Etiology and Determinants of In-Hospital Survival in Patients Resuscitated After Out-of-Hospital Cardiac Arrest in an Urban Medical Center

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Out-of-hospital cardiac arrest (OHCA) is a leading cause of mortality globally. The goals of this study were to describe common causes of OHCA in an urban US medical center, identify predictive factors for survival, and to assess whether neurological status upon return of spontaneous circulation might be predictive of outcomes: 124 consecutive patients aged 18 years and older with OHCA admitted at Advocate Illinois Masonic Medical Center were studied. All patients resuscitated in the field with return of spontaneous circulation then transferred to the emergency department were included. The Glasgow Coma Score (GCS) was evaluated immediately on hospital arrival. In the total group, 34% (42 of 124) were discharged alive. In patients with coronary artery disease (CAD), 51% (20 of 39) were discharged alive versus 26% (22 of 85) of non-CAD patients ($p < 0.01$). Initial GCS ≥ 9 was highly predictive of survival: 94% (34 of 36) of patients with GCS ≥ 9 survived versus 9% (8 of 88) with GCS ≤ 8 ($p < 0.0001$). Defibrillation in the field was predictive of survival (chi-square = 7.81, $p = 0.005$). In the CAD group, all 16 patients with GCS ≥ 9 on presentation to the Emergency Department survived whereas all 13 with GCS ≤ 5 died (both $p < 0.0001$). In the non-CAD group, 18 of 20 patients with GCS ≥ 9 survived, whereas only 2 of 52 with GCS ≤ 5 survived (both $p < 0.0001$). Multivariate analysis by logistic regression showed that the strongest predictor of survival in the non-CAD subgroup was GCS (OR 0.27, CI 0.19 to 0.55, $p < 0.001$). In conclusion, the etiology of the OHCA, immediate neurologic status, and defibrillation in the field (suggesting presenting arrhythmia) were predictive of survival. Immediate neurological recovery (GCS ≥ 9) regardless of etiology was a strong predictor of survival to discharge. Additional predictive factors depend on the etiology of the OHCA event. These data suggest that these straightforward factors can be helpful in predicting outcome in patients resuscitated after OHCA. **(Am J Cardiol 2020;130:78−84)**

Although the management of patients resuscitated with out-of-hospital cardiac arrest (OHCA) requires intensive medical treatment, high-risk procedural utilization, and substantial resources, the pre-discharge mortality rate remains high.[1,2] The accurate identification of predictors of prognosis in patients after resuscitation would greatly assist in subsequent decision-making regarding aggressive management.[3−5] Once admitted to the hospital, rapid strategic determinations regarding resource allocation for invasive procedures and continuation or withdrawal of treatment must be made. Several risk stratification scores exist for individuals with coronary artery disease (CAD) admitted to ICU after OHCA, and whereas strongly predictive in their derivation environment, wider applicability to other etiologies and other settings are unproven.[6,7] Few current studies evaluate differences based on etiology, particularly those not related to acute coronary events. Neurologic findings have shown an association with survival in some but not all studies.[8−13] Accordingly, the goals of this study were to define common causes of OHCA in an urban medical center, to identify predictive factors for survival, and to assess whether neurological status upon return of spontaneous circulation (ROSC) might be predictive of outcomes. The identification of clinical factors associated with survival with retained neurologic function to hospital discharge was also sought. Further, differences in predictive factors between those with acute coronary events and those with non-coronary causes were explored in depth.

METHODS

This study is designed to identify clinical factors predicting survival in patients who survived an outside hospital cardiac arrest (OHCA). The study was conducted at a single urban medical center in Chicago, Illinois. The protocol was designed by the principal investigator (LWK) and approved by the institutional review board.

All patients who regained ROSC after OHCA in the field, transported to the emergency department, and admitted with spontaneous circulation were enrolled in the study. 124 consecutive patients aged ≥ 18 years admitted to a single urban medical center with ROSC after an OHCA in the

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field due to a medical problem comprised the study group. Exclusion criteria were patients who were not resuscitated in the field or were pronounced dead in the Emergency Department (ED). Patients with OHCA due to trauma, shootings or stabbing were also excluded.

Numerous clinical and laboratory variables were collected. The information analyzed included the cause of OHCA, electrolytes (magnesium, calcium, and potassium), initial GCS in ED, left-ventricular ejection fraction, disposition, and various diagnostic and therapeutic interventions. Upon presentation to the ED, each patient had a 12-lead ECG and a neurological evaluation along with other relevant testing. All patients were treated based upon the underlying etiology of the OHCA by the ICU management team.

Patients were divided into 2 major categories: OHCA due to CAD and not due to CAD (non-CAD) causes. The categorization of presumed cardiac or non-cardiac causes was made clinically based on predefined factors previously validated and universally accepted.[14–16] Patients who had OHCA due to CAD were identified as having an acute ischemic event detected by ECG, angiographic, echocardiographic, and other clinical diagnostic testing.

The patients who suffered OHCA due to CAD were further divided into ST-elevation myocardial infarction (STEMI) and acute coronary syndrome (ACS) sub-groups. ACS includes non-ST elevation myocardial infarction (NSTEMI) and unstable angina (UA). Serial 12-lead ECGs were recorded to identify whether the patient met criteria for a STEMI; those that did not have ST elevations comprise the ACS subgroup, including both non-STEMI based on troponin elevation>1.0 mg/dl or unstable angina. Criteria used to diagnose STEMI were: new ST segment elevations > 2 mm in precordial leads and greater than 1 mm in limb leads, in 2 anatomically contiguous leads or a newly diagnosed left bundle branch block (LBBB). Patients were then divided into ACS or STEMI. Patients in the ACS group had troponins recorded per guidelines at 4-5-hour intervals. Patients who underwent coronary angiography and stenting within 24 hours of presentation comprised the early percutaneous intervention (PCI) subgroup.

Upon initial presentation to the ED, the GCS score was recorded to evaluate the initial neurological status of each patient. As suggested in the Traumatic Brain Injuries (TBI) classification,[11–13] the GCS scores were recorded according to Mild Injury (scores 13 to 15), Moderate Injury (scores 9 to 12), Severe Injury (scores 6 to 8), and Comatose (scores 3 to 5).

In non-CAD patients, the underlying cause was identified and treated per standard protocols. Patients suffering OHCA due to sepsis, aspiration, pulmonary embolism (PE), drug overdose, COPD, and other causes were treated accordingly. If no diagnosis was reached clinically, it was classified as “unknown” even if post-mortem examination did identify a cause. The use of mechanical circulatory support and hypothermia protocol was left to the discretion of the appropriate clinical team. All patients who were intubated were placed on ventilator support in the intensive care unit (ICU).

The primary end point was all-cause in-hospital mortality. Survivors are patients who were discharged alive to home with retained neurologic function from our institution. Mortality after referral to hospice or discharge to a long-term facility was considered a death. The requisite outcomes information was obtained from the medical records.

Categorical variables were compared for mortality using Pearson’s chi-square and Fisher’s exact test, stratified by CAD and non-CAD. Multivariate logistic regression was conducted in the non-CAD subgroup, using forward stepwise model selection approach including clinically and statistically significant variables. A p-value <0.05 was set as the level of significance for 2-sided testing and used for measurements of association and final model selection. All analyses were conducted with the use of SAS 9.4 (SAS Institute, Cary, NC).

RESULTS

124 consecutive patients over the age of 18 who had ROSC after OHCA were admitted within 1 hour of the event. 34% (42 of 124) of the patients studied were discharged alive. The mean age of the enrolled patients was 64 ± 18 years. The baseline characteristics of the patients in the CAD and non-CAD groups were similar, except for a higher prevalence of illicit drug use in those who suffered an OHCA due to non-CAD (22% vs 3%, p = 0.0054, see Table 1).

The cause of cardiac arrest was secondary to an acute coronary event in 32% (39 of 124) of CAD-patients. OHCA was due to non-CAD etiologies in 69% (85 of 124). 51% (20 of 39) of the CAD patients survived compared with 26% (22 of 85; p <0.01) of the non-CAD patients. Causes of OHCA not due to CAD are summarized in Figure 1.

In the total group, 34% (42 of 124) were discharged alive. In patients with CAD, 51% (20/39) were discharged alive versus 26% (22 of 85) of non-CAD patients (p <0.01). Tables 2 (CAD group) and 3 (non-CAD group) summarize the clinical and laboratory univariate variables predictive of survival.

Table 1
This table summarizes the baseline characteristics of the 2 groups and compares Non-CAD with CAD

Variable	Non-CAD, n = 85	CAD, n = 39	Missing	p-value
Coronary artery Disease	17 (21%)	12 (32%)	5	0.21
Stroke	11 (14%)	2 (5%)	5	0.18
Peripheral artery disease	2 (3%)	1 (3%)	5	0.96
Congestive heart failure	17 (20%)	11 (28%)	0	0.31
Cardiac surgery	10 (12%)	8 (21%)	5	0.22
Percutaneous coronary intervention	5 (6%)	4 (11%)	5	0.40
Dyslipidemia	27 (33%)	14 (37%)	4	0.67
Hypertension	44 (54%)	22 (58%)	4	0.66
Diabetes mellitus	26 (32%)	15 (40%)	4	0.40
Chronic obstructive pulmonary disease	14 (17%)	5 (13%)	4	0.58
Smoker	25 (31%)	10 (26%)	5	0.62
Excessive alcohol use	28 (35%)	8 (21%)	5	0.13
Use illicit drugs	19 (22%)	1 (3%)	0	0.0054

Only a history of illicit drug use was found to be significantly different.

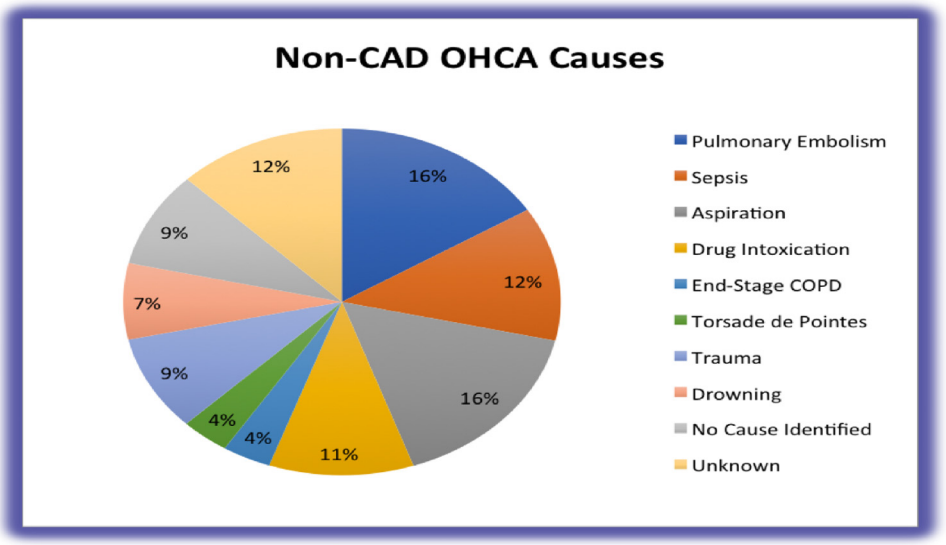

Figure 1. Etiology of OHCA in patients with non-CAD Causes.

GCS was highly predictive of outcomes in the total group. Initial GCS ≥ 9 was highly predictive of survival: 94% (34 of 36) of patients with GCS ≥9 survived versus 9% (8 of 88) of patients with GCS ≤8 (p <0.0001). Of those who expired, 83% (19 of 23, p <0.0001) of CAD-patients and 94% (61 of 65, p <0.0001) of non-CAD patients had GCS ≤8. In the CAD-group, all 16 patients with GCS ≥9 on presentation to the ED survived whereas all 13 with GCS≤5 died (both p <0.0001). In the non-CAD group, 18 of 20 patients with GCS ≥ 9 survived, whereas only 2 of 52 with GCS ≤ 5 survived (both p <0.0001).

Table 2
Specific patient variables of the patients who survived versus expired from the CAD group

Variable	Survived, n = 20	Expired, n = 19	Missing	p-value
Potassium (abnormal)	7 (35%)	7 (37%)	-	0.90
Magnesium (abnormal)	3 (15%)	10 (53%)	-	0.01
Calcium (abnormal)	10 (50%)	8 (42%)	-	0.62
Left-ventricular ejection fraction (normal)	4 (20%)	8 (42%)	5	0.33
Left-ventricular ejection fraction (40%-55%)	7 (35%)	1 (5%)	5	0.33
Left-ventricular ejection fraction (<40%)	9 (45%)	5 (26%)	5	0.33
Glasgow Coma Score (3-5)	0	13 (68%)	-	<0.0001
Glasgow Coma Score (6-8)	4 (20%)	6 (32%)	-	<0.0001
Glasgow Coma Score (9-12)	10 (50%)	0	-	<0.0001
Glasgow Coma Score (13-15)	6 (30%)	0	-	<0.0001
Hypothermia	8 (40%)	14 (74%)	2	0.22
Cardio-pulmonary resuscitation	17 (85%)	19 (100%)	2	0.30
Aged 65 and above	9 (45%)	12 (63%)		0.26
Defibrillation	16 (80%)	11 (58%)	-	0.13
Inotropes	8 (40%)	0	-	0.002
Immediate percutaneous coronary intervention	9 (45%)	3 (16%)		0.10
Coronary artery disease	7 (35%)	5 (26%)	1	0.49
Stroke	0	2 (11%)	1	0.14
Peripheral artery disease	1 (5%)	0	1	0.31
Congestive heart failure	11 (55%)	0	-	0.0001
Cardiac surgery	6 (30%)	2 (11%)	-	0.11
Percutaneous coronary intervention	1 (5%)	3 (16%)	1	0.29
Dyslipidemia	10 (50%)	4 (21%)	1	0.04
Hypertension	11 (55%)	11 (58%)	1	1.00
Diabetes mellitus	9 (45%)	6 (32%)	1	0.31
Chronic obstructive pulmonary disease	1 (5%)	4 (21%)	1	0.15
Smoker	5 (25%)	5 (26%)	1	1.00
Excessive alcohol use	5 (25%)	3 (16%)	1	0.53
Use ollicit drugs	0	1 (5%)	-	0.29

Abnormal magnesium levels, use of inotropes, history of CHF, history of DLD, and GCS scores were found to be significantly different.

Table 3
Specific patient variables of the patients who survived versus expired from the non-CAD group

Variable	Survived, n = 22	Expired, n = 63	Missing	p-value
Potassium (abnormal)	9 (41%)	30 (48%)	-	0.59
Magnesium (abnormal)	8 (36%)	35 (56%)	-	0.12
Calcium (abnormal)	9 (41%)	32 (51%)	-	0.42
Left-ventricular ejection fraction (normal)	9 (41%)	20 (51%)	21	0.88
Left-ventricular ejection fraction (40%-55%)	7 (32%)	13 (21%)	21	0.88
Left-ventricular ejection fraction (<40%)	5 (23%)	10 (16%)	21	0.88
Glasgow Coma Score (3-5)	2 (9%)	50 (79%)	-	<0.0001
Glasgow Coma Score (6-8)	2 (9%)	11 (18%)	-	<0.0001
Glasgow Coma Score (9-12)	12 (55%)	2 (3%)	-	<0.0001
Glasgow Coma Score (13-15)	6 (27%)	0	-	<0.0001
Hypothermia	7 (32%)	22 (35%)	2	0.86
Cardio-pulmonary resuscitation	17 (77%)	57 (91%)	3	0.36
Aged 65 and above	8 (36%)	39 (62%)		0.04
Defibrillation	11 (50%)	20 (32%)	-	0.12
Inotropes	18 (82%)	58 (92%)	-	0.17
Immediate percutaneous coronary intervention	4 (18%)	13 (21%)	4	0.70
Coronary artery disease	5 (23%)	6 (10%)	4	0.14
Stroke	0	2 (3%)	4	0.38
Peripheral artery disease	7 (32%)	10 (16%)	-	0.10
Congestive heart failure	3 (14%)	7 (11%)	4	0.83
Cardiac surgery	2 (9%)	3 (5%)	4	0.50
Percutaneous coronary intervention	7 (32%)	20 (32%)	3	0.89
Dyslipidemia	11 (50%)	33 (52%)	3	0.68
Hypertension	8 (36%)	18 (29%)	3	0.58
Diabetes mellitus	5 (23%)	9 (14%)	3	0.41
Chronic obstructive pulmonary disease	7 (32%)	18 (29%)	4	0.90
Smoker	7 (32%)	21 (33%)	4	0.75
Excessive alcohol use	6 (27%)	13 (21%)	-	0.52

GCS scores and age of 65 or older were found to be significantly different between the 2 groups.

Defibrillation in the field was predictive of survival (Chi-Square = 7.81, p = 0.005). 64% (27 of 42) patients who were defibrillated survived versus 38% (31/82) who were not defibrillated. This finding is explained by the initial cardiac rhythm at presentation; defibrillation represents an OHCA where the initial rhythm was shockable that is, ventricular tachycardia or fibrillation, versus not shockable, that is, pulseless electrical activity or asystole. Other univariate predictors of survival depended on grouping by CAD or non-CAD (Tables 2 and 3).

The strongest predictor of survival in CAD patients was GCS (p <0.001). The next strongest factor was a history of CHF (Table 2). Of the 39 CAD patients, 15 were STEMI, of whom 40% (6 of 15) survived. In the ACS subgroup, 58% (14 of 24) survived. The difference was not significant (p = 0.21). Early PCI was noted to trend toward significance

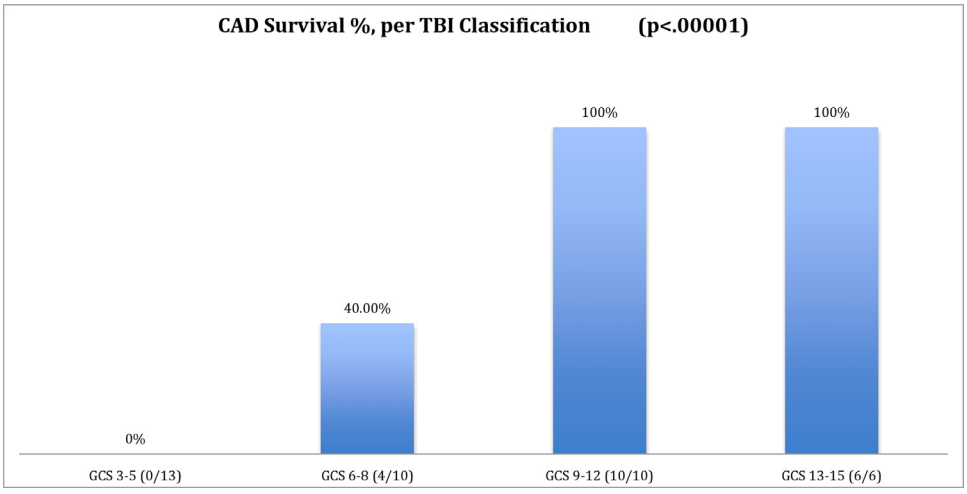

Figure 2. This graph shows the percentage of patients with a CAD cause of OHCA who survived and stratifies them based on GCS score on presentation. The bottom row separates the GCS scores based on TBI classification with GCS 13 to 15 indicating mild TBI, 9 to 12 indicates moderate TBI, 6 to 8 indicates severe TBI, and GCS 3 to 5 indicates the patient was comatose. There is a trend for improved survival with increasing GCS score as shown above.

as a predictor of survival in the CAD group; 67% (6 of 9) STEMI patients and 100% (3 of 3) ACS patients who received early PCI survived, versus 0% (0 of 6) in the STEMI population who did not (p = 0 of 10). Of the other 11 survivors in the ACS subgroup, 6 were treated with CABG and the remaining 5 were treated with medical management.

When the CAD group is further analyzed as STEMI or ACS, 100% (4 of 4) of STEMI patients and 100% (11 of 11) of ACS patients with a GSC Score ≥ 9 survived. Conversely, 18% (2 of 11) of STEMI patients and 25% (3 of 12) of ACS patients with an initial GCS score of 3 to 8 survived (p <0.0001). Figure 2 illustrates a stepwise increase was noted in the survival of patients when divided based on GCS scale.

In STEMI patients, 4 of 4 survived when they had an initial GCS ≥ 9 and underwent early PCI whereas 2 of 5 with a GCS ≤ 8 survived when undergoing early PCI. In the ACS population, similar trends were noted: 100% (3 of 3) survived when they had an initial GCS ≥ 9 and underwent early PCI. Of the other 11 survivors, 82% (9 of 11) who underwent CABG or medical management also had an initial GCS ≥ 9.

In the non-CAD population, patients with GCS ≥ 9 had a survival rate of 90% (18 of 20), whereas just 6% (4 of 65; p <0.0001) survived with an initial GCS ≤ 8. Figure 3 illustrates significantly improved survival with a stepwise increase in GCS score.

Multivariate analysis by logistic regression showed that the strongest predictor of survival in the non-CAD group was initial GCS (OR 0.27, CI 0.19 to 0.55, p <0.001, see Table 4). The next strongest factor was defibrillation in the field. In patients who survived, 36% (11 of 31) of non-CAD patients were defibrillated in the field (OR 1.19, CI .003 to 0.345, p = 0.0043). Age < 65 was also a multivariate predictor.

DISCUSSION

This is the first contemporary urban US series evaluating non-cardiac as well as cardiac causes and the first to consider what parameters predicted survival in all patients who were brought to the ED after ROSC without bias regarding candidacy for various invasive procedures. Etiology of OHCA, immediate neurologic status, and presenting arrhythmias were highly predictive. Additional factors depended on the etiology of the event. The findings of this study strongly reflect clinical practice in urban medical centers in the United States.

Although CAD is the predominant etiology for which procedures exist to specifically treat the underlying pathology, there is a diverse range of other potential cardiac and non-cardiac etiologies[1-3] in urban centers. The range of medical etiologies observed to cause OHCA in this series span multiple specialties. Pulmonary embolism, aspiration, sepsis, and drug intoxication were the predominant non-CAD etiologies observed. This study is the first in the US that collects these etiologies without bias regarding procedure or device use in its inclusion criteria.

In this patient population, OHCA patients of all etiologies with an initial GCS ≤ 8 at first medical evaluation after ROSC had significantly worse outcomes. If confirmed in other populations, it may be that neurological status upon ROSC should be a strong consideration in selecting patients for aggressive interventions. In this patient group, 94% (34/36) of patients with GCS ≥ 9 survived versus 9% (8/88) of patients with GCS ≤ 8 (p <0.0001), and was highly predictive in all etiologic subgroups. This result confirms that GCS at initial presentation is a powerful discriminator of outcome, although not an absolute one. There is no single test or sign at initial presentation that identifies with total accuracy which patients will not survive OHCA.

Although poor neurologic function has been previously identified as a factor for decreased survival, many previous

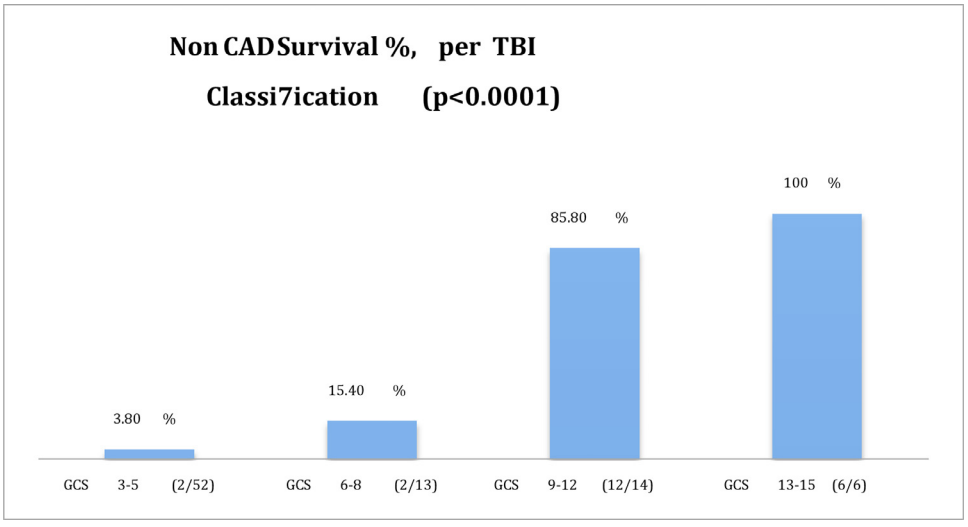

Figure 3. This graph shows the percentage of patients with a non-CAD cause of OHCA who survived and stratifies them based on GCS score on presentation. The bottom row separates the GCS scores based on TBI classification with GCS 13 to 15 indicating mild TBI, 9 to 12 indicates moderate TBI, 6 to 8 indicates severe TBI, and GCS 3 to 5 indicates the patient was comatose. There is a trend for improved survival with increasing GCS score as shown above.

Table 4
Results of multivariate regression for the non-CAD group. GCS score, history of CHF, and defibrillation were the only variables found to be statistically significant.

Variable	Adjusted OR	95% CI Lower	95% CI Upper	p-value
GCS (continuous)	0.2728	0.188	0.548	<0.001
Abnormal magnesium	0.9427	0.621	25.007	0.1457
Age 65 and above	0.9802	0.756	35.249	0.0940
Use of inotropic drugs	1.1048	0.043	3.282	0.3765
Hx of CHF	1.3237	0.005	0.944	0.0452
Use of Illicit Drugs	1.9658	0.012	25.891	0.7606
Defibrillation	1.1868	0.003	0.345	0.0043

studies do not quantitate the dysfunction. Neurologic signs post OHCA have been studied in detail; myoclonus and seizures are a poor prognostic sign[6–13] but the predictive power is ambiguous. The GCS has similarly been extensively studied with variable correlation with outcome, and at various time points.[10–13] Moreover, other studies evaluating GCS use neurologic function as an outcome, rather than to study its correlation with mortality. Neurologic findings at 24 hours are often highly predictive of neurologic recovery, but that time frame is too late to be clinically relevant to determining early aggressive management. Additionally, most previous studies score GCS at 48 or 96 hours, whereas in this study, initial GCS on presentation in the ED is evaluated.

Further, an initial shockable cardiac rhythm was associated with improved survival for all patient subgroups. This finding likely reflects the experience that shockable rhythms are typically due to acute medical issues and that this rhythm is more easily restored than asystole or pulseless electrical activity. Additional clinical factors associated with survival were dependent on CAD nor non-CAD etiology: CAD patients who survived had earlier invasive procedures performed, whereas those who survived non-CAD OHCA were younger and had less CHF.

Recent studies are highly variable in the predictive factors correlating with survival.[5–13,17–19] Age has been variable in its predictive capability, with several studies showing a strong correlation and others not.[17–19] Duration of the arrest and presence of shockable rhythm have also been found to be predictive.[18–20] Forty percent of patients with OHCA are found with ventricular fibrillation/ventricular tachycardia, yet only 22% achieve ROSC.[9] Several pre- and intra-arrest factors have been shown to be associated with unfavorable neurological outcomes and overall outcomes.[1,5,6,21–27] When deciding whether to offer invasive treatments, it is prudent to consider the presence of multiple co-morbidities that portend a poor short- and long-term prognosis.[19,21–26]

In CAD, a variety of predictive scores have been identified, but none have been definitively shown to be predictive in validation subsets in other patient groups. The Cardiac Arrest Hospital Prognosis score (CAHP)[21,25] demonstrated strong correlation with high and low scores but its mid-range was not as valuable. The CREST[23] model used 5 variables to stratify OHCA patients without STEMI according to the risk of circulatory death. The C-GRA*p*H score[26] assessed early stratification of neurologic outcome after OHCA with targeted temperature management (TTM). In non-CAD presentations, no predictive scores are validated, although some studies suggest[26] that prognosis is related to the particular etiology. However, no previous multivariate analyses distinguish among the factors identified in this study and the specific etiology. The Pittsburgh Cardiac Arrest Category illness severity score[27] similarly showed an association between early neurologic status and in-hospital survival, but there were exceptions, perhaps because the studied examinations occurred 6 hours or more after admission.

The main limitation of this study is its portrayal of a single center urban experience with a smaller number of patients than in registries, although the number comprising the study group compares favorably with other single center published experiences.

In conclusion, in individuals with ROSC after OHCA, immediate neurological recovery (GCS ≥9) regardless of etiology and defibrillation were strongly predictive of survival to discharge. These data suggest that these straightforward factors can be helpful in predicating outcomes in patients resuscitated after OHCA.

Author contributions

Amir J Khan, MD: Acquisition of Data, Interpretation of data, drafting the manuscript & revising the manuscript. Carmen Jan Liao, MD: Conception and design of study along with acquisition of data. Christopher Kabir, MS: Analysis of data and interpretation of data. Osama Hallak, MD: Acquisition of data and revising the manuscript. Mohammad Samee, MD: Conception and design of study. Steven Potts, MD: Conception and design of study. Lloyd W Klein MD, FACP: Conception and design of study, interpretation of data, drafting the manuscript, revising the manuscript and study supervision.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relations that could have appeared to influence the work reported in this report.

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