Influence of SCAI stage transition on clinical outcomes in patients with acute myocardial infarction

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ABSTRACT

Objective: To describe the association between the transition of SCAI stage and clinical outcomes in patients with acute myocardial infarction.

Subjects and methods: A cross-sectional study on 156 patients with acute myocardial infarction at Heart Institute, Bach Mai Hospital from 8/2022 to 2/2023. Clinical status, laboratory parameters and SCAI classification were assessed at the time of admission and 24 hours after admission. Clinical outcomes at the hospital included death and the patient was discharged in severe condition.

Results: The distribution of the patients across SCAI shock stages on admission was 46.2%, 19.2%, 30.8%, 2.5% and 1.3% to A, B, C, D and E, respectively. Statistically significant difference in SCAI stages were observed in clinical and laboratory parameters such as heart rate, cardiac biomarker concentrations, pH and blood lactate. The clinical outcomes were significantly higher in the more severe SCAI stage. The SCAI shock transition was determined at 24 hours, 14.1% improved, 60.3% remained and 25.6% worsened SCAI stage. Improved SCAI stages were associated with better clinical outcomes (OR 0.06; 95% CI 0.02 – 0.14). Worse SCAI stages were associated with an increase in clinical outcomes (OR 9.00; 95% CI 3.20–25.29).

Conclusion: Transitions of SCAI stage are valuable in predicting clinical outcomes in patients with acute myocardial infarction.

Keywords: Acute myocardial infarction, cardiogenic shock, SCAI classification.

INTRODUCTION

Cardiogenic shock due to myocardial infarction is a major challenge in clinical practice even in developed countries. The incidence of cardiogenic shock is about 5.9 - 8.3% of patients with acute myocardial infarction.[1,2] Early revascularization and culprit only revascularization in patients with cardiogenic shock due to myocardial infarction have been shown to improve cardiovascular events.[3–5] Other management strategies in acute myocardial infarction associated cardiogenic shock such as hypothermia and left ventricular assist devices have not been shown to improve mortality in the short or long term.[6–9] Therefore, the hospital mortality rate due to cardiogenic shock in patients with acute myocardial infarction is nearly 40 - 50% and does not seem to have changed in the last 20 years.[1,10–13] Cardiogenic shock team is a working group of many doctors in different specialties including critical care cardiology, advanced heart failure, interventional cardiology, cardiac surgery and

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ECMO service has been shown to be independent factor in improve clinical outcomes.[14,15]

Many different criteria have been published for the diagnosis and classification of cardiogenic shock but there is no common consensus among these criteria, which makes it difficult to practice and conduct research. In order to overcome these difficulties, the Society for Cardiovascular Angiography and Intervention (SCAI) and 8 other organizations including American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC), Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM) and Society of Thoracic Surgeons (STS) released the 2021 updated SCAI shock classification which replaced 2019 SCAI classification.[16,17] Three important domains including physical examination, biochemical, and hemodynamic criteria has been maintained in the updated SCAI classification. Importantly, the authors divided them into "typically include" and "may include" to emphasize cardiogenic shock stage. Thus, cardiogenic shock is not a disease stage but a continuous progression from A (At Risk) to B (Beginning), C (Classic), D (Deteriorating) and E (Extremis). The SCAI shock classification is an indication of shock severity and comprises one component of mortality risk prediction in patients with cardiogenic shock.[17] In the year of 2022, Consensus of the Vietnam National Heart Association on diagnosis and management of Cardiogenic Shock was impressed the role of SCAI shock classification in clinical practice.[18] This study was conducted to evaluate the effectiveness of SCAI classification on admission and SCAI stage transition in the clinical outcomes at the hospital of patients with acute myocardial infarction.

METHOD

Study population

This cross-sectional study included 156 patients with acute myocardial infarction at the Vietnam Heart Institute, Bach Mai Hospital from August 2022 to February 2023. ST segment elevation myocardial infarction patients were diagnosed according to the 2018 Fourth universal definition of myocardial infarction.[19] Non-ST-segment elevation myocardial infarction were diagnosed according to the 2020 European Society of Cardiology guideline.[20]

Data sources

We collected demographic, vital sign, medical histories, laboratory parameters and clinical outcomes data, as well as procedures and therapies performed during hospital stay. The vital signs, clinical measurements and laboratory values were recorded both on admission and 24 hours after admission.

SCAI SHOCK classification criteria and outcomes

Patients were assigned to a SCAI stage due to Updated SCAI shock classification at the time of admission and 24 hours after admission. SCAI A defines stage A patients as those at risk for cardiogenic shock and stable hemodynamic. Stage B patients are those exhibiting early symptoms including hypotension and/or tachycardia but not including hypo-perfusion and, therefore, do not require pharmacological or mechanical support. Stage C patients are those with hypo-perfusion (lactic acidosis, oliguria, cool/ clammy periphery, or altered mentation) requiring initial intervention. Stage D patients are those whose condition deteriorates despite initial intervention. Finally, stage E patients are those who have deteriorated further or impending circulatory collapse, including cardiac arrest with ongoing resuscitation.

The SCAI SHOCK stage at 24 hours was

compared to that at initial assessment. The group was divided into patients who have a better SCAI stage, unchanged and worse SCAI stage at 24 hours. Better SCAI stage was defined as lower SCAI stage and worse SCAI stage was higher SCAI stage at 24 hours.

Clinical outcomes at the hospital were death status and severe condition at discharge.

Statistical analysis

Patients were divided into 5 groups: SCAI A, SCAI B, SCAI C, SCAI D, and SCAI E. Baseline descriptive clinical and biochemical characteristics were summarized as mean and standard deviation for continuous variables and proportions for categorical variables, stratified by SCAI stage. Groups were compared using ANOVA test for continuous variables and χ^2 tests for categorical variables. P values with a significance level of $\alpha = 0.05$. Odd ratio (OR) and 95% CI were used to measure the association between the clinical outcomes and the transition of SCAI stage. Statistical analysis was performed using STATA 14.0 and SPSS 20.0.

RESULTS

156 patients with acute myocardial infarction including 109 patients with ST-segment elevation myocardial infarction and 47 patients with non-ST-segment elevation myocardial infarction. The general characteristics of the group are presented in Table 1.

Table 1. General characteristics of patients with acute myocardial infarction

	Result
Age[1]	68.9 ± 12.4
Female sex[2]	49 (31.4)
Hypertension[2]	99 (63.5)
Diabetes mellitus[2]	38 (24.4)
Prior myocardial infarction[2]	22 (14.1)
Prior stroke[2]	11 (7.1)
Chronic kidney disease[2]	4 (2.6)
Smoking[2]	13 (8.4)
Systolic blood pressure (mmHg)[2]	118.1 ± 27.3
Heart rate (beat per minute)[1]	87.0 ± 24.7
Creatinine (µmol/L)[1]	109.7 ± 81.6
NT-proBNP (pg/mL)[1]	9220.5 ± 11404.8
Troponin T (ng/L)[1]	2104.6 ± 2597.6
pH[1]	7.4 ± 0.1
Lactate (mmol/L)[1]	4.3 ± 4.5
EF (%)[1]	46.2 ± 12.2
Death status or severe condition at discharge[2]	42 (26.9)

[1] Data is presented as mean ± standard deviation,

[2] Data is presented as percentage

Cardiogenic shock is a continuum rather than a disease phase. The SCAI classification at the time of admission of patients with acute myocardial infarction is described in Table 2.

Table 2. SCAI classification at the time of admission in myocardial infarction patients

	SCAI A (n = 72)	**SCAI B (n = 30)**	**SCAI C (n = 48)**	**SCAI D (n = 4)**	**SCAI E (n = 2)**	**p**
Age[1]	65.6 ± 13.0	70.8 ± 10.4	74.3 ± 10.5	86.5 ± 5.6	73.5 ± 2.1	0.12
Female sex[2]	15 (20.8)	13 (43.3)	18 (37.5)	3 (75.0)	0 (0.0)	0.03
Hypertension	43 (59.7)	19 (63.3)	33 (68.8)	3 (75.0)	1 (50.0)	0.84

	SCAI A (n = 72)	SCAI B (n = 30)	SCAI C (n = 48)	SCAI D (n = 4)	SCAI E (n = 2)	p
Diabetes mellitus[2]	10 (13.9)	12 (40.0)	13 (27.1)	2 (50.0)	1 (50.0)	0.03
Prior myocardial infarction[2]	10 (13.9)	4 (13.3)	6 (12.5)	1 (25.0)	1 (50.0)	0.62
Prior stroke[2]	5 (6.9)	2 (6.7)	3 (6.3)	0 (0.0)	1 (50.0)	0.20
Chronic kidney disease[2]	0 (0.0)	1 (3.3)	3 (6.3)	0 (0.0)	0 (0.0)	0.32
Smoking[2]	3 (4.2)	3 (1.0)	6 (12.5)	1 (25.0)	0 (0.0)	0.36
Systolic blood pressure[2]	133.2 ± 19.7	122.7 ± 20.2	99.9 ± 14.9	88.8 ± 20.2	0 ± 0	0.20
Heart rate[1]	80.0 ± 14.8	89.5 ± 22.9	97.8 ± 28.4	98.3 ± 45.1	20.0 ± 28.3	0.00
Creatinine (µmol/L)[1]	85.3 ± 24.9	101.0 ± 54.8	129.3 ± 53.2	130.5 ± 24.2	581.0 ± 496.4	0.00
NT-proBNP (pg/mL)[1]	1146.2 ± 1120.1	7302.4 ± 10320.8	14559.8 ± 12024.3	11919.8 ± 15813.7	22065.5 ± 12633.9	0.00
Troponin T (ng/L)[1]	1297.9 ± 1909.9	1702.8 ± 2028.8	3589.8 ± 3274.9	1771.3 ± 671.7	2193.0 ± 2003.9	0.00
pH[1]	7.4 ± 0.4	7.4 ± 0.5	7.4 ± 0.1	7.2 ± 0.1	7.3 ± 0.7	0.00
Lactate (mmol/L)[1]	1.2 ± 0.5	2.2 ± 1.3	4.1 ± 2.3	14.3 ± 3.3	21.0 ± 1.4	0.00
EF (%)[1]	53.4 ± 9.1	41.9 ± 11.2	38.6 ± 9.3	32.0 ± 9.4	49.5 ± 34.7	0.047
Death or severe status at discharge[2]	0 (0.0)	7 (23.3)	29 (60.4)	4 (100)	2 (100)	0.00

[1]Data is presented as mean ± standard deviation, [2]Data is presented as percentage

The transition in SCAI classification at 24 hours from admission in relation to clinical outcomes is presented in Tables 3 and 1

Table 3. Relationship between the SCAI classification transition and clinical outcomes

	Non death and severe condition at discharge	Death or severe condition at discharge	p
Better SCAI stage	21 (18.4)	1 (2.4)	0,000
Unchanged SCAI stage	89 (78.1)	5 (11.9)	
Worse SCAI stage	4 (3.5)	36 (85.7)	

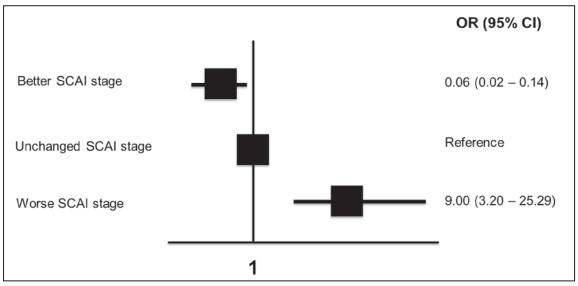

Figure 1. The association between SCAI classification transition and clinical outcomes

DISCUSSION

In this study of acute myocardial infarction, we stratified patients into 5 SCAI shock stages at the time of admission, reflecting a continuum of increasing shock severity using a simplified definition based on hypotension or tachycardia, hypo-perfusion, deterioration and refractory shock, which can be easily applied in clinical practice. The proportion of patients with SCAI shock stages A through E were 46.2%, 19.2%, 30.8%, 2.5% and 1.3%, respectively.

Baseline demographics, medical histories, comorbidities, laboratory parameters were presented detailly in Table 1. The average age of patients was 68.9 ± 12.4 years old. Most patients were male (68.6%). Hypertension, diabetes, and smoking are important risk factors for myocardial infarction. The rates of hypertension, diabetes and smoking in our study were 63.5%, 24.4% and 8.4%, respectively. The age of the patients and the proportion of female in our study were statistically significant higher than these characteristics in studies of Shrage and Hanson.[21,22] Population in Shrage's study were cardiogenic shock due to acute myocardial infarction complication, non-ischemic diseases and prior cardiopulmonary resuscitation. Patients in Hanson's study were acute myocardial infarction which was similar to ours.[22] The mean systolic blood pressure in our study was 118.1 ± 27.3 mmHg, which was statistically significantly higher than this parameter in Shrage's study, which was 113.7 mmHg ($p<0.05$). The average heart rate in our study was 87.0 ± 24.7 beats per minute, and was similar to Shrage's study, which was 87.7 beats per minute.[21] The rate of patients who were in death status or severe condition at discharge in our study was 26.9%.

Baseline characteristics per SCAI stage were shown in Table 2. We found no differences in mean age, history of smoking, history of diabetes, history of stroke, chronic kidney disease across different SCAI stages. The study with a population of more than 1000 patients by Schrage showed that there was a statistically significant difference in these above research parameters, but it was not clear to demonstrate the trend of increase or decrease of the parameters in different SCAI stages.[21] Proportion of women and clinical parameters at the time of admission such as heart rate, creatinine concentration, NT-proBNP concentration, troponin T concentration, pH, blood lactate concentration and left ventricular ejection fraction (EF) were statistically significant difference across SCAI stages. Heart rate tends to increase gradually according to the severity of cardiogenic shock. Table 2 showed the heart rate was lowest in SCAI A and highest in SCAI D but then decreased deeply in SCAI E. Concentration of creatinine was found to increase gradually as the grade of shock progressed. Similar results were observed when analyzing the concentration of NT-proBNP, troponin T, blood lactate. These results were similar to those in Schrage's study (2019) or Thayer's study (2020).[21,23] The development of cardiogenic shock is often directly related to the degree of myocardial damage. Progressive cardiogenic shock due to the ventricle can not pump effectively, leading to a decrease in stroke volume and cardiac output causing systemic hypo-perfusion. On the other hand, sympathetic

nervous system and systemic inflammation may play a role limiting the peripheral vascular compensatory response and may contribute to making the situation worse.[24,25,26] In light of the complex pathophysiology of cardiogenic shock, it is not surprising that heart rate, cardiac biomarkers, creatinine concentration, pH, blood lactate concentration will get worse as cardiogenic shock progresses.

The rates of clinical outcomes including death and serious status at discharge of patients with acute myocardial infarction increased as cardiogenic shock progressively worsened (Figure 2). The clinical outcomes rate from classic shock to critical stage in our study was 64.8% (35/54 patients). In which, the highest mortality rate was in SCAI D and SCAI E. With longer follow-up, the higher the SCAI classification, the worse outcomes within 30 days, 180 day, 1 year and 5 years after discharge.[21,27–29]

The transition of SCAI classification and clinical outcomes at the hospital

The SCAI classification was re-evaluated 24 hours after the patient was admitted to the hospital. The results show that at 24 hours, the proportion of SCAI grades A, B, C, D and E is 55.1%, 12.2%, 7.1%, 16.6% and 9.0%, respectively. For patients initially assessed, 14.1% improved SCAI stage, 60.3% remained at the SCAI stage and 25.6% worsened SCAI stage. We found a statistically significant difference in clinical outcomes when monitoring the progression of cardiogenic shock according to SCAI classification (Table 3). The clinical outcomes rate was lowest in the improved SCAI group (2.4%), higher than in the group with the same SCAI classification (11.9%) and highest in the SCAI group with progressive deterioration (85.7%). On the other hand, the OR correlation coefficient analyzing was proven that there is a very close correlation between the transition of the SCAI stage and clinical outcomes at the hospital. Patients with better SCAI stage at 24 hours have better prognosis, whereas patients with a worsening SCAI stage will have a worse prognosis for the outcome. When cardiogenic shock patients were followed longer, the difference still showed the same results. Baran and colleagues examined 166 patients with acute myocardial infarction and showed that the 24 hours reassessment of shock stage predicted 60-day mortality and 180-day mortality after discharge.[29]

Our study demonstrated that a down-grade SCAI stage at 24 hours after admission will predict better clinical outcomes. While the mortality rate of acute myocardial infarction with cardiogenic shock improved difficultly by current strategies, early assessment and early treatment to avoid severe prognosis may improve prognosis. Further trials will be conducted to prove this issue.

Limitations

Firstly, this is a prospective series of a relatively small number of patients in a single center. Secondly, we do not have long-term follow up is a limitation. In addition, invasive pulmonary artery catheter was not measured and laboratory parameters such as lactate and biomarkers were not taken in all patients.

CONCLUSIONS

Assessment of the SCAI shock stage on admission and 24 hours after admission on 156 patients with acute myocardial infarction resulted in:

There was a statistically significant difference in clinical and laboratory parameters such as female proportion, heart rate, creatinine concentration, NT-proBNP concentration, troponin T concentration, pH and blood lactate in different SCAI stages.

Clinical outcomes including death and severe condition at discharge increased as SCAI stage progressed worse.

Re-assessment of the SCAI stage at 24 hours predicted clinical outcomes.

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