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RESEARCH ARTICLE

INCIDENCE OF CARDIOGENIC SHOCK IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION – A PROSPECTIVE OBSERVATIONAL STUDY AT A TERTIARY CARE HOSPITAL

Tadwalkar Vivek D., *Sachdeva Abhishek, Singh Ratinder P. and Chugh Rajeev

Department of Cardiology, Max Super Speciality Hospital, Patparganj, New Delhi-110092, India

ARTICLE INFO	ABSTRACT Objective: To study the incidence of Cardiogenic Shock (CS) in patients with acute myocardial infarction (AMI) Methods: This is single centre prospective analytical observational study. The study was conducted		
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27 th July, 2018 Accepted 10 th August 2018	018 during 21St August 2010 to 20 th August 2012 in a tertiary care hospital. This study inclu-		
Published online 30 th September, 2018	Results: This analysis pertains to the 25 (10%) patients who unequivocally developed shock after admission. Out of 25 natients who developed shock 13 expired (52% mortality)		
Key Words.	 60-65 is the predominant age group to get shock. 		
Cardiogenic Shock, Incidence, Myocardial Infarction, Killip Class.	 64% of patients were males who developed shock. 40% and 60% of patients who developed shock had DM and HTN respectively. 40% of patients who developed shock had history of previous myocardial infarction. 64% patients who developed shock had smoking as addiction. Around 68% patients who developed shock were having Anterior wall MI. 7.76%, 25.6% and 25% belonging to Killip Class I, II and III respectively developed shock. 48% patients who developed shock had h/0 angina compared to 35.55% in the non-shock group. 		
	Conclusion: The incidence of CS was around 10% in ST-segment elevation myocardial infarction (STEMI) in our study. The strongest predictors for development of CS in our study were age, gender, diabetes mellitus, hypertension, previous myocardial infarction, smoking, anterior infarct, higher Killip class, and history of angina. The mortality among patients with CS in our study was 52%.		

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INTRODUCTION

Cardiogenic shock (CS) is the most common cause of death in patients hospitalized with Acute Myocardial Infarction (AMI) and is associated with a poor prognosis. The precise incidence is difficult to measure because patients who die before reaching the hospital are not given the diagnosis. In contrast, early and aggressive monitoring can increase the apparent incidence of CS. However, several trials show that CS complicates approximately 5% to 8% of ST-elevation myocardial infarction (STEMI) (Fox *et al.*, 2007) and 2.5% of non-ST-elevation myocardial infarction (non-STEMI) cases (Hasdai *et al.*, 2000). The prognosis of CS is extremely poor. Mortality rates were reported at 50% to 80% in older series. Inhospital mortality in the Should We Emergently Revascularize

Occluded Coronaries for Cardiogenic Shock (SHOCK) Trial Registry (Hochman *et al.*, 2000) was 60%. As a result of the high mortality of CS, identification of subgroups of patients with acute ischemic syndromes who are at high risk of developing shock is important.

METHODS

The current work represents a hospital based prospective study which was conducted during 21^{st} August 2010 to 20^{th} August 2012 in a tertiary care hospital. About 275 patients with confirmed ST Elevated Myocardial Infarction (STEMI) were included in this study. Of the 275 patients enrolled in the study, 4 were excluded because they developed shock prior to enrollment, and 21 were excluded because they either had severe hemodynamic compromise after enrollment or did not meet our strict criteria for shock. All of the patients included

^{*}Corresponding author: Sachdeva Abhishek,

Department of Cardiology, Max Super Speciality Hospital, Patparganj, New Delhi-110092, India

were primary thrombolysed. Patients in which Primary Percutaneous Coronary Intervention (p-PCI) was used as treatment and those with onset of symptoms >6 hours were excluded.

Criteria for Cardiogenic Shock: The clinical definition of CS is decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume. Hemodynamic criteria are sustained Hypotension (systolic blood pressure < 90 mm Hg for at least 30 minutes) and a reduced cardiac index (CI<2.2 L/min/m2) in the presence of elevated pulmonary artery occlusion pressure (PAOP > 15 mm Hg) (Hollenberg *et al.*, 1999; Ginsberg and Parrillo, 2009). Circulatory shock was diagnosed at the bedside by observing hypotension and clinical signs indicating poor tissue perfusion, including oliguria; clouded sensorium; and cool, mottled extremities.

Criteria for Myocardial Infarction (Joint European Society of Cardiology, 2000): Detection of rise and/or fall of cardiac biomarker values (preferably troponin) with at least one value above the 99th percentile of the upper reference limit and with at least one of the following:

- 1. Symptoms of ischaemia;
- 2. New or presumably new significant ST-T changes or new LBBB;
- 3. Development of pathological Q waves in the ECG;
- 4. Imaging evidence of new loss of viable myocardium, or new regional wall motion abnormality;

Inclusion Criteria

- Patients with confirmed acute myocardial infarction.
- Age>18 years

Exclusion Criteria

- Patients having CS because of other reasons.
- Patients of age<18 years

The protocol design, tools for data collection, consent forms and patient information sheets was reviewed and approved by the Institutional Ethics Committee.

RESULTS

The study included 250 cases fulfilling inclusion criteria. This analysis pertains to the 25 (10%) patients who unequivocally developed shock after admission. Out of 25 patients who developed shock 13 expired (52%mortality) (Table 1).

Table 1. Prevalence of Risk Factors in Patients with Cardiogenic Shock

	Cardiogenic shock (n=25)	No shock (n=225)
Age (Years)	60-65	55-60
Male (%)	64	73.8
Dm (%)	40	20
HTN (%)	60	53.33
Previous mi (%)	40	22
AWMI (%)	68	64
Smoking (%)	64	68.88
H/o angina (%)	48	35.55
Killip class (%)		
I	68	89
Ii	28	8.9
Iii	4	1.3

Age Wise Distribution of Cardiogenic Shock: Most of the Cardiogenic Shock occurred predominantly in the age group 60-65 years.

Gender Wise Distribution of Cardiogenic Shock: 64% of patients were males who developed shock. 12.23% of the total female patients developed shock compared to 8.79% of the total male patients.

Diabetes Mellitus and Cardiogenic Shock: 40% of patients who developed shock had DM as a risk factor.18.18% of total diabetic patients developed shock.

Hypertension and Cardiogenic Shock: 11.11% of the Hypertensive patients developed shock.60% of patients who developed shock had hypertension as the risk factor.

Previous MI and Cardiogenic Shock: 40% of patients who developed shock had history of previous myocardial infarction. On the same ground around 16.66% patients who had history of previous myocardial infarction developed shock.

Smoking and Cardiogenic Shock: 64% patients who developed shock had smoking as addiction.

Anterior MI and Cardiogenic Shock: Around 68% patients who developed shock were having Anterior wall MI.

Killip Class and Cardiogenic Shock: Patients who developed shock had higher Killip class. 7.76%, 25.6% and 25% belonging to Killip Class I, II and III respectively developed shock.

Angina and Cardiogenic Shock: 48% patients who developed shock had h/0 angina compared to 35.55% in the non-shock group.

DISCUSSION

The current work represents a hospital based prospective study of Acute Myocardial Infarction patients. In our study the incidence of CS was 10%. The strongest predictors were age, gender, diabetes mellitus, hypertension, previous myocardial infarction, smoking, anterior infarct, higher Killip class, and history of angina.

Age: Jeger *et al.* 2011 compared two randomized controlled trials (RCT), i.e. SMASH and SHOCK. After adjustment for confounding variables there was no significant difference in the treatment effect by age. Despite large differences in shock severity, Early revascularization (ERV) benefit is similar across all ages and not significantly different for the elderly. Advanced age was not found to be as strong a risk factor for survival in patients with CS in our study; comorbidities and less aggressive treatment appear to be the major factors resulting in poor outcomes in the elderly patient with CS.

Gender: In the large multicenter national registry genderrelated difference in mortality after ST-segment elevation myocardial infarction was studied by Sadowski *et al.* 2011. The study group consisted of 8,989 (34.5%) females and 17,046 (65.5%) males. Women were older (69.7 \pm 11 vs. 62 \pm 12 years) and had more risk factors. Percutaneous coronary intervention was performed significantly less in women (47.8% vs. 57.4%). There was a longer time delay in women at each stage of treatment. The in-hospital mortality was higher in the female group. Despite poor clinical characteristics, less than satisfactory management and a worse prognosis of STEMI in women, being a woman itself is not a risk factor for increased long-term mortality, however, other well known risk factors affecting the prognosis relate frequently to the female gender.

Diabetes Mellitus: Lindholm et al. 2005. studied the baseline characteristics and in-hospital complications to the infarction in 6676 patients with MI. Diabetes was present in 10.8% of the total population. A total of 443 developed CS with an incidence of 6.2% among non-diabetics and 10.6% among diabetics. As such it was concluded that CS develops approximately twice as often among diabetics as among nondiabetic patients with acute MI. The prognosis of diabetics with CS is similar to the prognosis of non-diabetic patients with CS. Fujiwara et al. 2002 concluded from their study that DM is not an independent predictor of death in patients with AMI after stenting, but diabetic patients had a higher incidence of Target Vessel Revascularisation (TVR), making DM an independent predictor of MACE. As such the presence of diabetes as co-morbidity in patients with AMI appears to be associated with increased mortality compared with nondiabetic patients, and this relationship may be potentially magnified in patients who develop CS.

Hypertension: The issue whether hypertension worsens the clinical course and short-term prognosis of patients with acute ST-elevation myocardial infarction (STEMI) has been addressed by several studies, however, the results were not uniform. In our study nearly 60% patients who developed shock had HTN. Rembek et al. 2010 studied 366 patients with STEMI, of whom 234 (63.9%) had a history of hypertension. They found that the clinical course of MI was more complicated in patients with hypertension who had more often cardiogenic shock (10 vs. 6%), pulmonary oedema (12 vs. 4%), sinus tachycardia>90 beats/min on admission (12 vs. 4%), ventricular tachycardia or fibrillation (20 vs. 11%) and complete atrioventricular block (11 vs. 4%). In-hospital deaths occurred in 18 (7.7%) patients with hypertension and 7 (5.3%) patients without hypertension (NS). As such patients with STEMI and hypertension have more cardiovascular risk factors and more complicated in-hospital course of MI than normotensive patients.

Prior MI: Despite improved secondary prevention efforts, acute coronary syndrome (ACS) recurrence among patients with prior history of coronary events remains high. In my study around 40% patient who developed shock had h/o previous MI. Motivala *et al.* 2008. A found in their study that patients with a prior MI were more likely to be older and have a higher incidence of diabetes mellitus, hypertension, hyperlipidemia, and peripheral vascular disease.

Smoking: 64% patients who developed shock had smoking as addiction. Gottlieb *et al.* 1996 found that smokers had smaller areas of damage to their heart after the heart attack than nonsmokers. In their study, that theory certainly appeared to be true, with 21.5% of nonsmokers dying within six months of their heart attack compared to only 7.9% of smokers. But when Gottlieb and colleagues adjusted the data to take into account age and age-related factors at the time of the attack, the differences between the two groups disappeared, leaving no

advantage for smokers. The average age of nonsmokers in the study was 67 years versus 57 years for smokers. The researchers concluded that the message of the study is not that smoking improves prognosis after a heart attack, but that it actually causes heart attacks earlier.

Type of Infarction: In our study around 68% patients who developed shock had Anterior Wall MI. Tsai *et al.* 2010 compared the 30-day mortality from CS caused by left anterior descending artery (LAD) occlusion with that caused by left circumflex (LCX) or right coronary artery (RCA) occlusion after primary percutaneous coronary intervention (PCI) in Taiwan. The researchers could not find any significant difference in 30-day mortality between patients with acute myocardial infarction and CS caused by LAD occlusion and by either RCA or LCX occlusion undergoing primary PCI. Among patients with CS, similar mortality was observed in patients with anterior myocardial infarction (MI) or inferior MI with/without right ventricle involvement in the study done by Lee *et al.* 2004.

Killip Class (Killip and Kimball, 1967): Worsening Killip class has been found to be independently associated with increasing mortality in my study.7.76%, 25.6% and 25% belonging to Killip Class I, II and III respectively developed shock. Hasdai et al. 2000 analyzed baseline variables associated with the development of shock after thrombolytic therapy in the Global Utilization of Streptokinase and Tissue-Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial. Using a Cox proportional hazards model, the researchers devised a scoring system predicting the risk of shock. This model was then validated in the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO-III) cohort. Shock developed in 1,889 patients a median of 11.6 h after enrollment. The major factors associated with increased adjusted risk of shock were, systolic blood pressure, heart rate and Killip class upon presentation. Together, these four variables accounted for >85% of the predictive information for the GUSTO-I and > 95% for GUSTO-III Trials.

H/O Angina: History of Angina was identified as one of the important risk factors for the in-hospital development of CS in the study done by Mark Hands *et al.* 1989, Holmes *et al.*, 1999 stated that prior angina is present in nearly 52% patients of acute myocardial infarction patients who developed shock.

Limitations: Though our study is one among the few prospective studies for determining incidence of CS in patients with acute myocardial infarction, however to determine the exact incidence we need large sample population covering primary, secondary and tertiary centers. Further we only studied patient who were primary thrombolysed.

Conclusion

The incidence of cardiogenic shock (CS) was around 10% in ST-segment elevation myocardial infarction (STEMI) in our study. The strongest predictors for development of CS in our study were age, gender, diabetes mellitus, hypertension, previous myocardial infarction, smoking, anterior infarct, higher Killip class, and history of angina. The mortality among patients with CS in our study was 52%.

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Recommendations: However, to determine exact incidence and risk factors of cardiogenic shock, large volume population studies across various hospitals are required, so that mortality due to cardiogenic shock can be reduced.

REFERENCES

- Fox KA, Anderson FA, Dabbous OH et al. 2007. Intervention in acute coronary syndromes: do patients undergo intervention on the basis of their risk characteristics? The Global Registry of Acute Coronary Events (GRACE). *Heart.*, 93:177-82.
- Fujiwara K, Hiasa Y, Takahashi T et al. 2002. Influence of diabetes mellitus on outcome in the era of primary stenting for acute myocardial infarction. *Circulation Journal*, 66:800-4.
- Ginsberg F, Parrillo JE. 2009. Cardiogenic shock in other heart. *Cardiogenic Shock*, 172.
- Gottlieb S, Boyko V, Zahger D et al. 1996. Smoking and prognosis after acute myocardial infarction in the thrombolytic era (Israeli Thrombolytic National Survey). *Journal of the American College of Cardiology*, 28:1506-13.
- Hands ME, Rutherford JD, Muller JE et al. 1989. The inhospital development of cardiogenic shock after myocardial infarction: incidence, predictors of occurrence, outcome and prognostic factors. Journal of the American College of Cardiology, 14:40-6.
- Hasdai D, Califf RM, Thompson TD et al. 2000. Predictors of cardiogenic shock after thrombolytic therapy for acute myocardial infarction. *Journal of the American College of Cardiology*, 35:136-43.
- Hasdai D, Harrington RA, Hochman JS et al. 2000. Platelet glycoprotein IIb/IIIa blockade and outcome of cardiogenic shock complicating acute coronary syndromes without persistent ST-segment elevation. *Journal of the American College of Cardiology*, 36:685-92.
- Hochman JS, Buller CE, Sleeper LA et al. 2000. Cardiogenic shock complicating acute myocardial infarction etiologies, management and outcome: a report from the SHOCK Trial Registry. *Journal of the American College of Cardiology*, 36:1063-70.
- Hollenberg SM, Kavinsky CJ, Parrillo JE. 1999. Cardiogenic shock. *Annals of internal medicine*, 131:47-59.

- Holmes DR, Berger PB, Hochman JS et al. 1999. Cardiogenic shock in patients with acute ischemic syndromes with and without ST-segment elevation. *Circulation*, 100:2067-73.
- Jeger RV, Urban P, Harkness SM et al. 2011. Early revascularization is beneficial across all ages and a wide spectrum of cardiogenic shock severity: a pooled analysis of trials. *Acute cardiac care*, 13(1):14-20.
- Joint European Society of Cardiology/American College of Cardiology Committee.. Myocardial infarction redefined. A consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *Eur Heart J.*, 2000; 21: 1502-1513.
- Killip III T, Kimball JT. 1967. Treatment of myocardial infarction in a coronary care unit: a two year experience with 250 patients. *The American Journal of Cardiology*, 20:457-64.
- Lee CH, Hoye A, Lemos PA et al. 2004. Predictors of survival after contemporary percutaneous coronary revascularization for acute myocardial infarction in the real world. *The Journal of invasive cardiology*, 16:627-31.
- Lindholm MG, Boesgaard S, Torp Pedersen C et al. 2005. Diabetes mellitus and cardiogenic shock in acute myocardial infarction. *European Journal of Heart Failure*, 7:834-9.
- Motivala AA, Tamhane U, Ramanath VS et al. 2008. A prior myocardial infarction: how does it affect management and outcomes in recurrent acute coronary syndromes?. *Clinical cardiology*, 31:590-6.
- Rembek M, Goch A, Goch J. 2010. The clinical course of acute ST-elevation myocardial infarction in patients with hypertension. Kardiologia Polska. *Polish Heart Journal*, 68:157-63.
- Sadowski M, Gasior M, Gierlotka M et al. 2011. Genderrelated differences in mortality after ST-segment elevation myocardial infarction: a large multicentre national registry. EuroIntervention: journal of Euro PCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology, 6:1068-72.
- Tsai TH, Chai HT, Sun CK et al. 2010. Comparison of 30-day mortality between anterior-wall versus inferior-wall STsegment elevation myocardial infarction complicated by cardiogenic shock in patients undergoing primary coronary angioplasty. *Cardiology*, 116:144-50.
