHg or 30 mmHg less than basal value for a minimum of 30min.), a raised difference in arterio-venous oxygen (>5.5 ml per dl), and a low heart index (< 2.2 surface area) accompanied by an elevation of the capillary pulmonary wedge pressure (>15 mmHg)⁽¹⁾.

Postmortem studies showed that the syndrome of cardiogenic shock is accompanied by loss of greater than 40 % of the heart muscle of left ventricle, 1, 2, 3 cardiogenic shock due to principally infarctions of the right ventricular, has recently been recognized⁽⁴⁾.

Supportive and resuscitative efforts must be started directly, at the same time with the diagnostic assessment. When modest hypotension and hypo-perfusion were found, dopamine is better, since peripheral vasoconstrictions is often required to preserve important organs perfusion^(5,6). Norepinephrines are used when intense low blood pressure do existing to maintain blood pressures. The inhibitors of phosphodiesterases and milrinone result in improvement in heart output with pulmonary pressure⁷ with fewer effects on myocardial workload⁸. The preservation of the sinus rhythms or the synchronous pacings, and the of RV function at systole^(9,10).

Though the usage of the intraaortic pumping balloon can yield a transiently the hemodynamic and clinical wellbeing,⁽¹¹⁻¹⁷⁾ survival was sustained only among the patients who consequently underwent revascularization in one observational study⁽¹⁸⁾.

The most effective approach to effectively manage acute myocardial infarction is quicker establishment of blood stream to the infarcted vessel⁽¹⁹⁾, and primary cardiac intervention resulting in superior prognosis than lytic drugs⁽¹⁹⁾. The innovative SHOCK trial prospectively randomized 302 cases with cardiogenic shock due to failure of the left ventricular with acute cardiac infarctions to emergent

early re-vascularization (ERV) with CABG or PCI vs. initial stabilization by medical treatment (IMS) with IABP and drug therapy. 64 % of the patients underwent PCI while CABG was done in 36% of the patients⁽²⁰⁾. In the National Registry of the Myocardial Infarction (NRM), primary coronary intervention use rise from 27.4% at year 1995 to 54.4% at year 2004. CABG was done in 3.2 % of the cases⁽²¹⁻²³⁾.

Immediate referral to the catheterization cardiac center for cardiac vessels visualization and emergent revascularization has essentially replaced thrombolytics as first-line management for cases with acute MI and shock⁽²⁴⁻²⁵⁾. The policy of administering fibrinolytic therapy with or without intraaortic balloon pumping is indicated if patient presents to a hospital that have no lab. for catheterization or when there is inevitable delay in transference to the cath. Laboratory⁽²⁶⁾.

Infarction of the Right Ventricle:- Infarction of the right ventricle can be associated with inferior MI and results in shock state. It has been predicted that 10-15 % of inferior MI were accompanied by right ventricular infarction⁽²⁷⁾. Up to one Liter of normal saline must be given. Inotropic drugs with intraaortic balloon pump are important in cases who have no response to fluid trial⁽²⁸⁾. Intraaortic balloon pump assist in decreasing the tension on the wall and rise perfusion pressure to the coronaries. Heart block and bradycardia must be treated,⁽²⁷⁻²⁸⁾.

Severe Acute Mitral regurgitation:- Severe acute MR, because of break of the papillary muscle head, was considered to be an infrequent reason for cardiogenic shock in myocardial infarction. In Shock trial Registry, of the 1,190 cases with shock, 6.9% had severe acute MR⁽²⁹⁾.

Ruptured Ventricular Septum Post infarction:- The interventricular septum rupture (VSR) may occur in acute myocardial infarction and may result in cardiogenic shock. It frequently happens in

the first 24Hrs of MI. VSR may be associated with either inferior or anterior wall infarction. The patient will presents with cardiogenic shock and pulmonary oedema, with a clear pansystolic murmur is found on examination ⁽³⁰⁾. 31 from 55 cases with VSR had surgical repair, with a mortality rate of 81% and only 4% of the caseslived without surgical correction ⁽³¹⁾.

Cardiac Tamponade and Rupture of the LV free wall:- Rupture of the free LV wall considered to be an infrequent, fatal consequences of myocardial infarction. It has been expected to happen in 1- 6% of cases with acute infarction ⁽³²⁾.

Patients and Methods

This study was an observational, prospective descriptive study. It was conducted at IbnAL-Bitar Center for Cardiac Surgery from March 2010 to March 2011. Patients who fulfilled the criteria of cardiogenic shock as defined below were involved in this research and the main focus of the research was on patients with myocardial infarction.

The study was strictly observational and there was no intension to interfere with the management of the patients or to compare the different modalities of therapy of the patients who were treated according to the discretion of the attending physician.

Any patient with cardiogenic shock was included in this study. The cases were necessarily to fulfill the ensuing principles for cardiac shock as it has been well-defined by the British Society of Cardiovascular Intervention: pulse ≥ 100 beats/min; Blood pressure < 90 mm Hg for a minimum of 30 min.; and the patient sweaty and cool, or requiring intra-aortic balloon pump (IABP), inotropes, or cardiopulmonary support to the circulation. Quantification of urinary output was not obligatory.

There were no necessity for measuring of heart filling pressure or heart output by any invasive procedures to diagnose cardiac shock .quantification of heart

output and left ventricle filling pressures were not obtained in the current study for two reasons: first, placement of a central line in the setting of thrombolytic therapy predisposes the patient to the risk of bleeding; and second, such an approach would have, of necessity, delayed the administration of therapy.

After clinical evaluation, an ECG, troponin test, basic blood tests and echocardiography were done for every patient. CT pulmonary angiography was done for all patients with suspected pulmonary embolism. Patients with shock from causes other than cardiac causes or cardiac catheterization laboratory complication or correctable arrhythmic causes constituted clinical exclusion criteria.

The major causes of Shock in myocardial infarction were defined as the following: Predominant LV failure, Isolated right ventricle shock and mechanical causes (VSR, Severe acute mitral regurgitation, or tamponade/ LV rupture). Principal LV dysfunction was considered as the cause of cardiogenic shock when no one of the other main shock groups were found: mechanical factors (VSR, Severe acute MR, or Left ventricle rupture/tamponade), isolated RV shock, previous severe valvular heart disease, excessive calcium or beta blockers, or shock results from complication in cardiac cath. lab.

The diagnosis of acute MI was established on a characteristic history of chest pain correlated with the appearance of diagnostic ECG changes and characteristic changes in serum enzymes. ECG locations of ST- elevation myocardial infarction were well-defined according to the Global Utilization of Streptokinase and t-PA for Occluded Coronary arteries-I trial (GUSTO I) ⁽³³⁾: V1-V4Anterior; II, III, aVF Inferior; V5-V6 apical; I,aVL lateral; V1-V2 posterior. Renal insufficiency was defined as serum Creatinine above1.2 mg/dl. In a sum of cases there were more than one cause for the shock, and for the purpose of assessment of fatality rate these

cases were classified as to have 1 reason was according to the following: 1) p LV dysfunction, 2) severe MR, 3) VSR, 4) Isolated right ventricular dysfunction and 5) Other reasons of shock.

Statistical Analysis

All data were analyzed using excel program. Categorical variables are offered in number of cases and percentages and compared using the chi-square test. Other constant variables are offered in mean \pm stander deviation or as range.

Results

During the period of the study from March 2010 through March 2011 One hundred seventeen cases who fulfilled the cardiogenic shock criteria were collected. The frequencies of the major causes of shock were assessed (Figure 1). There were 78 (66.7 %) patients with shock due to AMI and this group was used for analysis in this study. DCM was diagnosed in 18(15.3 %), PE in 13(11.1%), Valvular causes in 7(6%) and Left atrial Myxoma in 1 (0.9%). Baseline characteristics of AMI patients are revealed in table-1. The mean age was 59.59 ± 7.29 year. There were 50 (64.1%) male and 28 (35.9%) female. Two thirds of cases were referred from other hospitals 54 (69.2%), while direct admission represents 24 (30.8%) of patients. There were good percentages of history of hypertension MI, smoking and diabetes .Renal insufficiency was diagnosed in 41(52.6 %) of patients with the majority 35(85.3%) had serum creatinine ≥ 2 mg/dl. The mean LVEF was 34.97 ± 4.60 (24%-45%). Haemodynamic parameters showed that the mean systolic blood pressure was 71.35 ± 11.19 mmHg, the mean diastolic blood pressure was 44.55 ± 12.01 mmHg and the mean heart rate was 110.32 ± 15.85 beat per minute. The mean hospital stay was 4.77 ± 2.82 (<1-14) days. **Characteristics of MI:** Table 2 shows the major MI characteristics. STEMI was diagnosed in 64(82.1%), while Non STEMI was

diagnosed in 14(17.9%). About two thirds of STEMI patients had their MI in anterior location, more than half of them had multiple-sites of infarction and only a minority had right ventricular infarction. 23(76.6%) of inferior MI cases had a history of previous myocardial infarction and all cases of RV infarction are diagnosed in association with acute inferior MI.

Major shock categoriesin MI:- The frequencies of the main groups of cardiac shock were evaluated (Figure 2). Principally left ventricular dysfunction caused cardiogenic shock in 54(69.2%) of all patients. Ventricular septal rupture was present in 13(16.7%), acute mitral regurgitation in 6(7.7%) and right ventricular shock in 5(6.4%). The majority of patients with ventricular septal rupture 12(92.3%) were diagnosed in the setting of anterior MI, while all patients with MR were diagnosed in the setting of inferior MI.

Procedures utilization:- The frequency rates of procedures and drug utilization is shown in table-3 which shows a small percentage of angiography and revascularization.

Catheterization findings:- Coronary angiography was done in 18 patients (23.07%). The mean age of these patients was 47.38 ± 5.43 with a mean EF% of 41.87 ± 4.65 and the mean SBP and DBP were 83.35 ± 6.97 and 56.68 ± 6.86 respectively .Coronary angiography results were revealed in table 4. Half of the case shave three vessels disease and the infarct-related artery (culprit vessel) was the LAD in the majority of the patients 11(61.1%).

Fatality:- The in hospital fatality for the main shock group sarerevealed in Figure 3 with a global mortality rate of cardiogenic shock complicating acute MI of 64 (82.1%). The rates were considerably different among the four causes.

Patients with predominant LVF and those with VSR had higher mortality rates than patients with MR and those with RV shock, which was 85.2% and 84.6%

respectively. The mortality rate of those with MR was 66.7 % while the mortality rate of patients with RV infarction was 60%.

Predictors of in-hospital outcome:-

Figure 4 and table 5 shows the major predictive factors of in-hospital outcome. The usage of thrombolytic therapy in combination with IABP or alone was significantly accompanied with increased in hospital survival rate, while the use of IABP alone and the use of inotropics or CABG does not significantly improve the in-hospital survival. Revascularization using PCI was significantly associated with improved survival.

The presence of renal insufficiency and an ejection fraction of $\leq 35\%$ was significantly associated with increased in-hospital mortality. There was no important difference in in hospital outcome between those who were directly admitted or those who were transferred from other hospitals. An increased age of ≥ 65 years was significantly accompanied with increased in-hospital fatality. The presence of a history of previous MI and a history of DM was significantly accompanied by increasing in the in-hospital fatality.

Discussion

To the best of our information this is the main attempt to be made to characterize patient with cardiogenic shock in our country and although the number collected was not large it is considered significant owing to the resources used to collect the data.

Patients profile:- The baseline characteristics of patients who developed cardiogenic shock in this study (table-1) were very similar to those in other researches of cardiogenic shock⁽³⁴⁻³⁷⁾. Patients with cardiogenic shock are frequently male and elderly and have high rates of diabetes, hypertension and prior MI. However, mean age in this study (59.59 ± 7.29) was lower than the mean age in other studies, for example the mean age in the SHOCK trial registry was (68.7 ± 11.8) and this probably reflects the increased incidence of coronary artery disease at younger age group or to increased survival in western countries⁽³⁸⁾. The majority of the patients 54(69.2%) were referred cases from other hospitals and this can be attributed partly to the increased awareness of the treating physician to the importance of early coronary interference in those high hazard patients. Renal insufficiency which might adversely affect the outcome was present in more than half of the patients 41 (52,6%).

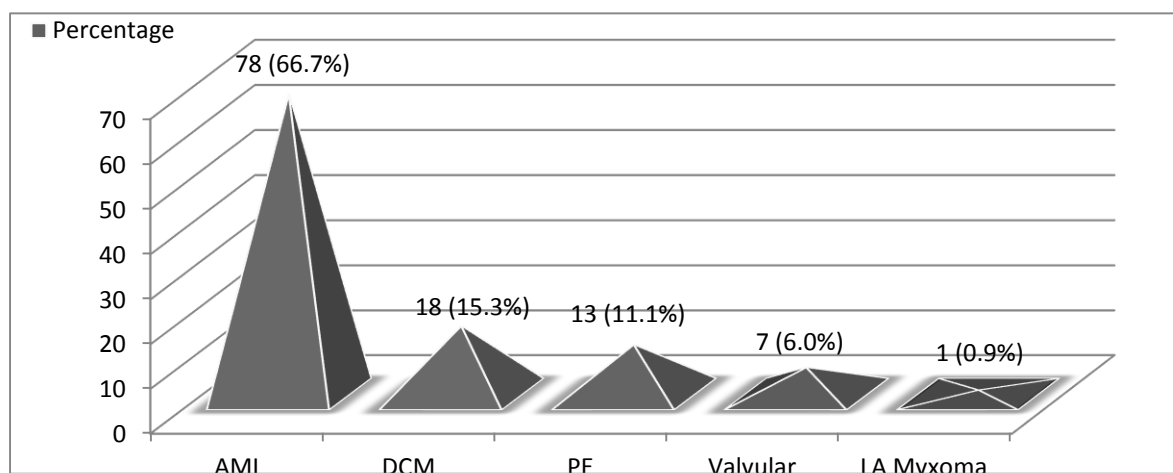


Figure 1. Major causes of shock.

AMI: acute myocardial infarction, DCM: dilated cardiomyopathy, PE: pulmonary embolism, LA: left atrium

Table 1. Baseline Characteristics of patients

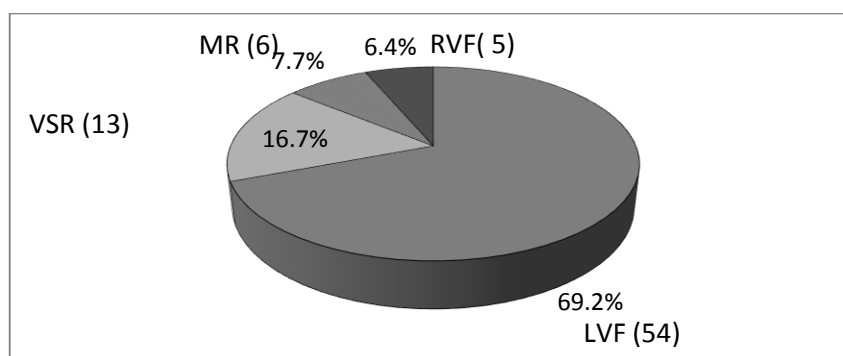
Characteristics		No.	%
Age (years) Mean \pm SD (Range)		59.59 \pm 7.29 (28-74)	
Gender	Male	50	64.1
	Female	28	35.9
Admission	Direct	24	30.8
	Referred	54	69.2
Diabetes mellitus		32	41.0
Hypertension		42	53.8
Smoking		45	57.7
History of MI		30	38.5
History of angina		35	44.9
History of PCI		8	10.3
History of CABG		9	11.5
Dyslipidaemia		28	35.9
Renal insufficiency		41	52.6
LVEF% (Mean \pm SD)		34.97 \pm 4.60	
Haemodynamics	SBP(Mean \pm SD)	71.35 \pm 11.19	
	DBP(Mean \pm SD)	44.55 \pm 12.01	
	HR(Mean \pm SD)	110.32 \pm 15.85	
Hospitalization (days)		4.77 \pm 2.82 (<1-14)	

MI:Myocardial Infarction, HT:Hypertension, PCI:Percutaneous coronary Intervention, DBP: Diastolic Blood Pressure, SBP:Systolic Blood Pressure, HR: Heart Rate, CABG: Coronary Artery Bypass Graft

Table 2. The ECG findings of AMI

ECG findings	No	%
NonSTEMI	14	17.9
STEMI	64	82.1
Anterior AMI	44	68.8
Inferior AMI	30	46.9
Posterior AMI	9	14.06
Lateral AMI	23	35.9
Apical AMI	13	20.3
RV Infarction	5	7.8
More than one site affected	37	57.8

AMI: Acute Myocardial Infarction, RV: Right Ventricular, STEMI: S Tlevation Myocardial Infarction,



VSR: Ventricular Septal Rupture, LVF: Left Ventricle Failure, RVF: Right Ventricle Failure, MR: Mitral Regurgitation,

Figure 2. Major shock categories

Table 3. Procedure utilization

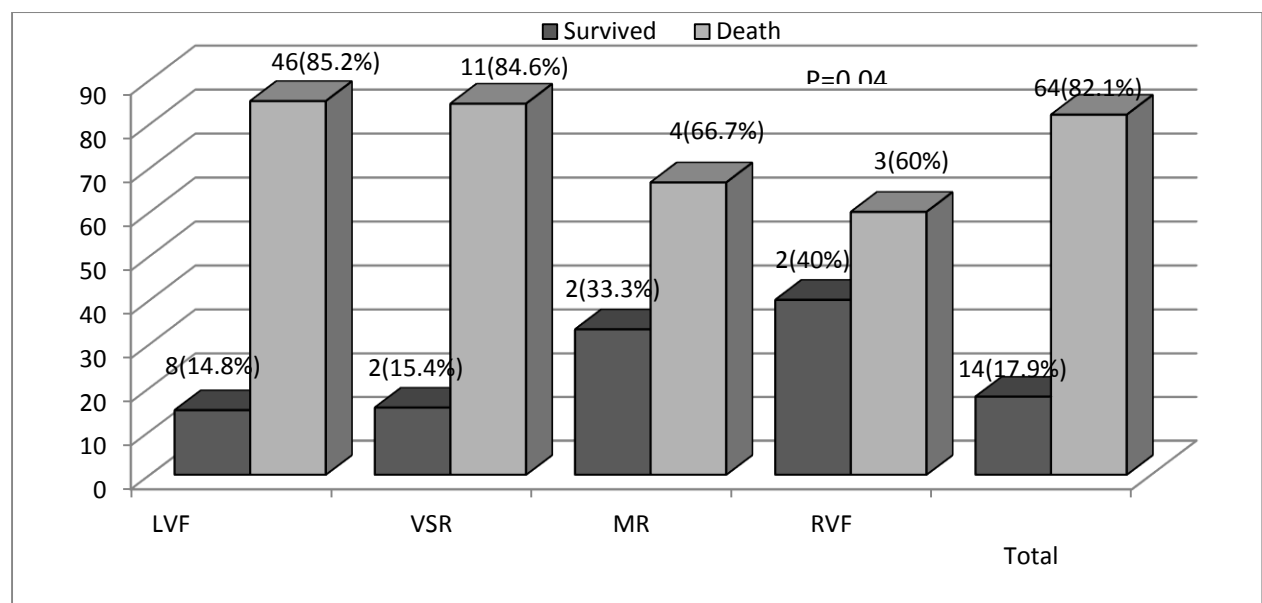
Procedures	No	%
IABP alone	20	25.6
Thrombolytic therapy(TT) alone	11	17.18
Both (IABP+TT)	8	12.5
Inotropics	70	89.7
Coronary angiography	18	23.07
PCI	10	12.8
CABG	3	3.8

IABP: Intraaortic Balloon Pump, PCI: Percutaneous coronary Intervention, CABG: Coronary Artery Bypass Surgery

Table 4. Findings on coronary angiography

Cath. findings		No	%
LMS		3	16.7
Extent of diseased coronary arteries	Single vessel	3	16.7
	Two vessels	6	33.3
	Three vessels	9	50.0
Culprit vessel	LAD	11	61.1
	LCX	2	11.1
	LMS	1	5.6
	RCA	4	22.2

LAD: Left Anterior descending artery, LMS: Left Main Stem vessel, LCx: Left Circumflex, RCA: Right Coronary Artery

**Figure 3.** The mortality of the major shock categories.

VSR: Ventricular Septal rupture, LVF: Left Ventricle Failure, MR: Mitral regurgitation, RVF: Right Ventricle Failure

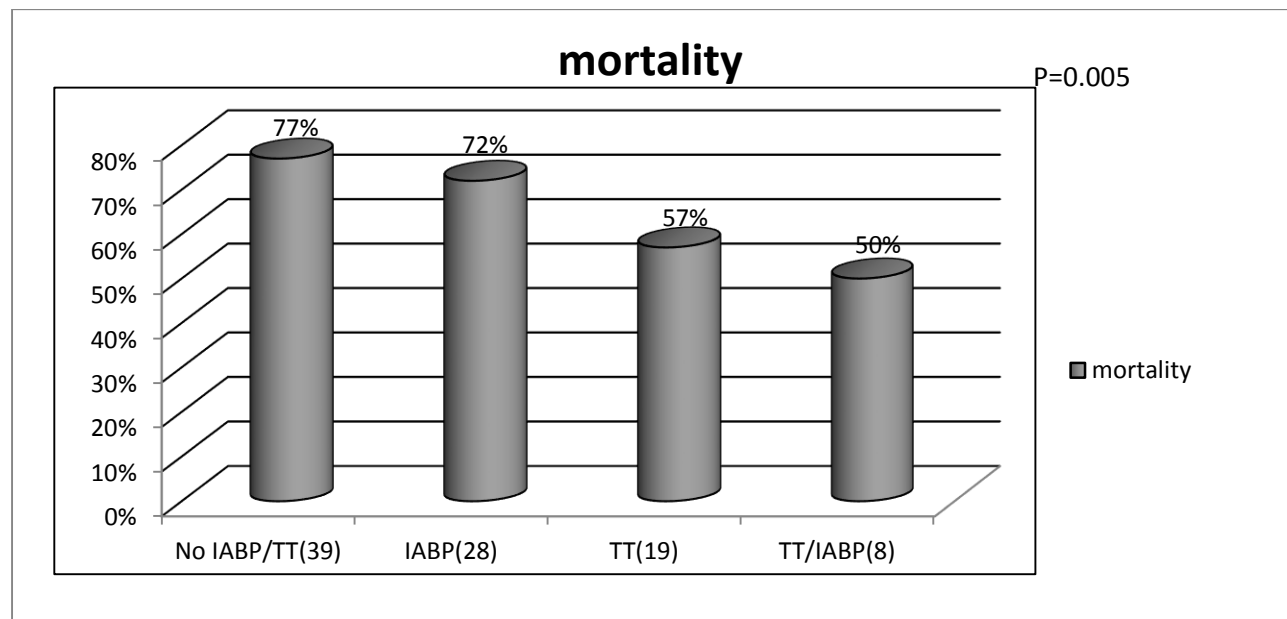


Figure 4. Effect of IABP and TT on outcome.

IABP: Intra-aortic balloon pump ; TT: thrombolytic therapy

Table 5. Predictors of outcome

Procedure	Death		Survived		P value
	No	%	No	%	
Inotropics	59	84.3	11	15.7	0.128
PCI	2	20.0	8	80.0	0.025*
CABG	1	33.3	2	66.7	0.187
EF % ≤ 35	54	69.2	6	7.6	0.002*
Renal insufficiency	37	90.2	4	9.8	0.047*
Direct admission	20	83.3	4	16.7	0.844
Referral admission	44	81.5	10	18.5	
Age ≥ 65	22	88	3	12	0.02*
LMS	2	66.67	1	33.3	0.043*
3 vessels disease	4	44.4	5	55.6	0.890
STEMI	54	84.4	10	15.6	0.253
NonSTEMI	10	74.4	4	28.6	
History of MI	25	83.3	5	16.7	0.047*
History of diabetes mellitus	29	90.6	3	9.3	0.046*
History of hypertension	35	83.3	7	16.7	0.750
History of smoking	39	86.7	6	13.3	0.215

* The Pearson Chi-square statistic is significant at the 0.05 level. IABP: Intraaortic Balloon Pump, PCI: Percutaneous Coronary artery Intervention, CABG: Coronary artery bypass Surgery, MI: Myocardial Infarction, STEMI: ST elevation Myocardial Infarction, LMS: Left Main Stem, EF: Ejection Fraction

Main reasons of cardiogenic shock:- The comparative occurrence of the several reasons of cardiogenic shock have not been formerly described in a distinct big prospective research apart from the SHOCK trial registry. The most frequent

reason of shock in myocardial infarction was predominant LV failure which was diagnosed in 54 (69.2%) of the patients (Fig.-2), most frequently with electrocardiographic results consistent with new total coronary obstruction MI

with anterior position (anterior STEMI) which was present in 44 (68.8%). These findings are consistent with the SHOCK trial registry findings⁽⁵⁰⁾. Though inferior MI happened frequently 30(46.9%), it was accompanied with previous myocardial infarction in greater than two-thirds of cases (76.6%), or was related to a mechanical reason of cardiogenic shock or RV infarction. This support the opinion that inferior myocardial infarction only rarely result in shock because of wide spread left ventricular dysfunction^(38,39).

Mechanical reasons of cardiogenic shock, comprising acute mitral regurgitation and VSR— necessitating prompt diagnosis and repair—constitute 24.4% of cases , While it was 12% in the registry of SHOCK trial and this might be attributed to the low rates of thrombolytics and revascularization procedures used in the current study⁽³⁹⁾.

Mortality:- In the current study the overall mortality of patients with shock due to acute myocardial infarction was 82.1% (Figure-3) which is much higher than that reported in other studies including the SHOCK trial registry (60%)⁽³⁹⁾. Utilization of IABP was 87% in the SHOCK trial and this may have account to the improved prognosis noted in this trial³⁹. The in-hospital fatality of patients entered with STEMI in Ibn AL-Bitar center for cardiac surgery in 2007 was (13.2%) which is higher than that reported in other studies⁴⁰. the main reason for that is that our hospital is a tertiary center, usually receives critical and complicated cases. In a study conducted at Ibn AL-Bitar hospital by Amjad R. Bairametal.at 2007, thrombolytic therapy was underused in patients with AMI (44.1%), and Delay of presentation to the hospital was the leading cause (50%) for not receiving thrombolytic treatment and unavailability of the drug was the second reason.⁴⁰

Before 1990, cardiogenic shock patients had a mortality rate in the hospital reaching 70–85%, but a report from Massachusetts , Worcester has recently

reported a decrease in mortality in the hospital from 80% between years 1975–1990 up to 60% between years 1995–1997⁴¹. The improvement in survival was associated with an increase in the usage of IABP (from 5% - 46%), fibrinolytic therapy (from 0% - 53%), PCI (from 0% - 42%) and CABG (from 0% - 14%). The detected proportion utilizations of IABP in the US increased from 35% in the GUSTO-I to 47% in the GUSTO-III (p =0.001), while the use of these procedures in our study was much lower than these figures (Table-3).

The death rates when ventricular septal reupture was the reason of cardiogenic shock was ominously great (Fig.-3), stressing the necessity for quick septal reparation before cardiogenic shock occure^{30,42}. In our study only three of thirteen patients with VSR underwent surgery, and these patients subjected to surgery after stabilization of their condition.

The mortality rate for patients who were directly admitted to Ibn AL-Bitar centre and those who were transferred from other hospitals was not significantly different (Table-5); However, patients transported to tertiary centers of SHOCK Trial had meaningfully lesser death than patients who were directly admitted to the SHOCK Trial centers³⁹. This can be explained by the delayed transfer of the patients and other road traffic obstacles (Poor infrastructures of the transporting system), or because most of the transferred patients were critically ill , and possibly the low rate of invasive therapeutic intervention performed in our hospital had contributed to the lack of variance in results between the transported and directly admitted patients.

Outcomes with thrombolysis and IABP:- The patients who are given thrombolytic therapy alone or in conjunction with IABP had lower death rates than those patients not getting these treatments (Table-5), and the combined

use of these therapies appeared to be additive⁴³. Investigational studies indicates that, the low rates of clot dissolution are returned when thrombolysis is given with IABP in patients with hypotension^{44,45}. Likewise, non-randomized clinical trials have stated lower death for these combined treatments^{46,47}. Fibrinolytic treatments has not been found to improve results in patients with cardiogenic shock⁴⁸, though it had been found to increase prognosis in cases with hypotension in the Fibrinolytic Therapy Trialists' (FTT) overview⁴⁹.

In this study the usage of IABP alone has not been resulted in a significant decrease in in-hospital fatality and this can be attributed to either the delay in IABP placement or the lack of revascularization procedures and sometimes the patients died before they receive these measures. Lesser randomized studies in the pre-thrombolytic era, yet, did not found that IABP alone improve outcome^{50,51}. IABP alone has not been considerably increase blood stream beyond a critical coronary artery stenosis⁵².

IABP may not be used as a free technique to treat shock. IABP may, still, be a vital supporting technique to need to be urgently carried out. In GUSTO trial, cases who were present with cardiogenic shock and had an quick intraaortic balloon pump arrangement revealed a tendency towards a lesser death rates, even though when excluding cases who have PCI^{53,54}. Same tendency was observed in the registry SHOCK trial⁵⁵. In hospitals with no direct PCI facility, calming with thrombolysis and IABP then referral to a tertiary center could be the optimal treatment choice.

PCI and CABG:- A lot of non-randomized trials had reported that death from cardiogenic shock was less in cases managed with PCI than in medically treated patients⁵⁶. Observational studies propose that an open infarct related artery in cases with cardiogenic shock complicating an acute myocardial

infarction associates powerfully with inhospital survival^(57,58). In a study of 200 cases from Duke, as example, the in hospital fatality was much less in cases with an opened compared to cases with a closed infarct related arteries (33 versus 75 percent); this association was liberated of how patency was accomplished (spontaneous or fibrinolytic therapy or coronary intervention)⁵⁷. Same results were observed in the SHOCK trial registry⁵⁸.

The results of the only completed trial in cases with cardiac shock, the (SHOCK) Trial, had been published in 1999¹⁰⁰. from 1993 to 1998, 302 patients with cardiac shock result from LV dysfunction due to an acute ST-elevation MI, were randomized to either urgent intervention (either surgery or PCI) or initial medical treatment. Early revascularization resulted in 67% improvement in survival compared to initial medical stabilization. Generally the rates of survival were 32.8% and 19.6% in the initial revascularization and early medical stabilization groups, correspondingly. The rates of Survival for survivors of in hospital shock were 62.4% vs 44.4% for the initial revascularization and early medical stabilization groups, correspondingly, with annual death rates of 8.3 vs 14.3 deaths per 100 patient-years⁵⁹.

Despite the fact that many studies emphasize the significance of initial intervention in patients with cardiac shock, in this study only few patients underwent catheter based or surgical revascularization (Table-3). The reason for this is multifactorial. Many cardiologist are reluctant to interfere with these patients who we reappearing to be dying, either because they are elderly and so critically ill and some patients cannot be flat or the patient has other comorbid condition including renal impairment, or sometimes the cath. Lab. Is occupied.

Other predictive factors:- Certain other factors might have adverse effect on the

outcome of cases with cardiac shock in whom the strategy of early intervention may have slight influence on the natural history of the illness (Table-5). It has been found that the result is linked to age, as in the SHOCK study, with cases more than 65 year (75 years in SHOCK trial) doing much worse than younger patients. Though, it was very hard to recognize with sure whether there is no any advantage for the older patients or after any age group⁶⁰. The best treatment of older patients is not clear, and death in SHOCK trial was bigger respective of management. 56 patients more than 75 year of age were included in SHOCK Trial, and those randomized to PCI or CABG had greater death rate at day 30 (75.0% vs 53.1%) and one year (79% vs 66%) than those randomized to medical treatment. Only 8 old patients undertook late revascularization, with a 30-day death rate of 25%. A practical treatment approach for the old patients may include first medical stabilization with careful use of later revascularization. In other patients, mainly those with less comorbidities, emergency revascularization may be the top treatment⁶⁰.

Cases with cardiogenic shock and prior myocardial infarction represents an exceptionally great risk patients. Trials of coronary intervention in this condition should be carefully measured, since it looks likely that the expected sequence of this illness may not be changed. All effort must be undertaken to decrease the occurrence of cardiogenic shock in those patients (decreasing the period to management and use of primary PCI in favorite to thrombolytic therapy)⁶¹.

Other independent factors that are associated with adverse outcome include a very low EF ($\leq 35\%$), presence of renal impairment, presence of LMS disease on cath. finding and a history of DM, a finding that was consistent with other studies^{61,62}.

Conclusions and recommendations

This study shows a high in-hospital mortality for patients with cardiogenic shock complicating acute myocardial infarction and despite the proven benefit of revascularization procedures there were only few patients had undergone these procedures. The utilization rates of thrombolytic therapy and intraaortic balloon pump were also low.

Early recognition of cases of cardiogenic shock complication acute MI, timely used thrombolytic therapy, judicious use of intraaortic balloon and early revascularization procedures were recommended aiming to reduce the high mortality rate in such cases.

References

- 1- Dole WP, O'Rourke RA. Pathophysiology and management of cardiogenic shock. *CurrProblCardiol* 1983;8:1-72.
- 2- Page DL, Caulfield JB, Kastor JA, DeSanctis RW, Sanders CA. Myocardial changes associated with cardiogenic shock. *N Engl J Med* 1971;285:133-137.
- 3- Alonso DR, Scheidt S, Post M, Killip T. Pathophysiology of cardiogenic shock: quantification of myocardial necrosis, clinical, pathologic and electrocardiographic correlations. *Circulation* 1973;48:588-596.
- 4- Gewirtz H, Gold HK, Fallon JT, Pasternak RC, Leinbach RC. Role of right ventricular infarction in cardiogenic shock associated with inferior myocardial infarction. *Br Heart J* 1979;42:719-725.
- 5- Gillespie TA, Ambos HD, Sobel BE, Roberts R. Effects of dobutamine in patients with acute myocardial infarction. *Am J Cardiol* 1977;39:588-594.
- 6- Goldstein RA, Passamani ER, Roberts R. A comparison of digoxin and dobutamine in patients with acute infarction and cardiac failure. *N Engl J Med* 1980;303:846-850.
- 7- Klocke RK, Mager G, Kux A, Hopp H-W, Hilger HH. Effects of a twenty-four-hour milrinone infusion in patients with severe heart failure and cardiogenic shock as a function of the hemodynamic initial condition. *Am Heart J* 1991;121:Suppl:1965-1973.
- 8- Monrad ES, Baim DS, Smith HS, Lanoue AS. Milrinone, dobutamine, and nitroprusside: comparative effects on hemodynamics and myocardial energetics in patients with severe congestive heart failure. *Circulation* 1986;73:Suppl III:III-168.

- 9- Zehender M, Kasper W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. *N Engl J Med* 1993;328:981-988.
- 10- Dell'Italia LJ, Starling MR, Crawford MH, Boros BL, Chaudhuri TK, O'Rourke RA. Right ventricular infarction: identification by hemodynamic measurements before and after volume loading and correlation with noninvasive techniques. *J Am CollCardiol* 1984;4:931-939.
- 11- Cohn JN, Guha NH, Broder MI, Limas CJ. Right ventricular infarction: clinical and hemodynamic features. *Am J Cardiol* 1974;33:209-214.
- 12- Dunkman WB, Leinbach RC, Buckley MJ, et al. Clinical and hemodynamic results of intraaorticballoonpumping and surgery for cardiogenic shock. *Circulation* 1972;46:465-477.
- 13- Scheidt S, Wilner G, Mueller H, et al. Intra-aortic balloon counterpulsation in cardiogenic shock: report of a co-operative clinical trial. *N Engl J Med* 1973;288:979-984.
- 14- Bardet J, Masquet C, Kahn J-C, et al. Clinical and hemodynamic results of intra-aortic balloon counterpulsation and surgery for cardiogenic shock. *Am Heart J* 1977;93:280-288.
- 15- Jackson G, Cullum P, Pastellopoulos A, Macarthur A, Jewitt D. Intra-aortic balloon assistance in cardiogenic shock after myocardial infarction or cardiac surgery. *Br Heart J* 1977;39:598-604.
- 16- Willerson JT, Curry GC, Watson JT, et al. Intraaortic balloon counterpulsation in patients in cardiogenic shock, medically refractory left ventricular failure and/or recurrent ventricular tachycardia. *Am J Med* 1975;58:183-191.
- 17- Johnson SA, Scanlon PJ, Loeb HS, Moran JM, Pifarre R, Gunnar RM. Treatment of cardiogenic shock in myocardial infarction by intraaortic balloon counterpulsation surgery. *Am J Med* 1977;62:687-692.
- 18- Forssell G, Nordlander R, Nyquist O, Schenck-Gustavsson K. Intraaortic balloon pumping in the treatment of cardiogenic shock complicating acute myocardial infarction. *Acta Med Scand* 1979;206:189-192.
- 19- The effects of tissue plasminogen activator, streptokinase, or both on coronary artery patency, ventricular function, and survival after acute myocardial infarction. The GUSTO Angiographic Investigators. *N Engl J Med* 1993; 329:1615-1522
- 20- Keeley EC, Boura JA, Grines CL, et al: Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: A quantitative review of 23 randomized trials. *Lancet* 2003; 361:13-20
- 21- Hochman JS, Sleeper LA, Webb JG, et al: Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med* 1999; 341:625-634
- 22- Hochman JS, Sleeper LA, White HD, et al: One-year survival following early revascularization for cardiogenic shock. *JAMA* 2001; 285:190-192
- 23- Jeger RV, Tseng CH, Hochman JS, et al: Inter-hospital transfer for early revascularization in patients with ST-elevation myocardial infarction complicated by cardiogenic shock—A report from SHOCK trial and registry. *Am Heart J* 2006; 152:686-692
- 24- Eagle KA, Guyton RA, Davidoff R, et al: ACC/ AHA 2004 guideline update for coronary artery bypass graft surgery: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1999 Guidelines for Coronary Artery bypass Graft Surgery). *Circulation* 2004; 100:e340
- 25- Duvernoy CS, Bates ER: Management of cardiogenic shock attributable to acute myocardial infarction in the reperfusion era. *J Intensive Care Med* 2005; 4:188-198
- 26- Trost JC, Hillis LD: Intra-aortic balloon counterpulsation. *Am J Cardiol* 2006; 97: 1391-1398
- 27- Anbe DT, Armstrong PW, Bates ER, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). ACC/AHA Practice Guidelines 2004. <http://www.acc.org/clinical/guidelines/stemi/index.pdf>. Accessed January 2007.
- 28- Mehta SR, Eikelboom JW, Natarajan MK, et al: Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am CollCardiol* 2001; 37:37-43
- 29- Bowers TR, O'Neill WW, Grines C, et al: Effect of reperfusion on biventricular function and survival after right ventricular infarction. *N Engl J Med* 1998; 338:933-940
- 30- Thompson CR, Buller CE, Sleeper LA, et al: Cardiogenic shock due to acute severe mitral regurgitation complicating acute myocardial infarction: A report from the SHOCK Trial Registry. *J Am CollCardiol* 2000; 36:1104-1109
- 31- Menon V, Webb JG, Hillis LD, et al: Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction:

- A report from the SHOCK Trial Registry. *J Am CollCardiol* 2000; 36:1110–1116
- 32- Killen DA, Piehler JM, Borkon AM, et al: Early repair of postinfarction ventricular septal rupture. *Ann ThoracSurg* 1997; 63:138–142
 - 33- Reardon MJ, Carr CL, Diamond A, et al: Ischemic left ventricular free wall rupture: Prediction, diagnosis, and treatment. *Ann ThoracSurg* 1997; 64:1509–1513.
 - 34- The GUSTO Investigators. An International randomized trial comparing four thrombolytic strategies for acute myocardial infarction. *N Engl J Med* 1993;329:673– 82.
 - 35- Holmes DR Jr, Bates ER, Kleiman NS, Sadowski Z, Horgan JH, Morris DC, Califf RM, Berger PB, Topol EJ. 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 CollCardiol* 1995;26:668 –74.
 - 36- Hands ME, Rutherford JD, Muller JE. The in-hospital development of cardiogenic shock after myocardial infarction: incidence, predictors of occurrence, outcome, and prognostic factors. The MILIS Study Group. *J Am CollCardiol* 1989;14:40–6.
 - 37- Killip T, 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.
 - 38- Goldberg RJ, Gore JM, Alpert J, et al. Cardiogenic shock acute myocardial infarction. Incidence and mortality from a communitywide perspective, 1975 to 1988. *N Engl J Med* 1991;325:1117–22.
 - 39- Hochman JS, Sleeper LA, Godfrey E, et al., for the SHOCK Investigators. SHould we emergently revascularize Occluded Coronaries for cardiogenic ShocK: an international randomized trial of emergency PTCA/CABG-Trial Design. *Am Heart J* 1999;137:313–21.
 - 40- Judith S. Hochman, Christopher E. Buller, Lynn A. Sleeper, Jean Boland, Vladimir Dzavik, Timothy A. Sanborn, Emilie Godfrey, Harvey D. White, John Lim, Thierry LeJemtel, for the SHOCK Investigators *J. Am. Coll. Cardiol.* 2000;36;1063-1070
 - 41- Amjad R. Bairam, Ahmed Sabbar, Muthanna Al-Quraishi, Ali Abdul Ameer Mohammed. ST segment elevation myocardial infarction, inhospital and 30 day outcome in a single centre in Baghdad. *The N Iraqi J Med.* 2010 ; 6(1):10-17
 - 42- Goldberg RJ, Samad NA, Yarzebski J, Gurwitz J, Bigelow C, Gore JM. Temporal trends in cardiogenic shock complicating acute myocardial infarction. *N Engl J Med* 1999; 340: 1162–8.
 - 43- Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA Guidelines for the management of patients with acute myocardial infarction. *J Am CollCardiol* 1996;28:1328–428.
 - 44- Sanborn TA, Sleeper LA, Bates ER, et al., for the SHOCK Investigators. Impact of thrombolysis, aortic counterpulsation, and their combination in cardiogenic shock: a report from the SHOCK Trial Registry. *J Am CollCardiol* 2000;36:1123–9.
 - 45- Prewitt RM, Gu S, Schick U, Ducas J. Intraaortic balloon counterpulsation enhances coronary thrombolysis induced by intravenous administration of a thrombolytic agent. *J Am CollCardiol* 1994;23:794–8.
 - 46- Prewitt RM, Gu S, Garger PJ, Ducas J. Marked systematic hypotension depresses coronary thrombolysis induced by intracoronary administration of recombinant tissue-type plasminogen activator. *J Am CollCardiol* 1992;20:1626 –33.
 - 47- Stomel RJ, Rasak M, Bates ER. Treatment strategies for acute myocardial infarction complicated by cardiogenic shock in a community hospital. *Chest* 1994;105:997–1002.
 - 48- Barron HV, Pirzada SR, Lomnitz DJ, Every NR, Gore JM, Chou TM. Use of intra-aortic balloon counterpulsation in patients with acute myocardial infarction complicated by cardiogenic shock (abstr). *J Am CollCardiol* 1998;31Suppl A:135A.
 - 49- Gruppo Italiano per lo Studio della Streptochinasinell' Infar to Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986; i: 397–402.
 - 50- 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 randomised trials of more than 1000 patients. *Lancet* 1994; 343: 311–22.
 - 51- O'Rourke MF, Norris RM, Campbell TJ, Chang VP, Sammel NL. Randomized controlled trial of intraaortic balloon counterpulsation in early myocardial infarction with acute heart failure. *Am J Cardiol.* 1981;47:815-20.
 - 52- Flaherty JT, Becker LC, Weiss JL, Brinker JA, Bulkley BH, Gerstenblith G, et al. Results of a randomized prospective trial of intraaortic balloon counterpulsation and intravenous nitroglycerin in patients with acute myocardial infarction. *J Am CollCardiol.* 1985;6:434-46.
 - 53- Kern MJ, Aguirre F, Bach R, Donohue T, Siegel R, Segal J. Augmentation of coronary blood flow by intra-aortic balloon pumping in patients after coronary angioplasty. *Circulation.* 1993;87:500-11.

- 54- An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. The GUSTO Investigators. *N Engl J Med*. 1993; 329:673-82.
- 55- Anderson RD, Ohman EM, Holmes DR Jr, Col I, Stebbins AL, Bates ER, et al. Use of intraaortic balloon counterpulsation in patients presenting with cardiogenic shock: observations from the GUSTO-I Study. *Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries. J Am Coll Cardiol*. 1997;30:708-15.
- 56- Hochman JS, Boland J, Sleeper LA, Porway M, Brinker J, Col J, et al. Current spectrum of cardiogenic shock and effect of early revascularization on mortality. Results of an International Registry. SHOCK Registry Investigators. *Circulation*. 1995;91:873-81.
- 57- French JK, White HD. Generalization of trial and registry cohort results to individual patients with cardiogenic shock. *ACC Current Journal Review* 1999; 8: 7-11.
- 58- Bengtson, JR, Kaplan, AJ, Pieper, KS, et al. Prognosis of cardiogenic shock after acute myocardial infarction in the interventional era. *J Am Coll Cardiol* 1992; 20:1482.
- 59- Wong, SC, Sanborn, T, Sleeper, LA, et al. Angiographic findings and clinical correlates in patients with cardiogenic shock complicating acute myocardial infarction: a report from the SHOCK Trial Registry. SHould we emergently revascularize Occluded Coronaries for cardiogenic shock. *J Am Coll Cardiol* 2000; 36:1077.
- 60- Hochman JS, Sleeper LA, Webb JG, et al: Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med* 1999; 341:625-634
- 61- Dzavik V, Sleeper LA, Cocke TP, et al. Early revascularization is associated with improved survival in elderly patients with acute myocardial infarction complicated by cardiogenic shock: a report from the SHOCK trial registry. *Eur Heart J* 2003;24:828-37.
- 62- Hands ME, Rutherford JD, Muller JE *et al*. 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.