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Cardiogenic Shock in the Setting of Acute Coronary Syndrome: Predictive Factors and Outcomes in a Moroccan population

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Abstract | **Original Research Article**

Introduction: 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 a Moroccan cohort of ACS patients. ***Methods:*** This was a retrospective, comparative, and analytical monocentric study, including 319 ACS patients admitted between January 2018 to April 2021 in MVMIH's cardiology center. 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. ***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.

INTRODUCTION

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 hypoperfusion [1].

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 [2]. All the current guidelines highlight the importance of early diagnosis and management to improve prognosis [1,3,4].

In Morocco, coronary heart disease is a major cause of death and associated with a high socioeconomic burden [5]. 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.

METHODS

Study design and population

Our work was a retrospective, comparative, and analytical monocentric study, which enrolled 319 ACS patients who were admitted in the Mohammed V Military Instruction Hospital's (MVMIH) Cardiac Intensive Care Unit in Rabat, Morocco, from January 2018 to April 2021. 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.

Definitions

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

hypoperfusion such as altered mental status or oliguria [3, 4].

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 [6, 7].

DATA COLLECTION

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.

STATISTICAL ANALYSIS

Statistical analysis was performed using SPSS 19.0. Values were expressed as mean±SD and discrete variables were presented as percentages. First, univariate analysis was performed to compare the « shock » and « no shock » groups and to identify potential risk factors; statistical comparisons between groups were performed using Student's t-test for continuous variables, and Pearson's chi-square test or Fisher's exact test for categorical variables, as appropriate. A value of $p < 0.05$ was considered statistically significant. Next, logistic regression analysis was performed on suspected risk factors to find independent predictors of CS development. Additional models were constructed for subgroups of patients depending on diagnosis (NSTEMI vs. STEMI) and the territory of the infarction (anterior vs. inferior vs. other).

RESULTS

Patient characteristics

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).

Peak high-sensitivity troponin was found to be much higher in the « shock » cohort (mean of 109662 vs 30851 ng/L; p < 0,001), as well as GRACE score (151 ± 24 vs. 118 ± 34; p < 0,001), and eGFR was reduced in that population (48,6 ± 26,7 vs. 72,9 ± 24,2; p < 0,001). 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.

The prognosis of the « shock » group was poorer, with a higher rate of complications such as left ventricular (LV) thrombus (9,5 vs. 1,7%; p = 0,018), arrhythmia (both supraventricular and ventricular), and acute kidney injury (AKI) (38,1 vs. 1,0%; p < 0,001), with a mortality rate reaching 57,1% (vs. 1,3%; p < 0,001).

Predictors of in-hospital development of cardiogenic shock design and population

A list of univariable 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-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**

CKD, chronic kidney disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; LV, left ventricle; LVEF, left ventricular ejection fraction; STEMI, ST-elevation myocardial infarction; WMA, wall motion abnormalities

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 non-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-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**
➔ 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**
CKD, chronic kidney disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; LV, left ventricle; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; STEMI, ST-elevation myocardial infarction; WMA, wall motion abnormalities			

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**
➔ 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**
CABG; coronary artery bypass grafting; CKD, chronic kidney disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; LV, left ventricle; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; WMA, wall motion abnormalities			

DISCUSSION

CS complicated 6, 6% of our cohort, which is consistent with previous studies reporting rates between 6 and 8% [8, 9]. CS remains a major clinical challenge, and ischemia is by far its most prevalent etiology, accounting for about 80% of cases [10]. 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 [2, 11]. CS is a spectrum ranging from pre-shock (stage A) to overt refractory shock (stage D-E), and most ACS are complicated within hours or days after admission (so-called "late CS") [3, 12]. 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 [13, 14]. These patients might also benefit from more aggressive monitoring and early transfer to tertiary care centers. As such, several attempts at a prediction score have already been made, notably the ORBI Risk Score, published in 2018 [15-17], but they have mostly been validated in European or Asian populations. Our study attempts to find predictive factors specific to the Moroccan setting.

General observations

CS patients were older, which is in line with most recent studies [15-19]. 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 [20, 21]. 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 [3, 12].

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 [3, 4].

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 IABP-SHOCK II-trial published in 2016 [25].

STEMI vs. NSTEMI

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.

In the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO) IIb trial, NSTEMI patients who developed CS were older, had a higher prevalence of diabetes mellitus (DM) and 3-vessel coronary artery disease, than STEMI patients who developed CS. Shock also developed much later (9,6 vs 76,2 hours). 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]. A more recent analysis of the National Cardiovascular Data Registry showed that NSTEMI patients who developed CS were older and more likely to be female, have DM, a history of MI or congestive HF compared with STEMI patients. 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.

Outcomes

The mortality rate of CS calculated in our study (57,1%) is in accordance with previous findings [16-19]. 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 [29].

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 [30].

Limitations

Our work was retrospective and observational, which suffers from limitations inherent to this kind of study. It was also monocentric, which is a source of bias.

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. 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. 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.

Competing interests

The authors declare no competing interest.

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