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RESEARCH ARTICLE

Predictive Factors and Outcomes in Cardiogenic Shock in the Setting of Acute Myocardial Infarction

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Abstract

Introduction: Acute myocardial infarction (AMI) is the one of the leading causes of death in the United States and worldwide. In recent years, there has been a decline in the incidence and case fatality of AMI, which is partly attributed to the advancements in management including timely reperfusion and medical therapies. Cardiogenic shock (CS) is a life-threatening complication in patients with acute coronary syndrome (ACS), and its development can be unpredictable. The aim of this study was to find independent predictive factors of CS in cohort of ACS patients.

Methods: This was a retrospective, comparative, and analytical monocentric study, including 319 ACS patients admitted at Department of Surgery, 250 Bed District Sadar Hospital, Habiganj, Bangladesh from January to December 2024. Patients who presented with CS on admission were excluded from the study. This population was divided into two groups: the shock group patients eventually developed in-hospital CS and the no shock group which did not, and we compared overall patient characteristics and outcomes. Studied characteristics included patient demographics (age, sex), medical history (cardiovascular risk factors and comorbidities), clinical status including the presence of heart failure (HF), electrocardiogram data, laboratory findings such as high-sensitivity troponin and glomerular filtration rate (eGFR), echocardiographic findings mainly left ventricular ejection fraction (LVEF) and left ventricular hypertrophy (LVH), and lesions found during coronary angiography.

Results: 319 ACS patients were included, among them 21 (6,6%) developed CS. Overall, the strongest predictive factors included the presence of acute heart failure on admission (OR = 14,83; 95% CI = 5,45 – 40,32; p < 0,001), GRACE score ≥ 140 (OR = 9,03; 95% CI = 3,20 – 25,46; p < 0,001), left ventricular ejection fraction < 50% (OR = 8,94; 95% CI = 3,08 – 19,53; p < 0,001), eccentric left ventricular hypertrophy (OR = 9,78; 95% CI = 2,61 – 36,70; p < 0,001), and right ventricular dysfunction (OR = 12,25; 95% CI = 2,55 – 58,93; p = 0,002). Complications were more prevalent in the shock group with a higher mortality rate of 57,1%.

Conclusion: CS in the setting of ACS is correlated with poorer prognosis and higher late mortality, justifying adequate and early diagnosis and management in high-risk patients.

Keywords: Cardiogenic Shock, Acute Coronary Syndrome, Predictive Factors.

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1. Introduction

Acute myocardial infarction (AMI) is the one of the leading causes of death in the United States and worldwide [1]. In recent years, there has been a decline in the incidence and case fatality of AMI, which is partly attributed to the advancements in management including timely reperfusion and medical therapies[2,3]. Despite these improvements, sex disparity still has an impact on AMI management and outcomes[3].The Cardiogenic shock (CS) is an uncommon but life-threatening complication of acute coronary syndrome (ACS), characterized by a low cardiac output state and end-organ hypo perfusion [4]. Despite major advancements in medical and interventional therapy, it remains a leading cause of death in ACS, and represents a real challenge for emergency and cardiology physicians [5]. All the current guidelines highlight the importance of early diagnosis and management to improve prognosis [4,6,7]. Cardiogenic shock is the most common cause of death in patients with AMI, resulting from left ventricular pump failure or as a consequence of post-MI mechanical complications such as papillary muscle rupture, ventricular septal rupture, free wall rupture or right ventricular failure [8,9]. These variables include age greater than 70 years old, previous stroke or transient ischemic attack, cardiorespiratory arrest on admission, previous STEMI, delay from initial medical contact to percutaneous transluminal coronary angioplasty greater than 90 minutes, Killip-Kimball classification. Cardiogenic shock affects 5%-10% of AMI cases and is associated with high mortality (up to 30%-40%), despite advances in pharmacological, mechanical and reperfusion endeavors [10,11]. Similar to AMI without cardiogenic shock, sex differences exist in management and outcomes among those with cardiogenic shock [12]. In this review, we discuss the sex disparities in the risk profile, management, and outcomes of cardiogenic shock in the setting of AMI, and present few solutions to the existing challenges. Most often the cause of cardiogenic shock is a serious heart attack. Other health problems that may lead to cardiogenic shock include heart failure, which happens when the heart can't pump enough blood to meet the body's needs; chest injuries; and blood clots in the lungs. Cardiogenic shock is the most common cause of in-hospital death after acute coronary syndromes. Myocardial dysfunction triggers a compensatory systemic vascular response. The key to diagnosis is demonstration of end-organ hypoperfusion. The purpose of our study was to identify independent predictors of the development of CS in a heterogeneous population of Moroccan patients admitted for ACS.

2. Methods and Materials

This was a retrospective, comparative, and analytical monocentric study, including 319 ACS patients admitted at Department of Surgery, 250 Bed District Sadar Hospital, Habiganj, Bangladesh from January to December 2024. Patients who presented with CS on admission were excluded from the study. This population was divided into 2 groups: the « shock » group patients eventually developed in hospital CS and the no shock, group did not. CS was defined as a sustained episode of hypotension (systolic blood pressure < 90 mmHg or the need of vasopressors to maintain systolic blood pressure > 90 mmHg) for >30 min associated with clinical or paraclinical evidence of elevated left ventricular filling pressures in addition to the presence of end-organ hypo perfusion such as altered mental status or oliguria [6,7]. ACS, as well as its three subtypes unstable angina (UA), non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI), was diagnosed using the latest European practice guidelines [13,14]. Studied characteristics included patient demographics (age, sex), medical history (cardiovascular risk factors and comorbidities), clinical status including the presence of heart failure (HF), electrocardiogram data, laboratory findings such as high-sensitivity troponin and glomerular filtration rate (eGFR), echocardiographic findings mainly left ventricular ejection fraction (LVEF) and left ventricular hypertrophy (LVH), and lesions found during coronary angiography.

3. Results

A total of 319 patients were included in our study. 21 (6,6%) patients developed in-hospital CS and were included in the « shock » cohort. Baseline characteristics as well as in-hospital outcomes of the « shock » and « no shock » groups can be found in the appendix. Patients in the shock group were older (67,1 ± 7,0 vs. 63,5 ± 9,6 years old; p = 0,036). Chronic kidney disease (CKD) was most associated with development of CS (23,8 vs. 7,4%; p = 0,041). The shock group most often presented with atypical symptoms, such as abdominal pain, dyspnea, and acute heart failure (HF) was much more prevalent (57,1 vs. 11,7% for left-sided HF and 4,8 vs 0,8% for right-sided HF). There was a higher proportion of « shock » patients presenting with atrial fibrillation (AF) or right bundle branch block (14,3 vs. 2,3% and 14,3 vs. 4,0%; p = 0,002 and 0,032 respectively).

Echocardiography performed on « shock » patients found reduced mean left ventricular ejection fraction (LVEF) (36,6 ± 11,5 vs. 51,8 ± 10,6%; p < 0,001), more left ventricular wall motion abnormalities (LVWMA) as well as a higher rate of LVH and right ventricular (RV) dysfunction. Proximal and mid coronary lesions were more common in that group as well. There was a high degree of correlation between the final diagnosis and CS development; CS patients were more likely to have STEMI (76,2 vs. 35,9%; p < 0,001). NSTEMI was associated with a lower risk (23,8 vs. 50,3%; p = 0,019), while none of the UA patients developed CS in our study.

Table 1. *Predictive factors of cardiogenic shock in patients with ACS*

Variables	Odds ratio	95% CI	p-value
Clinical characteristics			
Age ≥ 65 years old	3,23	1,22 – 8,56	0,018
CKD	3,92	1,31 – 11,71	0,014
Clinical presentation on admission			
Atypical symptoms (no chest pain)	7,34	2,65 – 20,35	< 0,001
Acute heart failure	14,83	5,45 – 40,32	< 0,001
Killip class ≥ II	9,87	3,86 – 25,24	< 0,001
ECG on admission			
Atrial fibrillation	6,93	1,65 – 29,06	0,008
Bundle branch block	2,69	1,02 – 7,85	0,007
Biological findings			
Peak troponin ≥ 50000 ng/L	4,22	1,68 – 10,59	0,002
eGFR < 60 mL/min/1,73 m²	4,11	1,66 – 10,15	0,002
GRACE score ≥ 140	9,03	3,20 – 25,46	< 0,001
Echocardiographic findings			
LVEF < 50%	8,94	2,94 – 27,30	< 0,001
LVEF < 40%	7,76	3,08 – 19,53	< 0,001
Number of LV segments with WMA ≥ 9	5,30	2,04 – 13,79	< 0,001
Eccentric left ventricular hypertrophy	9,78	2,61 – 36,70	< 0,001
Right ventricular dysfunction	12,25	2,55 – 58,93	0,002
Angiographic findings			
Proximal culprit lesion	2,91	1,17 – 7,23	0,021
Final diagnosis			
STEMI diagnosis	5,71	2,04 – 16,03	< 0,001

3.1 Predictors of In-Hospital Development of Cardiogenic Shock Design and Population

A list of unavailable predictors of in-hospital development of CS can be found in Table 1. In total, 17 variables were identified. The strongest included the presence of acute HF on admission (OR = 14,83; 95% CI = 5,45 – 40,32; p < 0,001), GRACE score ≥ 140 (OR = 9,03; 95% CI = 3,20 – 25,46; p < 0,001), LVEF < 50% (OR = 8,94; 95% CI = 3,08 – 19,53; p < 0,001), eccentric LVH (OR = 9,78; 95% CI = 2,61 – 36,70; p < 0,001), and RV dysfunction (OR = 12,25; 95% CI = 2,55 – 58,93; p = 0,002).

Table 2. *Predictive factors of cardiogenic shock in patients with ACS according to infarct localization*

Variables	Odds ratio	95% CI	p-value
Anterior Localization			
Clinical characteristics			
Age ≥ 65 years old	4,61	1,04 – 22,46	0,048
Clinical presentation on admission			
Atypical symptoms (no chest pain)	12,15	1,81 – 81,72	0,010
Acute heart failure	13,44	3,22 – 56,16	< 0,001
Killip class ≥ II	13,44	3,22 – 56,16	< 0,001
Biological findings			
GRACE score ≥ 140	5,74	1,42 – 23,31	0,014
Echocardiographic findings			
LVEF < 50%	15,11	1,86 – 122,66	0,011
LVEF < 40%	11.47	2,77 – 47,55	< 0,001
Eccentric left ventricular hypertrophy	11,58	1,69 – 79,48	0,012

Number of LV segments with WMA ≥ 9	8,18	2,13 – 31,38	0,002
Final diagnosis			
STEMI diagnosis	28,64	1,65 – 498,25	0,021

Table2. (Continue)

Variables	Odds ratio	95% CI	p-value
Inferior Localization			
ECG on admission			
Bundle branch block	29,67	3,06 – 287,94	0,004
Biological findings			
Peak troponin ≥ 50000 ng/L	20,27	2,11 – 194,26	0,009
eGFR < 60 mL/min/1,73 m²	7,60	1,17 – 49,46	0,034
GRACE score ≥ 140	5,74	1,42 – 23,31	0,014
Echocardiographic findings			
Right ventricular dysfunction	60,00	4,19 – 859,39	0,003
Other Localizations			
Clinical characteristics			
CKD	8,29	1,39 – 49,24	0,020
Clinical presentation on admission			
Atypical symptoms (no chest pain)	15,25	2,30 – 101,28	0,048
Acute heart failure	61,61	3,24 – 1172,36	0,006
Biological findings			
eGFR < 60 mL/min/1,73 m²	10,48	1,15 – 95,41	0,037
GRACE score ≥ 140	50,56	2,68 – 954,41	0,009
Echocardiographic findings			
LVEF < 50%	14,12	1,54 – 129,62	0,019
LVEF < 40%	9,83	1,61 – 59,93	0,013
Eccentric LVH	32,00	2,37 – 432,73	0,009

3.2 Subgroup Results

Subgroups of patients were created according to infarct localization (Table 2) and final diagnosis (Table 3). Predictive factors differed according to infarct localization; acute HF, reduced LVEF and eccentric LVH were the main variables isolated in no inferior ACS, while the presence of a bundle branch block and RV dysfunction played much more of a role in inferior ACS. Altered renal function was not a predictive factor in anterior ACS but was strongly associated with CS development in non-anterior ACS. In NSTEMI patients, the main predictive factors were acute HF, AF, renal failure, a high GRACE score, and both LV and RV dysfunction. Most of these variables were also found in STEMI patients, with bundle branch block instead of AF, in addition to advanced age, eccentric LVH and proximal culprit lesion.

Table 3. *Predictive factors of cardiogenic shock in patients with ACS according to diagnosis*

Variables	Odds ratio	95% CI	p-value
NSTEMI			
Clinical characteristics			
CKD	13,50	2,09 – 87,33	0,006
Prior CABG	12,25	1,03 – 145,05	0,047
Clinical presentation on admission			
Atypical symptoms (no chest pain)	21,00	3,14 – 140,51	0,002
Acute heart failure	59,68	3,19 – 1115,85	0,006
Killip class ≥ II	23,27	2,48 – 218,07	0,006
ECG on admission			
Infarct localizations other than anterior or inferior	27,18	1,47 – 502,24	0,026
Atrial fibrillation	18,50	1,38 – 248,54	0,028
Biological findings			
eGFR < 60 mL/min/1,73 m²	9,64	1,05 – 88,67	0,045
GRACE score ≥ 140	27,18	1,47 – 502,24	0,027
Echocardiographic findings			
LVEF < 50%	9,64	1,05 – 88,67	0,045
Right ventricular dysfunction	18,50	1,38 – 248,54	0,028

Table 3. (Continue)

Variables	Odds ratio	95% CI	p-value
STEMI			
Clinical characteristics			
Age ≥ 65 years old	5,61	1,38 – 22,74	0,015
Clinical presentation on admission			
Atypical symptoms (no chest pain)	5,61	1,38 – 22,74	0,015
Acute heart failure	10,22	3,24 – 32,28	< 0,001
Killip class ≥ II	7,89	2,55 – 24,37	< 0,001
ECG on admission			
Bundle branch block	8,00	1,46 – 43,84	0,016
Biological findings			
eGFR < 60 mL/min/1,73 m²	3,65	1,24 – 10,79	0,003
GRACE score ≥ 140	5,65	1,81 – 17,62	0,003
Echocardiographic findings			
LVEF < 50%	6,20	1,67 – 23,07	0,006
LVEF < 40%	7,31	2,38 – 22,45	< 0,001
Number of LV segments with WMA ≥ 9	6,16	1,94 – 19,56	0,002
Eccentric LVH	24,46	2,37 – 252,76	0,007
Right ventricular dysfunction	15,14	1,29 – 178,02	0,031
Angiographic findings			
Proximal culprit lesion	3,81	1,29 – 11,22	0,015

4. Discussion

Cardiogenic shock (CS) in acute coronary syndrome (ACS) is a critical disease with high mortality rates requiring complex treatment to maximize patient survival chances. Emergent coronary revascularization along with circulatory support is keys to saving lives. Coronary artery revascularization. The cornerstone of treatment that improved CS prognosis in AMI patients is emergent coronary revascularization (or Percutaneous Coronary Intervention PCI) in patients with coronary artery disease [4]. The main cause of cardiogenic shock is a heart attack, which is a complication of coronary heart disease. You can lower your risk of cardiogenic shock by taking steps to prevent a heart attack or other heart problems. This means adopting heart-healthy lifestyle changes to help prevent or treat coronary heart disease. CS remains a major clinical challenge, and ischemia is by far its most prevalent etiology, accounting for about 80% of cases [15]. Despite the recent progress made regarding revascularization therapy, the development of CS still portends an extremely poor prognosis, with mortality reaching 40 to 50% in some cohorts [5, 16]. Therefore, early identification of high-risk patients would be a major step for clinical decision. Some studies have even suggested preventive therapy such as early fibrinolysis to improve outcomes, especially in Morocco where primary PCI is not always readily available [17, 18]. They also presented a greater prevalence of CKD, which is associated with accelerated infarct expansion and enhanced inflammation making for a poorer prognosis in ACS patients [19,20]. Their initial clinical status was much poorer, with an increased incidence of acute HF and a higher Killip class. As previously stated, CS encompasses a spectrum that often begins with signs of HF before progressing into overt shock [6,21]. In our study, AF was a strong predictor of CS development; this is supported by a recent Portuguese study which reported that new-onset AF in ACS patients was correlated with a higher risk of congestive HF, CS, ventricular tachycardia as well as mortality [22]. AF precipitates heart failure by worsening left ventricular filling and lowering LVEF and contributes to thrombus formation. High troponin was also strongly correlated with CS development. Troponin measurements accurately predict infarct size, and it has been known for a long time that quantitative elevation was associated with a higher risk of major cardiac events in both NSTEMI and STEMI patients [23, 24]. Bedside echocardiography is routinely performed on ACS patients to assess hemodynamic status myocardial damage and to diagnose complications. Our study has showed that it could also be essential in the prediction of CS development: patients with lower LVEF, eccentric LVH or RV dysfunction were at higher risk of complication. LV pump failure is the main mechanism responsible for CS, therefore early recognition is absolutely essential in all patients presenting with ACS [6,7]. Angiography performed on our patients showed that proximal lesions were more common

in the « shock » group. Proximally located lesions imply a larger infarcted myocardial territory, making CS much more likely, as reported by a substudy of the IABPSHOCK II-trial published in 2016 [25]. Cardiogenic shock occurs more often in STEMI than in NSTEMI [19]; in our study, STEMI diagnosis was an independent predictor of CS development. Mortality remains high in both conditions. Despite this, many studies have found differences between the 2 entities. The 30- day mortality was higher, and NSTEMI was found to be an independent predictor of mortality in multivariable analysis [26]. The SHOCK trial registry reported similar differences in baseline characteristics and also found that NSTEMI patients were less likely to undergo angiography [27]. They presented with a lower LVEF. The NSTEMI group also had more 3-vessel disease, and mortality rate was higher (40.8 versus 33.1%) [28]. Therefore, NSTEMI and STEMI patients have different characteristics and comorbidities that influence management, furthermore the delay in NSTEMI revascularization compared to STEMI makes for a paradoxically poorer prognosis. For NSTEMI patients at high risk of developing CS, revascularization with the same urgency as STEMI shock is the best approach to improve outcomes. The mortality rate of CS calculated in our study (57,1%) is in accordance with previous findings [29-32]. Untreated CS invariably evolves into organ failure, as such many complications can arise (both cardiac and non-cardiac), contributing to the overall high mortality rate. In our study, arrythmias were much more prevalent in the « shock » group. They are common in CS patients and often result in hemodynamic deterioration; they were involved in 37% of deaths in the SHOCK Trial. The same thing can be said about LV thrombus formation, a common occurrence in CS patients, especially in the presence of low LVEF or AF. In the SHOCK trial, strokes caused 3,21% of deaths within the first 30 days [33]. In our study, AKI was much more prevalent in CS patients. Our rate of 38, 1% is similar to other reports which vary between 20 and 35%. AKI in the setting of CS is multifactorial, mainly due to renal hypoperfusion or toxicity due to medication. It is correlated with higher overall morbidity and mortality [34]. Medications to treat cardiogenic shock are given to increase your heart's pumping ability and reduce the risk of blood clots. Vasopressors. These medications are used to treat low blood pressure. They include dopamine, epinephrine (Adrenaline, Auvi-Q), norepinephrine (Levophed) and others.

5. Conclusion

Despite substantial improvements in management, the prognosis of post-ACS CS remains poor. Therefore, early identification of patients at high risk of CS development is of great interest to emergency physicians and cardiologists. Data from our study suggest that clinicians should pay great attention to elderly patients or those with CKD. Increased surveillance, intensive care, and potential interventions such as early revascularization and mechanical support of pre-shock patients (even in the setting of NSTEMI) could prevent the development of overt CS and improve outcomes. Bedside echocardiography is an essential tool as LV and RV assessment provide valuable data for risk stratification. Troponin and creatinine measurements should also help in management decision-making.

Conflict of Interest

None.

Source of Fund

Nil.

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