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In-hospital mortality of cardiogenic shock complicating ST-elevation myocardial infarction in Malaysia: a retrospective analysis of the Malaysian National Cardiovascular Database (NCVD) registry

Padmaa Venkatason,[1] Yong Z Zubairi,[2] Wan Azman Wan Ahmad,[1] Muhammad Imran Hafidz,[1] Muhammad Dzafir Ismail,[1] Mohd Firdaus Hadi,[1] Ahmad Syadi Mahmood Zuhdi[1]

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[1]Department of Medicine, University of Malaya Medical Centre, Kuala Lumpur, Malaysia
[2]Foundation Studies in Science, University of Malaya, Kuala Lumpur, Malaysia

Correspondence to
Dr Ahmad Syadi Mahmood Zuhdi;
syadizuhdi@yahoo.co.uk

ABSTRACT

Objectives Cardiogenic shock (CS) complicating ST-elevation myocardial infarction (STEMI) carries an extremely high mortality. The clinical pattern of this life threatening complication has never been described in Malaysian setting. This study is to investigate the incidence, clinical characteristics and outcome of STEMI patients with CS in our population.

Design A retrospective analysis of STEMI patients from 18 hospitals across Malaysia contributing to the Malaysian National Cardiovascular Database-acute coronary syndrome) registry (NCVD-ACS) year 2006–2013.

Participants 16 517 patients diagnosed of STEMI from 18 hospitals in Malaysia from the year 2006 to 2013.

Primary outcome measures In-hospital and 30 day post-discharge mortality.

Results CS complicates 10.6% of all STEMIs in this study. They had unfavourable premorbid conditions and poor outcomes. The in-hospital mortality rate was 34.1% which translates into a 7.14 times mortality risk increment compared with STEMI without CS. Intravenous thrombolysis remained as the main urgent reperfusion modality. Percutaneous coronary interventions (PCI) in CS conferred a 40% risk reduction over non-invasive therapy but were only done in 33.6% of cases. Age over 65, diabetes mellitus, hypertension, chronic lung and kidney disease conferred higher risk of mortality.

Conclusion Mortality rates of CS complicating STEMI in Malaysia are high. In-hospital PCI confers a 40% mortality risk reduction but the rate of PCI among our patients with CS complicating STEMI is still low. Efforts are being made to increase access to invasive therapy for these patients.

Strengths and limitations of this study

- To our knowledge, this is the first study to describe the outcome of cardiogenic shock complicating STEMI in Malaysia.
- The analysis was done on a large data consisting 16 517 patients from 18 hospitals across Malaysia. Hence, it is so far the most representative of Malaysian population in general.
- Patients were from multi-racial background representing the major racial groups in Asia, that is, Chinese, Indian and Malay.
- Confounding factors and inter-centre variations in terms of treatment and outcome from this retrospective study cannot be eliminated.
- This study focuses on in-hospital mortality only. The long-term outcome was not analysed due to insufficient follow-up data.

INTRODUCTION

Cardiogenic shock (CS) is an important cause of death in acute ST-elevation myocardial infarction (STEMI).[1–3] Left ventricular dysfunction is the most common underlying aetiology in CS accounting for about 74.5% of cases.[4 5] There is correlation with the severity of coronary artery disease whereby CS is strongly associated with triple vessel or left main stem coronary involvement[6]

Despite the advancement in reperfusion therapy with invasive percutaneous coronary intervention (PCI), the mortality rate remains high. The in-hospital mortality rate even after successful PCI is reported to be as high as 40%.[7–9] Although the incidence of CS complicating myocardial infarctions (MIs) is only around 4%–10%,[1 10] it remains a big challenge in terms of clinical management.

Due to various limitations locally, the rate of coronary reperfusion with primary PCI in STEMI is only about 7% in Malaysia.[11] Given the restriction in delivering the preferred

revascularisation therapy (primary PCI), the outcome of CS complicating MIs in our population has yet been fully described and no comparison ever made with other studies. Hence, we use data from the Malaysian National Cardiovascular Database-acute coronary syndrome 2006–2013 (NCVD-ACS 2006–2013) to investigate the characteristics and outcome of CS complicating STEMIs in Malaysia.

METHODS

Patient population

A total of 16 517 patients diagnosed with STEMI were identified from the Malaysian NCVD-ACS from year 2006 to 2013. The NCVD is a national registry involving 18 hospitals nationally. It captures clinical data on all patients admitted with acute coronary syndromes. The Ministry of Health Malaysia and the National Heart Association of Malaysia (NHAM) sponsor the registry. Data are collected on admission and throughout the patient stay using a standardised case reporting form. A unique national identification number is given to each patient to avoid duplication. Parameters recorded include baseline characteristics and clinical presentation, in-hospital treatment, procedural details and clinical outcome.

STEMI is defined as a persistent ST-segment elevation of ≥1 mm in two contiguous electrocardiographic leads or the presence of a new left bundle branch block in the setting of positive cardiac markers and/or typical cardiac pain. Patients were divided into two groups based on their Killip class on presentation. Those in Killip class IV were grouped under 'CS' (n=1753) while those in Killip classes I, II and III were grouped under 'non-CS' (n=14 764). The two groups were compared in terms of clinical characteristics, in-hospital invasive treatment, pharmacotherapy and all cause in-hospital mortality. A cross-check with the national death registry was also done to verify the patients' mortality status.

The results of the study will be made public in NHAM website through the NCVD annual reports in interest for the view of the participants. In this study, we use retrospective cohort studies looking at data that have already been existing.

Definition of Killip class

Killip class IV is defined as the presence of hypotension with a systolic blood pressure (BP) lower than 90 mm Hg and evidence of peripheral vasoconstriction. Below are the definitions of the other Killip classes:

Killip I: No clinical signs of heart failure.

Killip II: Presence of rales or crepitation in the lungs bases only or a third heart sound (S3).

Killip III: Presence of frank acute pulmonary oedema.

Killip IV: CS or hypotension (measured as systolic BP <90 mm Hg), and evidence of peripheral vasoconstriction.

Statictical analysis

Categorical variables were described as numbers and percentages. The differences were analysed by χ^2 test or Fisher exact test. Continuous variables were expressed as median and differences were analysed using t-test. To avert biases in the estimates and loss of power, missing data for explanatory variables were assumed to be missing at random. A generalised linear model with a log link, binomial distribution and a robust variance estimator was used to estimate the risk ratios. The risk ratios represent the relative risk for mortality of the non-CS group compared with the CS group. Subsequently, risk ratios of CS patients with PCI done and without PCI were also compared. Variables that were statistically significantly different (a two-sided p value of less than 0.05) between the CS and non-CS patients, that were of clinical importance, and that had sufficient outcomes in the respective subcategories were adjusted for. Finally, binary logistics regression was executed to determine the independent predictors of in-hospital mortality among CS patients.

Table 1 Baseline characteristics of patients with ST-elevation myocardial infarction

	CS (Killip IV) (n=1753)	Non-CS (Killip I–III) (n=14 764)	P value
Age			
64 years or less	1214 (71.4%)	11 141 (77.4%)	<0.001
>65 years	486 (28.6%)	3252 (22.6%)	
Gender			
Male	1455 (83.0%)	12 687 (85.9%)	0.001
Female	298 (17.0%)	2077 (14.1%)	
Ethnicity			
Malay	1113 (63.5%)	8631 (58.5%)	0.001
Chinese	285 (16.3%)	2632 (17.8%)	
Indian	247 (14.1%)	2466 (16.7%)	
Others	108 (6.2%)	1035 (7.0%)	
Risk factors			
Smoking (active/ex)	1109 (67.4%)	10 020 (70.0%)	0.028
Diabetes	732 (51.3%)	5257 (42.3%)	<0.001
Hypertension	891 (61.3%)	7270 (57.2%)	0.002
Hyperlipidaemia	372 (32.1%)	3754 (35.3%)	0.030
Family history	158 (9.0%)	1658 (11.2%)	<0.001
Premorbids			
Cerebrovascular	49 (3.4%)	386 (3.1%)	0.422
Previous MI	208 (15.1%)	1553 (12.6%)	0.009
Peripheral vascular disease	10 (0.7%)	35 (0.3%)	0.007
Chronic kidney disease	100 (7.1%)	461 (3.7%)	<0.001
Chronic lung disease	58 (4.1%)	285 (2.3%)	<0.001
Myocardial infarct type			
Inferior infarct	732 (41.8%)	5310 (36.0%)	<0.001
Anterior infarct	743 (42.4%)	6772 (45.9%)	0.001
LVEF mean±SD	38.7±12.2	46.1±11.1	0.025

CS, cardiogenic shock; LVEF, left ventricular ejection fraction; MI, myocardial infarction; non-CS, non-cardiogenic shock.

Table 2 Coronary reperfusion and revascularisation therapy in STEMI patients who have CS and do not have CS

	CS STEMI	Non-CS STEMI	P value
Thrombolysis			
Given	1216 (71.4%)	10885 (75.2%)	<0.001
Not given—proceeded to primary angioplasty	199 (11.7%)	1451 (10.0%)	
Not given—missed	129 (7.6%)	1690 (11.7%)	
Not given—patient refusal	4 (0.2%)	49 (0.3%)	
Not given—contraindicated	156 (9.2%)	391 (2.7%)	
In-hospital PCI*	537 (33.6%)	4083 (29.5%)	0.001
Door to needle time for thrombolysis (min)	45.0	60.0	<0.001
Symptom to door time (min)	249.98+/−224.74	239.34+/−215.37	0.074

*PCI done during index admission that was not primary angioplasty—includes rescue PCI, pharmacoinvasive PCI and early routine PCI.
CS, cardiogenic shock; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction.

All analyses were conducted using SPSS V.21 statistical software.

Patient and public involvement

There is no patient or public involvement in the development of this study's research question and outcome. All data were obtained retrospectively from the Malaysian NCVD-ACS.

RESULTS

Table 1 illustrates the comparison in baseline characteristics between the CS and non-CS group. A total of 1753 out of 16517 patients (10.6%) presented with CS. Demographically, the CS group contained more patients over the age of 65 (28.6% vs 22.6% p<0.001). Females and Malay ethnic groups were also seen to be significantly more prevalent in the CS group. In terms of cardiovascular risk factors, they had higher rate of diabetes and hypertension but unexpectedly lower rate of smoking, hyperlipidaemia and premature family history. Other related premorbid conditions were unfavourable to the CS group where they had higher rate of previous MI, cerebrovascular, peripheral vascular, chronic kidney and chronic lung diseases.

Table 3 In-hospital pharmacotherapy

Medications	CS STEMI (n=1753)	Non-CS STEMI (n=14764)	P value
Aspirin	1024 (75.7%)	12470 (93.3%)	<0.001
ADP-antagonist	632 (67.8%)	8346 (81.4%)	<0.001
ACE-I/ARB	529 (30.3%)	8128 (55.8%)	<0.001
Beta blocker	659 (51.1%)	9185 (71.5%)	<0.001
Statin	957 (70.9%)	12024 (90.5%)	<0.001

ACE-I, ACE converting enzyme inhibitor; ADP, ADP diphosphate; ARB, angiotensin receptor blocker; CS, cardiogenic shock; STEMI, ST-elevation myocardial infarction.

Table 2 compares the revascularisation treatment between the two groups. Intravenous thrombolysis remained the main emergency reperfusion therapy for both CS and non-CS patients. Although there was no significant difference of symptom to door times between the two groups, the door to needle time was significantly shorter for CS patients (45 min vs 60 min p<0.001). The difference in the rate of primary PCIs between the two groups was small (11.7% CS vs 10.0% non-CS). Total rate of in-hospital PCIs (inclusive of primary PCIs) was however significantly higher in CS patients (33.6% vs 29.5% p=0.001). Table 3 shows the administrative rate of evidence-based pharmacotherapy during the admission, which favoured the non-CS patients across all class of medications especially antihypertensives.

Table 4 compares the all cause in-hospital mortality rate between patients with CS and non-CS. The mortality rate was different between the two groups (34.1% CS vs 5.6% non-CS, p value <0.001) After multivariate adjustment of confounding factors, we found that the CS group had 7.14 times higher mortality risk compared with the non-CS group.

Mortality data were obtained from official records from the National Registration Department of Malaysia and cross-referenced to patients, however we were unable to get information for 29 patients (0.017%) in the CS group for undetermined reasons. Table 5 shows the sub-analyses of in-hospital mortality rates among CS patients. Those who had PCI done during the admission had a lower rate of in-hospital mortality (27.0% vs 38.9%) compared with those who did not. Adjusted mortality risk ratio showed that there was a 40% mortality risk reduction in those with PCI done.

Table 6 shows univariate analysis of clinical variables related to mortality in the CS group. All variables that were statistically significant from this table were then grouped into a multivariate logistic regression to determine the independent predictors of in-hospital mortality within the CS group. The result of the multivariate logistic regression is tabulated in Table 7. We found that the presence of

Table 4 In-hospital and 30 day mortality rates

	No of patients	Death (%)	Unadjusted risk ratio	Adjusted risk ratio	P value
In-hospital mortality					
CS	1753	598 (34.1)	6.827 (6.104, 7.954)	7.143 (6.365, 8.017)	<0.001
Non-CS	14764	821 (5.6)	1	1	
30-Day mortality					
CS	1753	634 (36.2)	7.587 (7.002, 9.552)	8.863 (7.848, 10.009)	<0.001
Non-CS	14764	1085 (7.3)	1	1	

CS, cardiogenic shock.

Table 5 Comparison of mortality rates between cardiogenic shock with or without PCI

In-hospital mortality	No of patients	Death (%)	Unadjusted risk ratio	Adjusted risk ratio	P value
PCI done	537	145 (27)	0.535 (0.428, 0.670)	0.600 (0.513,0.700)	<0.001
PCI not done	1063	414 (38.9)	1	1	

PCI, percutaneous coronary intervention.

hypertension, diabetes mellitus, chronic lung and kidney diseases, and age of over 65 carried statistically significantly higher mortality risks and hence they seem to be independent predictors of in-hospital mortality. Table 8 shows the length of stay between the two groups. Patients with CS have significantly longer duration of inpatient stay compared with non-CS.

DISCUSSION

CS is a clinical state where cardiac dysfunction results in inadequate tissue perfusion. CS is characterised by a state of haemodynamic insufficiency that may involve hypotension (systolic blood pressure <90 mm Hg), significant decrease in mean arterial pressure from baseline and reduced cardiac index. CS can be multifactorial but most commonly occurs secondary to MI.

CS complicating an MI more commonly occurs in ST elevation myocardial infarctions (STEMIs) compared with non-STEMIs and is a predictor of poor prognosis. Data from our NCVD registry showed in-hospital mortality rates of 34.1%. This figure is lower than other MI registries and trials such as the SHOCK trial, which reported in hospital mortality rates of at least 48%. Reasons for the lower figures are unclear, but may be contributed to by a common practice of early hospital discharging of STEMI patients, which may not capture data on patients who

Table 6 Comparison of clinical factors between survivors and non-survivors of cardiogenic shock

	Survivors (n=1126)	Non-survivors (n=598)	P value
Age >65 years	226 (20.8%)	253 (43.1%)	<0.001
Diabetes	429 (47.5%)	295 (58.2%)	<0.001
Hypertension	520 (56.5%)	361 (70.4%)	<0.001
Smoking status			
Active/ex-smokers	607 (67.0%)	219 (48.6%)	<0.001
Non-smokers	299 (33.0%)	232 (51.4%)	
Dyslipidaemia	224 (30.3%)	143 (35.3%)	0.083
Previous MI	126 (14.0%)	82 (17.8%)	0.061
Chronic lung disease	25 (2.7%)	32 (6.6%)	0.001
Cerebrovascular disease	27 (2.9%)	21 (4.4%)	0.161
Peripheral vascular disease	8 (0.9%)	2 (0.4%)	0.337
Chronic renal disease	46 (5.0%)	54 (11.2%)	<0.001

MI, myocardial infarction.

Table 7 Logistic regression of predictors for in-hospital mortality in cardiogenic shock

	P value	Risk ratios	95% CI for EXP(B) Lower	Upper
Age >65	**0.000**	**2.470***	**2.073**	**2.944**
Dyslipidaemia	0.040	0.828	0.691	0.992
Hypertension	**0.000**	**1.427***	**1.180**	**1.726**
Diabetes mellitus	**0.000**	**1.600***	**1.343**	**1.907**
Smoking status	0.000	0.675	0.567	0.804
Previous MI	0.175	1.177	0.930	1.490
Chronic lung disease	**0.032**	**1.744***	**1.048**	**2.903**
Chronic renal disease	**0.000**	**2.853***	**2.079**	**3.915**
Cerebrovascular disease	0.922	1.023	0.648	1.615
Peripheral vascular disease	0.256	0.410	0.088	1.909
Constant	0.000	0.052		

The bold fonts indicate the variables that predict in hospital mortality in cardiogenic shock patients.
*Statistically significant predictors of mortality.
MI, myocardial infarction.

Table 8 Length of stay of cardiogenic shock (CS) versus non-CS patients

Total day stay	Cardiogenic shock	Non-cardiogenic shock	P value
Mean	8.17 (7.53, 8.82)	5.21 (5.12, 5.29)	0.014
SD	11.561	5.102	

died at home early after discharge that would be reflected in 30-day outcomes if these data were available.

Preexisting conditions including hypertension, diabetes mellitus, chronic kidney and lung disease conferred a higher risk of death in our patients, which may reflect poor pre-hospital reserve that is ill prepared to cope with a major stressor such as CS. Increasing age was also a predictor of mortality in our cohort with adults over 65 years of age more than twice more likely to die in hospital if they had CS complicating a STEMI. Age was also found in another study to be the parameter most strongly associated with developing CS after an MI with every 10-year increase in age the risk of developing shock was greater by 47%.[12] We observed an interesting finding of significantly lower rates of smoking, family history and dyslipidaemia in the CS group. It is not clear whether this represents under-reporting or under-diagnosis of risk factors or these are paradoxical risk factors for developing CS in STEMI in our population. Nonetheless, further studies would be appropriate to investigate this further, perhaps with future data from NCVD.

Data show that CS patients in the setting of acute MI who were treated non-invasively had poorer outcome and primary PCI is superior to thrombolytic therapy.[12–16] Similar to other registries and studies, our data showed improved survival for patients who underwent in-hospital PCI including primary PCI.[12] The adjusted risk of death was reduced by 40% for patients who received PCI during the index admission compared with those who did not. Intravenous thrombolysis remains the most frequent mode of achieving reperfusion in Malaysia due to several factors. PCI in Malaysia is more costly than thrombolysis and primary or urgent PCI services are limited to patients presenting to one of several PCI centres or their network hospitals, which explains why around only 10% of patients received primary PCI. Nonetheless, in the SHOCK trial, thrombolysis was superior to medical therapy only and is recommended in many guidelines as a reperfusion strategy when PCI is not possible or delayed, particularly when patients present within 3 hours of symptoms.[17] We did not have any data on intra-aortic balloon pump or assist devices in our patients in this registry. Our data showed a shorter door to needle time in patients presenting with CS compared with non-CS. We postulate several factors—CS patients would be appear more ill during initial assessment and the presence of hypotension would likely push for more urgent and swift diagnostic and management steps. In our personal experience, patients with non-CS STEMI may also present in atypical ways that may delay or make assessment less urgent, hence explain the longer door to needle time. Ideally, we would have included the door to balloon data for comparison, however that data are contained in a separate registry called the NCVD-PCI registry, which we did not have access to.

Efforts are being made to increase coverage of primary PCI through the development of a hub and spoke model for STEMI s called the MySTEMI Network. Non-PCI centres (hub) are paired with a PCI capable centre (spoke) whereby patients presenting to a non-PCI hospital with a STEMI are transferred to a PCI centre for primary PCI.[18] We hope that with the rolling out of this MySTEMI Network nationally, we are able to offer PCI as the main reperfusion modality for STEMI patients. Efforts are also being made to improve prescribing rates of evidence-based therapy through clinical audits and CME sessions. There was a low rate of antiplatelet prescription particularly in the CS group, which has been noted in other local studies.[11 19] Although the exact reasons to explain the low prescription rates in our population were not detailed in the NCVD registry, one factor could be the increased bleeding rates in patients with CS.[20] We recognise that although our findings are based on the NCVD data, these may not be truly representative of the current situation. The current NCVD is incomplete as there are still several hospitals that are not yet fully contributing towards NCVD data; efforts are however being taken to improve this. Increased reporting will only improve the accuracy of future studies and allow better allocation of resources in improving outcomes.

CONCLUSION

CS complicated STEMI in about 10.6% of our patients. The in-hospital mortality was high (34.1%) and invasive coronary revascularisation lowered the mortality rate substantially. Similar to other studies, multiple comorbidities including increased age were predictors of poor prognosis. Greater effort is needed to improve outcomes and increased effort is being made to improve the rate of primary and in-hospital PCI.

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Contributors Original idea of the study originated from ASMZ. PV, YZZ and ASMZ designed the study. WAWA, MIH, MFH and MDI wrote the initial draft of the manuscript. PV, MIH, MFH, YZZ, WAWA and MDI analysed the data and revised the manuscript. All authors interpreted the data analysis and scrutinised the final manuscript.

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