

ORIGINAL ARTICLE

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Cardiogenic shock as a first presentation in acute myocardial infarction in Egyptian's patients and its outcome

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Abstract

Cardiogenic shock secondary to acute myocardial infarction continues to be a frustrating problem of great clinical significance. With improvements in the medical management of AMI, particularly the treatment of ventricular arrhythmias, CS has emerged as the most common cause of death (7 to 12%) of all patients admitted with myocardial infarction. Was to minimise the impact of acute MI and/or its complications especially cardiogenic shock by early detection and rapid diagnosis to improve management processes. The study was descriptive, comprehensive study that included 60 patients presented with acute myocardial infarction with or without cardiogenic shock. Firstly assessed clinical, Electrocardiography and cardiac enzymes with treatment accordingly to Advanced Cardiac Life Support (ACLS) guidelines and treat the life threatening conditions if present with follow-up throughout their hospital stays for Major Adverse Cardiac Events (MACE) that included death, reinfarction. The incidence of cardiogenic shock as the presenting problem in acute MI was 13.3%. The Patients with cardiogenic shock stayed more in the hospital with significantly more incidence of reinfarction and in-hospital mortality. Cases with CS were older in age, had a higher incidence of diabetes mellitus, angina, previous MI and heart failure, a larger size of infarction, as reflected in increased level of cardiac enzymes, and was found in patients with CS.

Keywords: Acute myocardial infarction, cardiogenic shock, heart

Introduction

Cardiogenic shock (CS) is a state of inadequate tissue perfusion due to cardiac dysfunction, usually acute myocardial infarction (AMI) [1].

It occurs in $\approx$ 5% to 8% of patients hospitalized with ST-elevation myocardial infarction (STEMI). The definition of CS includes hemodynamic parameters: persistent hypotension (systolic blood pressure $<$ 80 to 90 mmHg or mean arterial pressure 30mmHg lower than baseline) with severe reduction in cardiac index ($<$ 1.8l/min/m² without support or $<$ 2.0 to 2.2L/min/m² with support) and adequate or elevated filling pressure (e.g., left ventricular end-diastolic pressure $>$ 18mmHg or right ventricular end-diastolic pressure $>$ 10 to 15 mmHg) [2].

Hypoperfusion may be manifest clinically by cool extremities, decreased urine output, and/or alteration in mental status. Hemodynamic abnormalities form a spectrum that ranges from mild hypoperfusion to profound shock, and the short-term outcome is directly related to the severity of hemodynamic derangement [1].

MI with LV failure remains the most common cause of CS. It is critical to exclude complicating factors that may cause shock in MI patients. Chief among these are the mechanical complications: ventricular septal rupture, contained free wall rupture, and papillary muscle rupture. Mechanical complications must be strongly suspected in patients with CS complicating non-anterior MI, particularly a first MI. Echocardiography is the technique of choice to rule out these entities and should be performed early unless the diagnosis is extensive anterior MI and the patient is undergoing prompt percutaneous coronary intervention (PCI). In addition, the detection of valvular disease before angiography may alter the revascularization approach [2].

Any causes of acute, severe LV or RV dysfunction may lead to CS. Acute myopericarditis, takotsubo cardiomyopathy, and hypertrophic cardiomyopathy may all present with ST elevation, release of cardiac markers, and shock in the absence of significant coronary artery disease. Stress-induced cardiomyopathy, also known as apical ballooning or takotsubo cardiomyopathy, is a syndrome of acute LV dysfunction after emotional or respiratory distress that leads to CS in 4.2% of cases [3].

Acute valvular regurgitation, typically caused by endocarditis or chordal rupture due to trauma or degenerative disease, may also cause CS. Aortic dissection may lead to CS via acute, severe aortic

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insufficiency or MI. Acute stress in the setting of aortic or mitral stenosis can also cause shock. Cardiac tamponade or massive pulmonary embolism can present as cardiogenic shock without associated pulmonary congestion [3].

Identification of patients at risk

The only way to prevent CS appears to be very early reperfusion therapy for MI. A randomized trial of early, in-ambulance thrombolysis versus primary PCI found no CS among patients assigned to prehospital thrombolysis [4].

Among PCI-assigned patients, just 0.5% developed CS in the group randomized < 2 hours from symptom onset. A major focus of public health campaigns is the very early recognition and reperfusion of MI, which should reduce CS incidence. Risk factors for development of CS in the context of MI include older age, anterior MI, hypertension, diabetes mellitus, multivessel coronary artery disease, prior MI or angina, prior diagnosis of heart failure, STEMI, and left bundle-branch block. There may be clues to impending shock: heart rate is higher and blood pressure lower on hospital presentation among patients who develop CS after admission [5].

The aim of work was to minimize the Impact of acute MI and/or its complications especially cardiogenic shock by early detection and rapid diagnosis to improve management processes in the Suez Canal University Hospital (SCUH) within four months duration of the study.

Material and Methods

Study Design and Site

This was a descriptive, comprehensive study that included all patients presented with acute myocardial infarction with or without cardiogenic shock.

Study site

The study was conducted at the emergency and CCU departments, SCUH.

Study objectives

1. To study the incidence of cardiogenic shock as the presenting problem in acute MI patients
2. To outline different factors that may induce cardiogenic shock as an early presentation
3. To highlight the impact of different lines of management of acute MI on the incidence of cardiogenic shock.

Study period: 4 months duration

Study population: All patients presented to the emergency, SCUH with acute myocardial infarction with or without cardiogenic shock throughout the period of four months.

Inclusion criteria

- Patients presented with acute myocardial infarction with or without cardiogenic shock.
- Both sexes and all ages.

Exclusion criteria

- Patients with end stage valvular or myocardial disease.
- Patients presented with other types of shock.

All the patients were subjected to the followin

1- History taking: thorough history was taken from each patient with special highlight on the following:

- Chest pain analysis as regard nature, radiation and duration, aggravating and relieving factors and or drugs. The presence of one or more of the risk factors for ischemic heart diseases, such as smoking, hypertension, diabetes mellitus, hyperlipidemia and a positive family history for ischemic heart disease.
- The use of thrombolytic therapy.

2- Complete physical examination was performed to rule out any other medical problems

- General examination: e.g., general condition, pulse, blood pressure, respiratory rate, temperature, neck veins, edema of both lower limbs, chest auscultation for evidence of pulmonary venous congestion.
- Local examination: auscultation of heart sounds, additional sounds and murmurs.

3- Diagnosis of cardiogenic shock was based on the presence of severe systemic hypotension, with cool extremities and oliguria (urine output <400 ml /24 hours) and respiratory distress caused by pulmonary congestion as a complication of acute MI [6].

4- For the Diagnosis of acute MI (STEMI or NSTEMI), 2 out of the following criteria must be present:

- Typical angina chest pain of 30 minutes duration.
- A standard 12-Lead ECG with paper speed of 25 mm/s, 10mm/mv was assessed for the presence of ST segment elevation or depression in chest or limb leads.
- Measurement of myocardial markers:

Serial increase in plasma CK-MB level (more than 25% increase between two measurements) (normal values 0-25 mg/dl). CK-MB to total CK levels is more than 5%. Total CPK level (normal: 24-171 mg/dl)

Within first 12 hours, Troponin I values exceeded 0.1 ng/ml. (normal: 0-0.10 ng/ml)

Troponin I Assay: The assay was done using IMMULITE® / IMMULITE® 1000 Troponin I Kits, Catalog No: LKTI1 (100 tests).

5- Echocardiographic study

All patients underwent transthoracic Echocardiographic examination using HP machine, Probe 2.5 MHz for the assessment of the left ventricular ejection fraction using Simpson's rule.

6- Follow up

All patients were followed up throughout their hospital stay for Major Adverse Cardiac Events (MACE) that included death, reinfarction.

Data collection: Data were collected by the researcher himself and well-trained medical personnel.

Statistical analysis

The data were collected, organized and tabulated using SPSS (statistical package for social science) computer software version 16.0, on an IBM compatible computer.

For quantitative data, mean and standard deviation were calculated, for comparison between two means, students (t) test was used, for comparison between the values of a parameter at two different intervals, paired (t) test was used, for categorical data, chi2 or Fisher's exact test were calculated, for interpretation of results, p value was considered significant when it equal or less than 0.05.

Ethical consideration: All Patients give consent to participate in the study without affecting their course of treatment accordingly permission obtained from ethical committee of faculty of medicine in Suez Canal University

- 1) Approval of Research ethics committee.
- 2) Singing written informed consent from participants.
- 3) Confidentiality of data.
- 4) Explanation of our study to the participants.

- 5) An informed consent was taken from each patient or relatives.

Results

The present study included 60 cases presented with acute myocardial infarction; 8 of them had cardiogenic shock. Thus, the incidence of cardiogenic shock in acute MI was 13.3%.

The studied population was divided into 2 subgroups:

*Patients with acute MI and cardiogenic shock that included 8 patients.

* Patients with acute MI without cardiogenic shock that included 52 patients.

Both subgroups were compared.

Age: in the studied group, age ranged from 48 to 83 years with a mean of 62.16 ± 7.61 years. Patients with MI + cardiogenic shock were significantly older as shown in Table (1).

Table 1. Age distribution in the studied population

Cardiogenic shock	Mean age in years	S. D	Minimum Age	Maximum age	P value
+ve n (8)	70.25	12.66	48.00	83.00	
-ve n (52)	60.92	5.76	49.00	71.00	0.001(S)

Gender: As a whole, male gender was dominant in the studied population (40 patients) as compared to female gender (20 females)

$p < 0.05$. However, gender had no significant impact on the incidence of cardiogenic shock. These data are shown in Table (2).

Table 2. Gender distribution in the studied population

	Cardiogenic shock		Acute MI only			
	n.	%	n.	%	n.	%
Male	6	75.0%	34	65.4%	40	66.7%*
Female	2	25.0%	18	34.6%	20	33.3%
Male: female ration		3:1		1.9: 1		2: 1
						0.010(S)
						0.59(NS)

*= statistically significant increase in total number of males in comparison to total number of females.

Risk stratification: The presence of angina pectoris, previous MI, diabetes mellitus and heart failure was significantly higher among patients presenting with cardiogenic shock as shown in Table (3).

thrombolytic therapy 7 in the cardiogenic subgroup (88%) and 27 (52%) in the acute MI subgroup $p < 0.05$. these data are shown in table (5).

Hemodynamics: As regards hemodynamic parameters, there was a significantly higher heart rate and lower systemic blood pressure among patients with cardiogenic shock as shown in table (4).

*10 patients had absolute contraindications.

*15 patients passed the widow time of thrombolysis, 9 of them were referred for PCI few days later.

*5 patients had NSTEMI.

*4 patients had LBBB which was not confirmed to be recent.

Thrombolytic Therapy: There was 34 Patients who didn't receive

Table 3. Risk stratification of patients in the studied groups

	Cardiogenic Shock		Acute MI Only		P value
	n.	%	n.	%	
Hypertension	3	37.5%	11	21.2%	0.33(NS)
Angina	6	75.0%	17	32.7%	0.022(S*)
Previous MI	4	50.0%	8	15.4%	0.023(S*)
Diabetes mellitus	3	37.5%	5	9.6%	0.031(S*)
Heart failure	4	50.0%	7	13.5%	0.013(S*)

Table 4. Comparison between groups as regard hemodynamic parameter

	CS	Mean	S. D	Minimum	Maximum	P value
HR (b/min)	Yes	118.12	14.74	84.00	130.00	<0.001(S)
	No	78.98	3.63	70.00	86.00	
	Total	79.80	4.22	70.00	90.00	
SBP (mmHg)	Yes	87.50	2.67	85.00	90.00	0.018(S)
	No	95.86	9.63	80.00	120.00	
	Total	94.75	9.45	80.00	120.00	
DBP (mmHg)	Yes	49.37	4.17	45.00	55.00	<0.001(S)
	No	59.13	6.91	50.00	80.00	
	Total	57.83	7.38	45.00	80.00	

Table 5. Percent administration of thrombolytic therapy in the studied population

	Cardiogenic shock		No cardiogenic shock		Total	
	n.	%	n.	%	n.	%
Thrombolysis	1	12.5%	25	48.1%	26	43.3%
No	7	87.5%	27	51.9%	34	56.7%
P value	0.044(S)					

ECG findings: Based on the ECG findings, STEMI was reported in 51 cases (85%), 25 of them (41.6%) were anterior and 26 of them (43.3%) were inferior, and left bundle branch block was found in 4 cases (6.7%). NSTEMI was reported in 5 cases (8.3%). The location or the type of MI did not show any significant difference

in those with and those without cardiogenic shock. As shown in table (6). Cardiac biomarkers: As regard cardiac enzymes, there was statistically significant increase of all cardiac enzymes in cases with CS in comparison to cases without CS. The details are shown in table (7).

Table 6. ECG findings in the studied population

	Cardiogenic Shock n=8		Acute MI Only n=52		Total n=60		P value
	n.	%	n.	%	n.	%	
Anterior STEMI	3	37.5%	22	42.3%	25	41.6%	0.79(NS)
Inferior STEMI	3	37.5%	23	44.2%	26	43.3%	0.72(NS)
Left bundle branch block	2	25.0%	2	3.8%	4	6.7%	0.026(S)
NSTEMI	0	0.0%	5	9.6%	5	8.3%	0.36(NS)

Table 7. Cardiac enzymes in the studied population

	CS	Mean	S. D	Minimum	Maximum	P value
CKMB	Yes	122.88	25.473	80.00	150.00	< 0.001 (S*)
	No	90.46	12.03	80.00	140.00	
CK	Yes	1167.1	220.61	882.00	1500.00	< 0.001 (S*)
	No	818.65	103.46	720.00	1200.00	
CTI	Yes	0.640	0.149	0.48	0.89	< 0.001 (S*)
	No	0.416	0.039	0.30	0.52	

CKMB = creatine kinas-MB fraction CK = creatine kinas. CTI= cardiac Troponin I

Echocardiographic findings: As regards echocardiographic parameters, patients with cardiogenic shock had a significantly lower EF as compared to those presenting with MI without cardiogenic shock (25.37 ± 1.99 vs. 37.73 ± 4.76 respectively). These data are shown in table (8). In hospital follow up: all patients were followed up during their hospital stay that ranged from 5 to 13 days with a mean of 8.43 ± 1.9 . A statistically significant increase in duration of hospital stay in cases with CS in comparison to those without CS

(11.25 ± 1.48 vs. 8.00 ± 1.62 days respectively $p=0.001$). These data are shown in table (9). Reinfarction: The incidence of in-hospital reinfarction was significantly higher in the group with CS (37.5% vs. 9.6% $p<0.05$). these data are shown in table (10).

In hospital mortality: As regard in-hospital mortality, 50% of patients with CS died as compared to 5.8% in the other group $p=0.001$. This is shown in table (11).

Table 8. Comparison between groups as regard echocardiographic findings.

Ejection fraction % (EF %)		Mean	S. D	Minimum	Maximum	P value
	CS	25.37	1.99	24.00	30.00	<0.001(S)
	No	37.73	4.76	25.00	48.00	

Table 9. Duration of hospital stay in both subgroups

	Mean	S. D	Minimum	Maximum	P value
Cardiogenic shock	11.25	1.48	9.00	13.00	<0.001(S)
No cardiogenic shock	8.00	1.62	5.00	11.00	
Total	8.43	1.94	5.00	13.00	

During their stay, 15 patients had MACE (25%), 7 patients in the cardiogenic group (88%) and 8 patients in the acute MI only group (15%) $p < 0.0$

Table 10. The incidence of in-hospital reinfarction

Reinfarction	Cardiogenic Shock (8)		Acute MI Only (52)		Total (60)		P value
	n.	%	n.	%	n.	%	
Yes	3	37.5%	5	9.6%	8	13.3%	0.031(S)
No	5	62.5%	52	90.4%	52	86.6%	

Table 11. The incidence of in hospital mortality in both groupss

	Cardiogenic shock		No cardiogenic shock		Total	
	n.	%	n.	%	n.	%
Died	4	50.0%	3	5.8%	7	11.7%
Alive	4	50.0%	49	94.2%	53	88.3%
P value	0.001(S)					

Discussion

Cardiovascular diseases (CVD) are the leading cause of mortality in both men and women worldwide. According to a WHO report, about 55% of deaths in European women are related to CVD, including 23% caused by coronary artery disease (CAD) and 18% due to stroke. Conversely, in men, CVD is the cause of 43% of deaths, including CAD (21%) and stroke (11%) [7].

Acute myocardial infarction can be defined from a number of different perspectives related to clinical, electrocardiographic (ECG), biochemical, and pathological characteristics [8].

Acute myocardial infarction (AMI) is the most serious disease of the cardiovascular system, and cardiogenic shock (CS) is one of the most dangerous complications of it. Cardiogenic shock occurs in approximately 7-10% of patients with AMI and formerly led to 70-80% in-hospital death [9].

CS causes cardiac mechanical inability to maintain adequate output. Patients with CS demonstrate clinical signs of low cardiac output and adequate intravascular volume [10].

The current guidelines pertain to patients presenting with ischemic symptoms and persistent ST-segment elevation on the ECG (STEMI). The great majority of these patients will show a typical rise of biomarkers of myocardial necrosis and progress to Q-wave myocardial infarction [11].

Separate guidelines have been developed by another Task Force of

the ESC (European Society of Cardiology) for patients presenting with ischemic symptoms but without persistent ST-segment elevation [12].

While there are numerous clinical complications that are associated with the development of acute myocardial infarction (AMI), none are more potentially devastating or carry a worse prognosis than cardiogenic shock [7].

Despite marked advances in medical treatment, revascularization techniques, and mechanical support during the past 2 decades, cardiogenic shock is still the most common cause of hospital mortality associated with AMI [8].

On the other hand, data obtained from a limited number of recent studies suggest possible decline in the hospital mortality associated with cardiogenic shock [13], partially associated with the implementation of early revascularization therapy based on recent practice guidelines [11].

The present study was designed to determine the incidence of cardiogenic shock as the presenting problem in acute MI patients and to outline different factors that may induce cardiogenic shock as an early presentation.

The present study included 60 patients presented with acute MI with or without cardiogenic shock; they were recruited from the emergency and CCU departments, SCUH.

All the studied patients were subjected to full history taking,

complete physical examination, laboratory investigations especially cardiac enzymes and finally ECG echocardiographic assessment. Then, all patients were followed up throughout their hospital stay for MACE.

In the present study, the incidence of cardiogenic shock in acute MI was 13.3%. Goldberg et al. [14] reported that, the incidence rates of cardiogenic shock after AMI have ranged from 5 to 15 percent in previously published studies. They reported that, in a study sample of 13,663 residents hospitalized with AMI 6.6% (n=905) developed cardiogenic shock. In addition, Lindholm et al. [5] reported that, cardiogenic shock was present in 444 patients, corresponding to an incidence of 6.7%.

The Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-1) trial reported an incidence of (7.2%), and another published population based study reported an incidence of (7.1%) [8,9].

This relatively wide range reflects the varying definitions of AMI and cardiogenic shock utilized, time periods under study, and use of therapeutic options that may reduce the risk of cardiogenic shock.

The overall incidence rates of cardiogenic shock observed in the current study fall within this range.

In the present study, increased age is one of the factors favoring the development of CS.

These results are in agreement with that reported by Lindholm [5] who reported that, the mean age of cases with CS was 74 compared to 69 in cases with acute MI only.

In the present study, Male gender predominates in the sample presented with AMI (66.7%); on the other hand, this gender difference did not show any significance on the incidence of CS. These findings agree with the results of Lindholm et al. [5] who reported that males represent 67% of all cases with acute MI and represent 68% of cases with CS compared to 60% of cases without CS. This difference was statistically significant. Also, Goldberg et al. [14] reported that 58.5% of their sample presented with AMI were males. As regard risk factor distribution for IHD, patients with diabetes and those with angina, previous MI or heart failure had a higher incidence of CS.

This finding is supported with the study of Lindholm et al. [5] who reported that, among the classical risk factors in relation to ischemic heart disease, diabetes, angina and previous myocardial infarction were more common in the group of patients with cardiogenic shock. In addition, a history of heart failure was twice as prevalent among the shock patients. Diabetics also are twice as likely to develop cardiogenic shock as non-diabetics with AMI [8].

Furthermore, as regard in-hospital mortality, 50% of patients with CS died as compared to 5.8% in the other group (p=0.001).

In the present study, heart rate ranged from 84 to 130 with a mean of 118.12 ± 14.74 b/min with statistically significant increase in HR in cases with cardiogenic shock in comparison to those without CS (118.12 ± 14.74 vs 78.98 ± 3.63 respectively). In addition, there was statistically significant decrease in both systolic and diastolic

blood pressure in cases with cardiogenic shock in comparison to those without CS (87.50 ± 2.67 , 49.37 ± 4.17 vs 95.86 ± 9.63 , 59.13 ± 6.91 respectively).

Goldberg et al. [14] reported that, patients with cardiogenic shock had significantly lower blood pressure at the time of hospital admission, but higher heart rates and serum glucose levels, than patients who did not develop shock.

These results are in agreement with those of the present study. Similar results were reported by Hameed et al. [15], but the difference was statistically insignificant. Furthermore, Nicholls et al. [16] reported that, there was statistically significant decrease in systolic, diastolic and mean arterial pressure and increase in HR in patients with CS in comparison to cases who had acute MI without CS. These results are in agreement with that of the present work.

In the present study, thrombolytic therapy was practiced in 26 cases (43.3%) and there was statistically significant increase in thrombolysis in non-cardiogenic shock cases in comparison to cardiogenic shock cases (48.1% vs. 12.5%). These results are in agreement with Lindholm et al. [5] who reported that, thrombolytic therapy were used less in the shock population as compared to the non-shock patients, reflecting that patients not being revascularized are at higher risk of developing cardiogenic shock.

In the present study, anterior Q-wave MI was reported in 25 cases (41.6%), inferior Q-wave MI reported in 26 cases (43.3%), ST segment elevation reported in 51 cases (85%), NSTEMI reported in 5 cases (8.3%) and bundle branch block reported in 4 cases (6.7%) and there was statistically significant increase in cases with bundle branch block in cardiogenic shock cases in comparison to cases with acute MI without CS (25% vs. 3.8% respectively).

These results are partially goes in agreement with Lindholm et al. [5] who reported that, anterior Q-wave myocardial infarction and bundle branch block were more common in the shock population, whereas the presence of inferior myocardial infarction and ST-segment elevation did not differ between the groups.

It also reported that, infarctions were located anteriorly in most of the patients (55%) in the SHOCK trial registry. While 46% of the infarctions were inferior, 21% were posterior, and 50% were in multiple locations [1]. Other studies have found similar results. Around 60% of patients had triple vessel disease while left main disease was encountered in 20% [17]. The left anterior descending artery (LAD) was found to be the most frequently involved artery unrelated to the time of shock onset. Thus, severe arterial disease precedes shock development [18].

As regard cardiac enzymes there was statistically significant increase of all cardiac enzymes in cases with CS in comparison to cases without CS indicating increased myocytes destruction and larger size of infarction in patients with CS.

In the present study, ejection fraction percent ranged from 24 to 48 with a mean of 36.08 ± 6.17 and there was statistically significant decrease in cases with CS in comparison to those without CS (25.37 ± 1.99 vs. 37.73 ± 4.76 respectively).

These results are in agreement with Picard et al. [19] who demonstrated reduced LV systolic function and mitral regurgitation

in CS. In addition, Nicholls et al. [16] reported that, there was statistically significant decrease in left ventricular ejection fraction in cardiogenic shock patients in comparison to cases with acute MI without CS (25% vs. 60% respectively).

We reported a longer hospital stay in patients with CS. These results are in agreement with those reported by Hameed et al.[15] who reported that, the mean duration of hospitalization for the MI and CS patients were 10 ± 3 and 12 ± 4 days respectively with statistically significant difference between groups

As regards the in hospital follow up, revealed an increased incidence of reinfarction and death. These results are in agreement with Hochman et al. [1] who reported that, cardiogenic shock is the leading cause of death in patients with acute myocardial infarction with an in-hospital mortality rate greater than 50%.

Goldberg et al. [14] reported that, overall, 65.4% of patients with AMI who developed cardiogenic shock died during hospitalization in comparison to 10.6% of patients who did not develop cardiogenic shock ($p < 0.001$). These results are supported by those reported in the present work.

In another study, the overall in-hospital mortality was high (63%) but was found to reduce ($P = 0.004$) over time from 1992 to 1997. This was partially attributed to the greater use of revascularization procedures, which are known to improve outcome [9].

Summary

Despite more aggressive therapeutic interventions in the last decade, the incidence of CS after AMI has remained relatively stable, averaging 7%.

Furthermore, mortality for CS has not decreased significantly, despite intensive medical management including inotropes, thrombolysis, and intraaortic balloon pump (IABP) support, and revascularization, mortality from CS remains in excess of 50%. Several mechanisms can lead to the development of cardiogenic shock after AMI.

The relative incidence of the various causes of CS includes the following:

- Predominant left ventricular (LV) failure (78%).
- Acute mitral valve regurgitation (7%).
- Ventricular septal rupture (4%).
- Acute tamponade/free-wall ruptures (1.5%).
- Isolated right ventricular shock (3%).
- Other causes (6.5%).

This latter group includes cardiac-related diagnoses such as dilated cardiomyopathy, medication excess, and cardiac catheterization complications. It is important to emphasize that the mechanical complications secondary to myocardial rupture (free wall, septum, or papillary muscle rupture) account for the minority of the cases. In the majority of the patients, the cause of death is low cardiac output secondary to predominantly LV failure, which in turn leads to organ failure, arrhythmias, and irreversible shock.

The present study is a descriptive, comprehensive study designed to determine the incidence of cardiogenic shock as the presenting problem in acute MI patients and to outline different factors that

may induce cardiogenic shock as an early presentation

The present study included 60 patients presented with acute MI with or without cardiogenic shock; they were selected from the emergency and CCU departments. SCUH.

Both sexes and all ages were included. Patients with end stage valvular or myocardial disease and other types of shock were excluded. All the studied patients were subjected to full history taking, complete physical examination, laboratory investigations especially cardiac enzymes, ECG and finally echocardiographic assessment.

Then, all patients were followed up for Major Adverse Cardiac Events (MACE) till their discharge or death. Finally statistical analysis of the collected data was done.

As regards results of the study we found that

* There was a significantly higher heart rate and lower systemic blood pressure among patients with cardiogenic shock.

* Cardiogenic shock predominates in patients who didn't receive thrombolytic therapy.

* The location or the type of MI did not show any significant difference in those with and those without cardiogenic shock.

* There was statistically significant increase of all cardiac enzymes in cases with cardiogenic shock in comparison to cases without cardiogenic shock.

* Patients with cardiogenic shock had a significantly lower EF as compared to those presenting with MI without cardiogenic shock.

Limitations of the study

Although we used a larger sample size than that used in Elbaih et al. [20] the sample size was still small and the study could not be blinded which might have introduced some bias into the results. Additionally, the accuracy CS in chest pain could not be precisely detected from history and diagnosis which was based on clinical manifestations.

In cases of CS patients there were contraindication to transport these patients to done more investigations e.g. primary PCI unless to finished resuscitations especially if primary PCI not available on 24 hours so little studied were done for CS in ACS so this considered some limitations.

Some patients refused to be enrolled in this study, Some ER physicians were aware of this study, this study was conducted in one center (Emergency Department of Suez Canal University Hospital and some ACS patients shifted between more than one doctors in the ER department.

Conclusions

The results of the present study revealed that:

1. The Incidence of CS among patients with acute MI was 13.3%
2. Cases with CS were older in age, had higher incidence of diabetes mellitus, angina, previous MI and heart failure

3. Larger size of infarction, as reflected in increased level of cardiac enzymes, was found in patients with CS.
4. Patients with CS stayed longer in the hospital with a higher incidence of in hospital complications and death.

Recommendations

1. In order to improve management process in CS patients, we recommend:
2. Larger multicenter study for the incidence of CS and the predisposing factors all over Egypt.
3. Prospective studies that test the effect of different treatment modalities on the incidence and the fate of CS.
4. To increase health education about risk factors of ischemic heart diseases (especially; modifiable risk factors).
5. To conduct health programs that increase knowledge about early manifestations of coronary diseases and the appropriate first aid.
6. To supply more facilities to hospitals to increase their ability to manage acute and massive complications of coronary diseases.
7. Primary PCI in SCUH should be Available in 24 hours on day.

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