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ORIGINAL ARTICLE

Clinical and Angiographic Predictors of Mortality in Sudden Cardiac Arrest Patients Having Cardiac Catheterisation: A Single Centre Registry

James Xu, MBBS [a,b], Leia Hee, PhD [a,b], Andrew Hopkins, MBBS [a,b], Craig P. Juergens, MD [a,b], Sidney Lo, MBBS [a,b], John K. French, PhD [a,b], Christian J. Mussap, PhD [a,b*]

[a]Cardiology Department, Liverpool Hospital, Sydney, NSW, Australia
[b]South Western Sydney Clinical School, The University of NSW, Sydney, NSW, Australia

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Background Immediate cardiac catheterisation (CC) is recommended in ST-elevation myocardial infarction (STEMI) following sudden cardiac arrest (SCA). Guidelines advise urgent CC for SCA patients without-STEMI, at clinician discretion. We examined the clinical and angiographic factors predicting mortality in SCA patients having CC.

Methods Consecutive SCA patients having CC at Liverpool Hospital, Sydney (January 2011–September 2015) were retrospectively analysed. Patient data were retrieved from hospital records, and angiographic SYNTAX scores (SS) were quantified online. Independent predictors of mortality were derived using multivariate logistic analysis.

Results The study cohort comprised 104 SCA patients; mean age 61 $\pm$ 12 years, and 79% male. Immediate CC (<2 hours post-SCA) was performed in 35% overall. Compared to the without-STEMI subgroup, STEMI patients had more ventricular fibrillation (91 vs 50%; $p < 0.0001$), and higher mean peak serum high-sensitivity troponin-T (8.25 $\pm$ 14.7 vs 1.97 $\pm$ 6.13 ug/L; $p = 0.006$); in the context of higher median SS (18 vs 6.5; $p = 0.002$) and target-lesion SS (tSS, 10 vs 0; $p < 0.001$). Percutaneous coronary intervention (PCI; 75 vs 23%; $p < 0.0001$) and target vessel revascularisation (11 vs 0%; $p = 0.005$) were more frequent for STEMI. All-cause mortality was 39%, at 1.3 $\pm$ 1.5 years follow-up. Independent mortality predictors were: delayed CC (HR 4.08), serum lactate >7 mmol/L (HR 3.47), and tSS (HR 1.05).

Conclusions Elevated serum lactate, tSS, and delayed CC, were predictive of longer-term mortality in SCA patients having CC. Late CC in patients without-STEMI suggest scope for improvement in real-world systems of care. Closer scrutiny of target lesion complexity may aid prognostication in SCA survivors.

Keywords PCI: Percutaneous coronary intervention • SCA: Sudden cardiac arrest • STEMI: ST-elevation myocardial infarction

*Corresponding author at: Cardiology Department, Liverpool Hospital, Liverpool, NSW 2170, Sydney, Australia. Tel.: +612 9828 3073, Fax: +612 9828 3054, Email: Christian.Mussap@health.nsw.gov.au

Introduction

Sudden cardiac arrest (SCA) is a devastating and often unheralded event, associated with high morbidity and mortality [1]. Even for the relatively few patients who achieve return of spontaneous circulation (ROSC), and survive to hospital admission, the mortality rates are in the range of 50% to 62% [2,3]. In developed countries, SCA is commonly a manifestation of coronary artery disease (CAD), typically presenting as an acute coronary syndrome (ACS) with ST-elevation myocardial infarction (STEMI) [4]. However, non-ischaemic pathologies, such as structural heart conditions and ion channel derangements, may precipitate a SCA event. Lately, there have been advances in therapeutic pathways of care for SCA patients, resulting in modest improvements in survival and neurologic outcomes [5].

The recognition that SCA may be associated with CAD has led to recent guideline recommendations for expedited cardiac catheterisation (CC). Indeed, several registry studies have suggested that urgent CC, and percutaneous coronary revascularisation (PCI), may improve hospital survival [6,7]. Therefore, the standard practice in many centres is to activate the cardiac catheterisation laboratory for most SCA presentations. Immediate referral for CC seems valid in the setting of STEMI [4,8,9]. However, it is recognised that the majority of patients successfully resuscitated from SCA do not have STEMI [2,7,10–13]. Observational data on clinical outcomes have been conflicting for patients having urgent CC based solely on the electrocardiograph (ECG) findings [6,11,14]. Moreover, there are no randomised trials investigating the efficacy of CC for SCA patients, and no specific recommendations on timing of CC for SCA with undifferentiated ECG abnormalities.

At our tertiary-referral centre, SCA patients presenting with STEMI have an immediate CC, with follow-on PCI to the culprit coronary lesion. Sudden cardiac arrest survivors without STEMI are referred for CC, at the discretion of the treating physician. The primary aim of this study was to examine the impact of demographic, clinical, and angiographic factors on all-cause mortality for SCA patients having CC at our facility. In particular, we investigated independent predictors of longer-term mortality in our cohort of SCA survivors.

Methods

Patient Population

We retrospectively screened all patients who presented following an SCA event, and were treated at Liverpool Hospital, Sydney from January 2011 to September 2015. We included in the study cohort all consecutive SCA patients who had CC (n = 104). Patients were sub-grouped according to the initial ECG: STEMI vs without-STEMI. We identified patients having immediate CC (<2 hours), urgent CC (2–6 hours), delayed CC (6–24 hours) and late (>24 hours) after SCA. The present study was approved by the local institutional ethics committee *(LNR/16/LPPP/611)*.

Resuscitation of SCA, Coronary Angiography, and PCI

All SCA patients were attended by critical care first-responders, and treated according to established advanced cardiac life support guidelines [15]. Liverpool Hospital is a tertiary referral facility with round-the-clock CC and PCI capability. All ECG deemed to fulfill STEMI criteria were immediately transmitted via smartphone technology to an on-call, experienced interventional cardiologist, who determined whether to refer for CC. Electrocardiographs with ST-segment deviations >2 mm (>0.2 mV) in two contiguous precordial leads, or >1 mm in two contiguous limb leads, or >1.5 mm in lead aVR, were considered significant for STEMI according to current guidelines [16,17]. Sgarbossa's criteria [18] were applied in cases of left bundle branch block. In the acute setting, a critical care team was onsite to support patient ventilatory and circulatory functions. Unless contraindicated, all patients were administered loading doses of aspirin (300 mg), in combination with either clopidogrel (300 mg), ticagrelor (180 mg), or prasugrel (60 mg). Dual-antiplatelet therapy was recommended for minimum 12 months in STEMI, and post-PCI. An interventional cardiologist performed CC (and PCI) according to established principles, and using standard techniques [19]. Arterial access was via the radial or femoral artery approach using 6-7 French introducer sheaths and catheters. Coronary artery flow was semi-quantified using the Thrombolysis in Myocardial Infarction (TIMI) classification [20]. Coronary artery stenoses were considered significant if ≥50% luminal narrowing by angiography [4,6,13]. Follow-on PCI with adjunctive use of aspiration thrombectomy, optical coherence tomography (OCT), intravascular ultrasound (IVUS), and circulatory support devices, were at operator discretion. Patients were recovered to a co-located critical care ward and managed according to standardised post-resuscitation pathways of care [21], including therapeutic hypothermia (32–34 °C) for minimum 24 hours. Left ventricular (LV) systolic function was quantified using two dimensional (2D) echocardiography, performed at the bedside by experience sonographers.

Syntax Score Calculation

The Syntax Score (SS) algorithm (www.syntaxscore.com) was used to semi-quantify the burden and complexity of coronary artery disease [22]. In brief, the total SS was the summated score from all coronary artery lesions with ≥50% stenosis in a vessel ≥1.5 mm diameter on angiography. The target-lesion SS (tSS) was measured from the coronary artery lesion targeted for PCI, and the residual SS (rSS) was calculated as that remaining after completion of PCI [23,24]. Two experienced interventional cardiologists, working independently and blinded to clinical outcomes, scored the archived coronary angiograms retrospectively.

Data Collection and Outcomes Evaluation

Patient demographic, clinical, procedural, and outcomes data were retrieved from electronic hospital records, and/

or by telephone follow-up from primary care physicians and/or treating cardiologist. The primary longer-term clinical outcome for the study was all-cause mortality. The secondary outcomes were in-hospital mortality, target vessel revascularisation (TVR), major actionable bleeding according to BARC2 criteria [25], and stroke. To evaluate longer-term mortality, we screened cases between January 2011 and September 2015 within our inclusion criteria and with a cut-off census date of 13 May 2016.

Statistical Analysis

The study population was stratified based on the presence or absence of STEMI on the initial ECG. Normality of distribution for continuous variables was assessed using the Shapiro-Wilk test. We compared baseline characteristics using the t-test for continuous variables and Chi-squared test or Fisher's exact test, as appropriate, for categorical variables. Summary statistics were reported as means with standard deviations (SD), or median with interquartile range (IQR), for continuous variables; categorical variables were expressed as count and percentage. Logistic regression analysis was used to construct the final model using a stringent cut-off value ($p = 0.01$) of univariate predictors. Analyses were carried out using SPSS for Windows, version 23.0 (SPSS, Inc., Armonk, NY, USA). A p value <0.05 was considered statistically significant.

Results

Demographics and Baseline Characteristics

We analysed a total of 104 patients who were successfully resuscitated (achieved ROSC) following SCA, and had CC. Demographic data and baseline characteristics for the study cohort were collected retrospectively, and are listed in Table 1. Mean age was 61 years and 79% (n = 82) were male. Patients were sub-grouped according to the initial ECG: STEMI (42%, n = 44) vs without-STEMI (58%, n = 60). Patients with STEMI were more likely to have ventricular fibrillation (VF) as the initial cardiac rhythm, compared to those with without-STEMI (91% vs 50%, $p < 0.0001$). Importantly, the rate of delay in return of spontaneous circulation (ROSC > 30 min) was similar for all sub-groups. Initial mean systolic blood pressure was lower in STEMI subgroup (108 ± 17 vs 119 ± 29 mmHg, p = 0.018). There were no group differences in peri-procedural cardiogenic shock, which was low overall (13%).

The incidence of end-stage kidney disease (ESKD) was higher for without-STEMI vs STEMI (12% vs 0; p = 0.02), although absolute numbers were low. Otherwise, there were similar rates of medical co-morbidities between STEMI and without-STEMI patients (Table 1). Mean serum pH and lactate concentration were similar between subgroups. However, the mean peak serum high-sensitivity troponin-T was greater for STEMI vs without-STEMI patients (8.25 vs 1.97 μg/L, p = 0.006). Overall, LV systolic function was preserved or mildly impaired in 33%, moderately impaired in 23%, and severely impaired in 25%, while echocardiography not performed in 18%. There was no difference in LV systolic function between subgroups (p = 0.20).

Angiographic and PCI Procedural Characteristics

There was a significant difference in door-to-balloon time (DBT) time between STEMI and without-STEMI patients (Table 2). The median DBT for STEMI was 1.7 [1–2.3], compared to 49 [2–192] hours for without-STEMI. The majority (65%) of STEMI patients had immediate CC (<2 hours of SCA), while most without-STEMI patients had late CC (>24 hours). An infarct-related artery was identified in 61% of the total cohort, with higher incidence of PCI for STEMI vs without-STEMI (89 vs 40%, $p < 0.0001$). STEMI patients were more likely to require mechanical circulatory support during CC. Higher SS and tSS were recorded for STEMI compared to without-STEMI (18 vs 6.5 and 10 vs 0, $p < 0.01$ for both). Indeed, a SS = 0 was not recorded for any STEMI patient, but was observed in 33% of without-STEMI patients. Follow-on PCI was more likely for the STEMI group (75 vs 23%; $p < 0.0001$). Overall rates of multi-vessel stenting were low (19%, Table 3), and rSS was similar for STEMI and without-STEMI (6 vs 3.5; p = 0.17). Procedural characteristics of patients who received percutaneous coronary intervention were shown in Table 3. Among the 47 patients who received PCI, drug-eluting stents (DES) were used in 45% cases. There was no difference in the use of glycoprotein-IIbIIIa (GpIIbIIIa) inhibitors, thrombus aspiration or intra-coronary imaging between the subgroups.

Outcomes and Independent Predictors for All-Cause Mortality

Mean follow-up duration was 1.3 ± 1.5 years. All-cause mortality was 39% (Table 4), with similar survival rates observed for all subgroups (Figure 1). In-hospital mortality, procedural death (occurring during CC), and bleeding rates were similar among subgroups. We observed a 5% TVR rate in our study cohort, all of which occurred in STEMI patients (p = 0.005). Independent predictors of mortality, analysed by multivariate Cox regression, were: delayed CC (HR 4.08, 95% CI 1.86–8.93, p = 0.001), serum lactate >7 mmol/L (HR 3.47, 95% CI 1.46–8.22, p = 0.005), and tSS (HR 1.05, 95% CI 1.02–1.08, p = 0.003).

Discussion

An improved strategy is required to identify SCA patients who may benefit from CC. Recent studies suggest that urgent CC may affect short and long-term clinical outcomes [6,8,9,26,27]. The primary objective of this study was to investigate the impact of clinical and angiographic factors on all-cause mortality in SCA patients having CC.

Overall, we observed high rates for both in-hospital (36%) and longer-term (39%) mortality (Table 4), although the

Table 1 Patient demographic and characteristics.

	All n = 104	STEMI n = 44	without-STEMI n = 60	p Value
Age, years	61 ± 12	59 ± 8	63 ± 14	0.06
Male	82 (79)	38 (86)	44 (73)	0.11
Out-of-hospital	58 (56)	22 (50)	36 (60)	0.31
Initial cardiac rhythm VF	70 (67)	40 (91)	30 (50)	<0.0001
Initial systolic blood pressure, mmHg	114 ± 23	108 ± 17	119 ± 26	0.018
No bystander CPR	69 (66)	28 (64)	41 (68)	0.62
Unwitnessed arrest	17 (16)	6 (14)	11 (18)	0.52
ROSC > 30 mins	34 (33)	16 (36)	18 (30)	0.49
Severe LV impairment	27 (26)	8 (24)	19 (37)	0.20
Cardiogenic shock <90 mmHg SBP	13 (13)	6 (14)	7 (12)	0.76
Medical comorbidities				
Diabetes mellitus	24 (23)	6 (14)	18 (31)	0.06
Dyslipidaemia	48 (46)	18 (43)	30 (51)	0.43
Hypertension	54 (52)	18 (43)	36 (61)	0.07
Smoker	43 (41)	20 (47)	23 (39)	0.45
Family history of CVD	10 (10)	6 (14)	4 (7)	0.21
Prior PCI	13 (13)	4 (9)	9 (15)	0.37
Prior CABG	3 (13)	8 (19)	5 (8)	0.12
ESKD	7 (7)	0	7 (12)	0.02
Biochemistry values				
Haemoglobin, g/L	139 ± 23	149 ± 19	131 ± 23	<0.0001
Lactate >7 mmol/L	46 (44)	19 (56)	27 (49)	0.53
pH <7.2	52 (50)	22 (60)	30 (55)	0.64
pH at admission	7.1 ± 0.2	7.1 ± 0.2	7.1 ± 0.2	0.78
Lactate at admission, mmol/L	7.9 ± 4.6	8.2 ± 5.0	7.7 ± 4.4	0.58
Troponin T (peak), μg/L	4.78 ± 11.2	8.25 ± 14.7	1.97 ± 6.13	0.006
Existing PPM/ICD	5 (5)	0 (0)	5 (8)	0.05
ICU length of stay, d	2 (0–7)	2 (0–8)	2 (0–6)	0.99
Hospital length of stay, d	10 (4–24)	7 (4–19)	12 (4–27)	0.16

All data presented as mean ± SD or median (IQR) for continuous variables, and number (percentage) for categorical variables.
Abbreviations: CABG, coronary artery bypass grafting; CPR, cardiopulmonary resuscitation; CVD, cardiovascular disease; ESKD, end stage kidney disease; ICD, implantable cardioverter defibrillator; PCI, percutaneous coronary Intervention; PPM, permanent pace maker; ROSC, return of spontaneous circulation; VF, ventricular fibrillation.

incidence of death was slightly less compared to other studies [28]. This may relate to the selective nature of our study cohort, with SCA survivors referred for CC at the discretion of the treating clinician. Our data attest to the overall high morbid risk inherent in SCA patients, but notably the incidence of CC-related death was reassuringly low. This suggests that expedited CC (and PCI) is safe following an SCA event. In general, STEMI portends a worse prognosis for ACS patients [29]. We expected the STEMI subgroup to have greater overall burden of CAD, which was supported by moderately high SS and tSS (18 and 10 respectively, Table 2). The high morbid risk for STEMI was coincident with greater need for peri-procedural haemodynamic support, and higher peak serum levels of troponin-T (Table 2). It is possible that the high-risk inherent for STEMI may have been offset by the greater incidence of VF amenable to rapid cardioversion [30] and higher rates of immediate or early referral for CC.

Current therapeutic guidelines categorise STEMI post-SCA as a Class I indication for immediate CC [19,31], and the goal is to undergo PCI within 90 minutes. Yet, it is unclear whether guideline recommendations for CC in SCA survivors are adhered to in real-world practice. At our tertiary referral hospital, the DBT for STEMI SCA patients was longer (at median 1.7 hours) than recommended. The majority (65%) of STEMI patients received CC within 2 hours (and 92% had CC within 6 hours). Our observations reflect real world practicalities, and limitations, in delivering timely CC and PCI to this challenging subgroup, and we speculate that delays administering immediate CC post-SCA may be widespread. Typically, SCA

Table 2 Angiographic characteristics.

	All n = 104	STEMI n = 44	without-STEMI n = 60	p Value
Door-to-balloon time:				<0.0001
Immediate, <2 hours	36 (35)	29 (65)	7 (12)	
Urgent, 2–6 hours	29 (28)	12 (27)	17 (28)	
Delayed, 6–24 hours	5 (5)	1 (3)	4 (7)	
Late, >24 hours	34 (32)	2 (5)	32 (54)	
Femoral access	94 (90)	43 (98)	51 (86)	0.05
Infarct related artery:	63 (61)	39 (89)	24 (40)	<0.0001
LAD	25 (24)	21 (48)	4 (7)	
LCx	8 (8)	3 (7)	5 (8)	
RCA	16 (15)	9 (21)	7 (12)	
LM	12 (12)	6 (14)	6 (10)	
Graft	1 (1)	0	1 (2)	
Left dominant	15 (14)	8 (18)	7 (12)	0.37
Syntax score = 0	20 (19)	0	20 (33)	<0.0001
Syntax score, SS	13 (3–30)	18 (11–38.5)	6.5 (0–23)	0.002
Target Syntax score, tSS	0 (0–12)	10 (0–17.5)	0 (0–3)	0.001
Residual Syntax score, rSS	5 (0–18.5)	6 (0–30)	3.5 (0–15)	0.17
PCI	47 (45)	33 (75)	14 (23)	<0.0001
LM PCI	5 (5)	2 (5)	3 (5)	0.9
TIMI 0-2 post angiogram	9 (9)	5 (11)	4 (7)	0.4
Treatment				
ECMO	4 (4)	4 (9)	0	0.02
IABP	30 (29)	20 (46)	10 (17)	0.001
TPM	3 (3)	1 (2)	2 (3)	0.75

All data presented as mean ± SD or for continuous variables and number (percentage) for categorical variables.
Abbreviations: CABG, coronary artery bypass grafting; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LM, left main; PCI: percutaneous coronary intervention; TIMI, Thrombolysis In Myocardial Infarction; TPM, temporary pacemakers.

Table 3 Procedural characteristics of patients who received percutaneous coronary intervention.

	All n = 47	STEMI n = 33	without-STEMI n = 14	p Value
Stent calibre, mm	2.8 ± 0.4	2.9 ± 0.4	2.7 ± 0.4	0.25
Stent length, mm	32 ± 15	31 ± 15	35 ± 17	0.45
Stent number	1.3 ± 0.9	1.4 ± 0.7	1.4 ± 1.1	0.90
DES	21 (45)	15 (48)	6 (50)	0.92
Multi-vessel stenting	9 (19)	5 (15)	4 (29)	0.29
Thrombus aspiration	8 (17)	7 (21)	1 (7)	0.24
IVUS/OCT	2 (4)	1 (3)	1 (7)	0.52
Screening time, min	21 ± 13	18 ± 9	26 ± 19	0.09
Contrast volume, ml	205 ± 79	219 ± 73	173 ± 86	0.07
GpIIbIIIa inhibitor	15 (32)	14 (67)	1 (20)	0.06

All data presented as mean ± SD or for continuous variables and number (percentage) for categorical variables.
Abbreviations: DES, drug eluting stent; IVUS, intravascular ultrasound; OCT, optical coherence tomography.

patients are clinically heterogeneous [32] and often need advanced cardiorespiratory resuscitation, involving multiple specialty teams, prior to CC. These interventions involve multidisciplinary decision-making, and are time-consuming to administer. Furthermore, clinicians have to consider co-morbidities, advanced care directives, and treatment wishes of next-of-kin, which may contribute to delayed CC. There is scope for further improvements in systems of care for SCA

Table 4 Outcomes at a follow-up of 1.3 ± 1.5 years.

	All n = 104	STEMI n = 44	without-STEMI n = 60	p Value
All-cause mortality	40 (39)	17 (39)	23 (38)	0.88
Neurology	10 (25)	5 (29)	5 (22)	
Cardiac	10 (25)	4 (24)	6 (26)	
Multi-organ failure	15 (38)	7 (41)	8 (35)	
Others	5 (13)	1 (6)	4 (17)	
In hospital mortality	37 (36)	16 (36)	21 (35)	0.89
Target vessel revascularisation	5 (5)	5 (11)	0 (0)	0.005
Bleeding	1 (1)	1 (2)	0 (0)	0.24
Stroke	0 (0)	0 (0)	0 (0)	–
Cath lab death	4 (4)	1 (2)	3 (5)	0.48

All data presented as number (percentage).

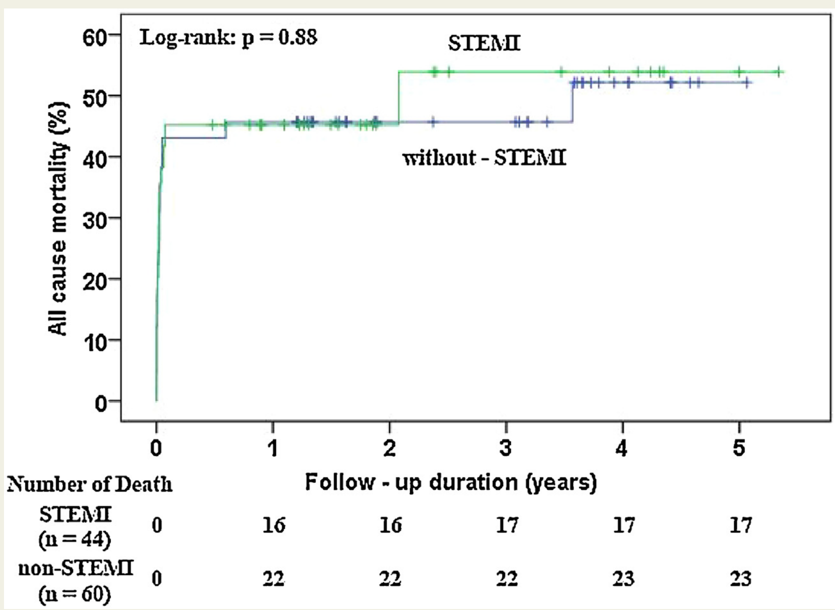

Figure 1 Kaplan Meier curve for all-cause mortality (n = 104).

survivors, including protocolling post-SCA pathways of care and more closely integrating critical care and cardiac interventional teams.

There is ongoing uncertainty regarding the role and efficacy of an early invasive strategy in SCA survivors without-STEMI. A complicating factor is that an undifferentiated post-resuscitation ECG has poor negative predictive accuracy for ACS [7,13]. Expedited CC for without-STEMI SCA survivors is supported by favourable survival and neurologic outcomes [33]. Others have proposed CC be limited to those with favourable clinical features [13]. In our study, without-STEMI patients represented two-thirds of the study cohort, yet only 11% of patients having immediate CC. Timing to CC for without-STEMI appeared to be bi-modal, with approximately half receiving immediate or early CC, and half having late CC (>24 hours). Previous angiographic studies indicate that acute coronary syndrome in the absence of STEMI does not preclude a significant coronary artery lesion, or vessel occlusion [33]. Indeed, about 40% of patients without-STEMI had a clearly identified infarct-related artery, with low-range tSS suggesting low complexity PCI. Yet and similar proportion, 33% of without-STEMI patients, scored zero for total SS, which implies negligible CAD. This reflects the protean aetiology for SCA in patients without-STEMI. Our findings show that SCA survivors without-STEMI may be particularly susceptible to delayed CC, although this appeared not to adversely affect outcomes in our non-randomised cohort. We conclude that immediate (or early) CC in without-STEMI SCA survivors is safe and should be protocoled to avoid CC delays. It should be

Table 5 Predictors for all cause mortality at follow-up duration of 1.3 ± 1.5 years.

	Univariate		p Value	Multivariate		p Value
	HR	95% CI		HR	95% CI	
Age	1.04	1.00–1.07	0.05			
Gender	1.89	1.18–5.32	0.23			
pH <7.2	2.85	1.18–6.88	0.02			
Lactate >7.0 mmol/L	5.13	2.04–12.9	0.001	3.47	1.46–8.22	0.005
Diabetes mellitus	1.57	0.62–3.97	0.34			
Smoking	0.38	0.16–0.90	0.03			
ROSC >30 min	1.42	0.62–3.28	0.41			
Shock <90 mmHg	2.85	0.86–9.45	0.09			
ESRF	1.29	0.27–6.08	0.75			
Unwitnessed arrest	3.67	1.23–10.9	0.02			
No bystander CPR	1.58	0.67–3.73	0.3			
Non-VF initially	2.46	1.06–5.69	0.04			
Out of Hospital arrest	0.95	0.43–2.11	0.9			
STEMI	1.01	0.46–2.25	0.97			
Immediate angiogram (<2 hr)	0.90	0.46–1.77	0.77			
Delayed angiogram (<24 hr)	3.73	1.92–7.27	0.001	4.08	1.86–8.93	0.001
Stent length	1.01	0.98–1.04	0.52			
Multi-vessel PCI	3.53	0.83–15.0	0.09			
SS	1.04	1.01–1.06	0.004	1.02	1.00–1.04	0.054
Target SS	1.07	1.02–1.12	0.005	1.05	1.02–1.08	0.003
Residual SS	1.02	1.00–1.05	0.09			

Abbreviations: CPR, cardiopulmonary resuscitation; ESRF, end stage renal failure; PCI, percutaneous coronary intervention; ROSC, return of spontaneous circulation; SS, Syntax Score; STEMI, ST Segment Elevation Myocardial Infarction; VF, ventricular fibrillation.

individualised based on patient co-morbidites and safety considerations. Larger cohort studies are needed to clearly define the benefit of immediate CC in SCA patients' without-STEMI.

Multivariate regression showed that elevated serum lactate (>7.1 mmol/L), delayed angiography, and tSS, were independent predictors for long-term death (Table 5). Elevated lactic acid is associated with a three-fold increased mortality [34], and greater likelihood of neurologic injury [35]. Approximately half our cohort had a significantly elevated serum lactate and a commensurate acidosis (Table 1). A recent, large observational study showed that expedited CC may improve survival [28], but patients had favourable risk factors, such as a witnessed arrest and shockable rhythm [36]. Patient selection bias is a feature of all observational studies on this topic; notably our SCA and CC cohort included a significant number without-STEMI patients. It is likely that undifferentiated, without-STEMI patients may need additional multi-disciplinary input prior to selection for CC. The inclusion of such patients, some with non-cardiac pathologies, may have contributed to the adverse risk associated with delayed CC.

This is the first study of SCA patients to include SYNTAX scoring of coronary angiograms. The SS has been validated in randomised trials, and shown to correlate with clinical and angiographic outcomes [37]. In the FREEDOM trial, a SS > 22 in the setting of ACS and diabetes was indicative of triple-vessel disease, and was best revascularised with coronary artery bypass grafting (CABG) [38]. The STEMI subgroup was characterised by moderate burden of CAD (SS = 18). None of the STEMI patients recorded a SS = 0, with most requiring follow-on PCI (75%). Thus our data concur with guideline recommendations that STEMI SCA survivors receive urgent CC. Overall DES use was low (45%), indicating a reluctance of PCI operators to commit SCA survivors to longer dual antiplatelet use when co-morbidities (and outcomes) may not be known. The tSS is a specific measure of PCI target-lesion complexity, and mean tSS of 10 for the STEMI subgroup was comparable to that reported in our earlier study of unselected patients having PCI [24]. We observed that tSS was an independent risk factor for mortality post-SCA, which suggests enhanced risk with complex PCI in this setting.

This is a single centre observational retrospective study, and we acknowledge that the study population is small. The modest sample size limits the power to detect the full array of clinical and procedural characteristics influencing outcomes in SCA. A larger study may enable analysis of subgroups, and assess the predictive value of delayed CC in patients without-STEMI. Our study is somewhat unique in that the cohort comprised all SCA survivors having CC. Thus, our observations reflect real-world experiences in the treatment of these challenging patients. Our analysis excluded SCA patients deemed ineligible for CC. This likely introduced selection bias, perhaps skewed in favour of lower-risk survivors. However, the incidence of risk factors concurs with earlier studies. Information regarding the cause of the SCA event, and the neurologic status of survivors, was not available for retrospective analysis, but we have reported the cause of death.

Conclusions

The clinical heterogeneity of SCA patients and ambiguity of the post-resuscitation ECG, may delay coronary artery revascularisation pending multi-disciplinary risk stratification. A high rate of mortality for all SCA patient subgroups selected for CC, suggests further scope for improvements in systems of care to expedite CC. Our study identified elevated lactic acid, tSS, and delayed CC as independent predictors of longer-term mortality. The association between tSS and survival suggests that closer scrutiny of PCI complexity in the setting of SCA, may aid prognostication. These findings may assist therapeutic decision-making for SCA patients, but further large-scale, randomised studies are needed.

Disclosure Statement

None.

References

[1] Nichol G, Thomas E, Callaway CW, Hedges J, Powell JL, Aufderheide TP, et al. Regional variation in out-of-hospital cardiac arrest incidence and outcome. JAMA 2008;300:1423–31.

[2] Mooney MR, Unger BT, Boland LL, Burke MN, Kebed KY, Graham KJ, et al. Therapeutic hypothermia after out-of-hospital cardiac arrest evaluation of a regional system to increase access to cooling. Circulation 2011;124:206–14.

[3] Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med 2004;30:2126–8.

[4] Spaulding CM, Joly L-M, Rosenberg A, Monchi M, Weber SN, Dhainaut J-FA, et al. Immediate coronary angiography in survivors of out-of-hospital cardiac arrest. N Engl J Med 1997;336:1629–33.

[5] Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes 2010;3:63–81.

[6] Dumas F, Cariou A, Manzo-Silberman S, Grimaldi D, Vivien B, Rosencher J, et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest insights from the PROCAT (Parisian Region Out of Hospital Cardiac ArresT) registry. Circ Cardiovasc Interv 2010;3:200–7.

[7] Dumas F, White L, Stubbs BA, Cariou A, Rea TD. Long-term prognosis following resuscitation from out of hospital cardiac arrest: role of percutaneous coronary intervention and therapeutic hypothermia. J Am Coll Cardiol 2012;60:21–7.

[8] Bendz B, Eritsland J, Nakstad A, Brekke M, Kløw N, Steen P, et al. Long-term prognosis after out-of-hospital cardiac arrest and primary percutaneous coronary intervention. Resuscitation 2004;63:49–53.

[9] Garot P, Lefevre T, Eltchaninoff H, Morice M-C, Tamion F, Abry B, et al. Six-month outcome of emergency percutaneous coronary intervention in resuscitated patients after cardiac arrest complicating ST-elevation myocardial infarction. Circulation 2007;115:1354–62.

[10] Sideris G, Voicu S, Yannopoulos D, Dillinger J-G, Adjedj J, Deye N, et al. Favourable 5-year postdischarge survival of comatose patients resuscitated from out-of-hospital cardiac arrest, managed with immediate coronary angiogram on admission. Eur Heart J Acute Cardiovasc Care 2014;3:183–91.

[11] Bro-Jeppesen J, Kjaergaard J, Wanscher M, Pedersen F, Holmvang L, Lippert FK, et al. Emergency coronary angiography in comatose cardiac arrest patients: do real-life experiences support the guidelines? Eur Heart J Acute Cardiovasc Care 2012;1:291–301.

[12] Möllmann H, Szardien S, Liebetrau C, Elsässer A, Rixe J, Rolf A, et al. Clinical outcome of patients treated with an early invasive strategy after out-of-hospital cardiac arrest. J Int Med Res 2011;39:2169–77.

[13] Anyfantakis ZA, Baron G, Aubry P, Himbert D, Feldman LJ, Juliard J-M, et al. Acute coronary angiographic findings in survivors of out-of-hospital cardiac arrest. Am Heart J 2009;157:312–8.

[14] Zanuttini D, Armellini I, Nucifora G, Grillo MT, Morocutti G, Carchietti E, et al. Predictive value of electrocardiogram in diagnosing acute coronary artery lesions among patients with out-of-hospital-cardiac-arrest. Resuscitation 2013;84:1250–2154.

[15] Link MS, Berkow LC, Kudenchuk PJ, Halperin HR, Hess EP, Moitra VK, et al. Part 7: adult advanced cardiovascular life support 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2015;132:S444–64.

[16] Wagner GS, Macfarlane P, Wellens H, Josephson M, Gorgels A, Mirvis DM, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: Part VI: acute ischemia/infarction a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol 2009;53:1003–11.

[17] Surawicz B, Childers R, Deal BJ, Gettes LS. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: Part III: intraventricular conduction disturbances a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol 2009;53:976–81.

[18] Sgarbossa EB, Pinski SL, Barbagelata A, Underwood DA, Gates KB, Topol EJ, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. N Engl J Med 1996;334:481–7.

[19] Steg PG, James SK, Atar D, Badano LP, Lundqvist CB, Borger MA, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 2012 ehs215.

[20] Thygesen K, Alpert JS, Jaffe AS, White HD, Simoons ML, Chaitman BR, et al. Third universal definition of myocardial infarction. J Am Coll Cardiol 2012;60:1581–98.

[21] Tømte Ø, Andersen GØ, Jacobsen D, Drægni T, Auestad B, Sunde K. Strong and weak aspects of an established post-resuscitation treatment protocol—a five-year observational study. Resuscitation 2011;82:1186–93.

[22] Sianos G, Morel MA, Kappetein AP, Morice MC, Colombo A, Dawkins K, et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. EuroIntervention 2005;1:219–27.

[23] Généreux P, Palmerini T, Caixeta A, Rosner G, Green P, Dressler O, et al. Quantification and impact of untreated coronary artery disease after percutaneous coronary intervention: the residual SYNTAX (Synergy Between PCI With Taxus and Cardiac Surgery) score. J Am Coll Cardiol 2012;59:2165–74.

[24] Hee L, Thomas L, Ullah I, Juergens CP, Lo S, Hopkins A, et al. ''Target-lesion'' SYNTAX score (tSS) determines radiation dose in percutaneous coronary intervention. J Interv Cardiol 2015;28:157–63.

[25] Mehran R, Rao SV, Bhatt DL, Gibson CM, Caixeta A, Eikelboom J, et al. Standardized bleeding definitions for cardiovascular clinical trials a consensus report from the bleeding academic research consortium. Circulation 2011;123:2736–47.

[26] Geri G, Dumas F, Bougouin W, Varenne O, Daviaud F, Pène F, et al. Immediate percutaneous coronary intervention is associated with improved short-and long-term survival after out-of-hospital cardiac arrest. Circ Cardiovasc Interv 2015;8:e002303.

[27] Hosmane VR, Mustafa NG, Reddy VK, Reese CL, DiSabatino A, Kolm P, et al. Survival and neurologic recovery in patients with ST-segment elevation myocardial infarction resuscitated from cardiac arrest. J Am Coll Cardiol 2009;53:409–15.

[28] Vyas A, Chan PS, Cram P, Nallamothu BK, McNally B, Girotra S. Early coronary angiography and survival after out-of-hospital cardiac arrest. Circ Cardiovasc Interv 2015;8:e002321.
[29] Roe MT, Messenger JC, Weintraub WS, Cannon CP, Fonarow GC, Dai D, et al. Treatments, trends, and outcomes of acute myocardial infarction and percutaneous coronary intervention. J Am Coll Cardiol 2010;56:254–63.
[30] Dichtwald S, Matot I, Einav S. Improving the outcome of in-hospital cardiac arrest: the importance of being EARNEST. Cardiothorac Vasc Anesth 2009;19–30.
[31] O'Gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK, De Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61:e78–140.
[32] Noc M, Fajadet J, Lassen JF, Kala P, MacCarthy P, Olivecrona GK, et al. Invasive coronary treatment strategies for out-of-hospital cardiac arrest: a consensus statement from the European Association for Percutaneous Cardiovascular Interventions (EAPCI)/Stent for Life (SFL) groups. EuroIntervention 2014;10:31–7.
[33] Kern KB, Lotun K, Patel N, Mooney MR, Hollenbeck RD, McPherson JA, et al. Outcomes of comatose cardiac arrest survivors with and without ST-segment elevation myocardial infarction: importance of coronary angiography. JACC Cardiovasc Interv 2015;8:1031–40.
[34] Andersen LW, Mackenhauer J, Roberts JC, Berg KM, Cocchi MN, Donnino MW. Etiology and therapeutic approach to elevated lactate levels. Mayo Clin Proc 2013;1127–40.
[35] Holzer M, Bernard SA, Hachimi-Idrissi S, Roine RO, Sterz F, Müllner M, et al. Hypothermia for neuroprotection after cardiac arrest: systematic review and individual patient data meta-analysis. Crit Care Med 2005;33:414–8.
[36] Rab T, Kern KB, Tamis-Holland JE, Henry TD, McDaniel M, Dickert NW, et al. Cardiac arrest: a treatment algorithm for emergent invasive cardiac procedures in the resuscitated comatose patient. J Am Coll Cardiol 2015;66:62–73.
[37] Serruys PW, Morice MC, Kappetein AP, Colombo A, Holmes DR, Mack MJ, et al. Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease. N Engl J Med 2009;360:961–72.
[38] Farkouh ME, Domanski M, Sleeper LA, Siami FS, Dangas G, Mack M, et al. Strategies for multivessel revascularization in patients with diabetes. N Engl J Med 2012;367:2375–84.