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Clinical paper

The acute respiratory distress syndrome after out-of-hospital cardiac arrest: Incidence, risk factors, and outcomes

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Abstract

Objective: To define the incidence of the acute respiratory distress syndrome (ARDS) following out-of-hospital cardiac arrest (OHCA) and characterize its impact on outcome.

Methods: This was a retrospective cohort study conducted at two urban, tertiary, academic hospitals from 2007 to 2014. We included adults with non-traumatic OHCA and survived for ≥48 h. Patients who received mechanical ventilation for ≥24 h, had 2 consecutive arterial blood gases with a ratio of the partial pressure of oxygen to the fraction of inspired oxygen ≤300, and bilateral radiographic opacities within 48 h of hospital admission were defined as having ARDS. We examined the associations between ARDS and outcome using multivariable analyses and performed sensitivity analyses excluding patients with evidence of cardiac dysfunction.

Results: Of 978 OHCA patients transported to the study hospitals, 600 were mechanically ventilated and survived ≥48 h. A total of 287 (48%, 95% CI 44–52%) met criteria for ARDS within 48 h of admission. There were no differences in demographics, OHCA etiology, or cardiac rhythm according to ARDS status. Patients with ARDS had higher hospital mortality, longer ICU stays, more ventilator days, and were less likely to survive with full neurologic recovery. Upon excluding patients with cardiac dysfunction, the incidence of ARDS was unchanged.

Conclusion: Nearly half of initial OHCA survivors develop ARDS within 48 h of hospital admission. ARDS was associated with poor outcome and increased resource utilization. OHCA should be considered among the traditional ARDS risk factors.

Keywords: Out-of-hospital cardiac arrest, OHCA, Cardiac arrest, Acute respiratory distress syndrome, ARDS, Cardiopulmonary resuscitation, Post-arrest care

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Introduction

Approximately 350,000 persons in the United States suffer out-of-hospital cardiac arrest (OHCA) each year.[1] Many who survive the initial event will suffer from the post-cardiac arrest syndrome (PCAS), a highly inflammatory state characterized by reperfusion injury, oxidative stress, and multi organ dysfunction.[2–6]

The acute respiratory distress syndrome (ARDS) is defined, according to the Berlin definition, by hypoxemia and bilateral radiographic opacities not fully explained by volume overload, that occur acutely in relation to a clinical insult.[7] Cardiac arrest is not listed among the traditional "clinical insults" known to cause ARDS.[8] Possible mechanisms of ARDS development in patients with PCAS might include reperfusion injury, oxidative stress, pulmonary contusion from chest compressions, ventilator-induced lung injury, aspiration, and infection. Additionally, there is substantial overlap between the pathophysiology of ARDS and PCAS, including inflammation, leukocyte activation, activation of coagulation pathways, and altered permeability of alveolar endothelial and epithelial barriers.[9,10]

The exact incidence of ARDS after OHCA is unknown. One study described an 5% incidence of post-arrest ARDS, while another study reported that 65% of patients had a ratio of the partial pressure of arterial oxygen to fraction of inspired oxygen (P_aO_2:F_IO_2)$\leq$ 300, implying a much higher incidence.[11,12] Because prior studies report widely variable incidences, we sought to describe the epidemiology of ARDS in a modern OHCA cohort using the contemporary Berlin definition.[7] Further, while ARDS itself is associated with a mortality of up to 40% (depending on severity), the impact of ARDS after OHCA on overall outcome has not been described.[13,14] Our primary aim is to describe the incidence of ARDS in patients who have suffered OHCA. We also test the association between ARDS and survival and favorable neurologic outcome at hospital discharge.

Materials and methods

Ethics approval and setting

The University of Washington Human Subjects Division approved this study (Study ID #49480). This study site was Seattle, Washington, a U. S. city with 725,000 residents and 83.9 square miles. The Seattle Fire Department's emergency medical services system, Seattle Medic One, has been described previously.[15]

Harborview Medical Center (HMC) is a 413-bed public hospital and trauma center. University of Washington Medical Center (UWMC) is a 450-bed quaternary, University-affiliated hospital. HMC and UWMC are core teaching sites for the University of Washington School of Medicine and affiliated residency and fellowship programs. Combined, HMC and UWMC receive approximately 100,000 emergency department visits and 120–140 patients with OHCA cases each year.

Study design and population

This was a retrospective cohort study of adult patients who suffered non-traumatic OHCA in the City of Seattle between January 1, 2007 and December 31, 2014, were resuscitated, transported to HMC or UWMC. We included patients who survived for at least 48 h to ensure that they had sufficient time to develop ARDS prior to death.

Outcomes

The primary outcome was the incidence of ARDS, which we defined by two consecutive arterial blood gas (ABG) analyses with P_aO_2:$F_IO_2 \leq 300$ in a mechanically ventilated patient with bilateral radiographic opacities.[7] We elected a priori to use two consecutive ABGs to ensure that patients with transient hypoxemia were not included. We performed several sensitivity analyses excluding patients with markers of cardiac dysfunction. We screened patients for development of ARDS within the first 48 h (from hospital admission) to identify patients with ARDS more likely related to the initial OHCA event.

Secondary outcomes included ICU length of stay (LOS), hospital LOS, ventilator days, hospital mortality, and favorable neurologic status at hospital discharge. Seattle Medic One has comprehensive outcome information on neurologic function using the Cerebral Performance Category score.[16] Favorable neurologic outcome at hospital discharge was defined as a Cerebral Performance Category score of 1–2.

Data collection

Qualifying OHCA patients were identified in the Seattle Fire Department cardiac arrest registry.[15] Prehospital records were linked to hospital medical records and discrete hospital admissions using unique identifiers (name, date of birth, address, pre-hospital and hospital encounter date and time). Emergency department and inpatient data were abstracted electronically from the hospital medical record. Chest radiographs and computed tomography scans obtained during hypoxemic episodes were screened for bilateral pulmonary opacities which could represent ARDS.[7,8] Using a validated tool, radiology reports were queried for key qualifying (e.g., bilateral opacities, edema, alveolar filling) and disqualifying (e.g. pleural effusions, masses, lobar collapse) terms.[17] Blinded chest radiograph review by two critical care physicians was used to refine the search algorithm and adjudicate all "possible" qualifying radiographs. Additionally, approximately 10% of radiographs were randomly assigned for review by a blinded critical care physician, and agreement between the query tool and physician reviewer was assessed.

ARDS risk factors were abstracted from ICD-9 codes (pneumonia, non-pulmonary sepsis, pulmonary contusion, pancreatitis, drug overdose), and from prehospital and hospital medical records (documentation of witnessed aspiration, use of vasopressors, blood transfusion). Comorbidities were tabulated using the Charlson index.[18] The Acute Physiology Score (APS) of APACHE (Acute Physiology and Chronic Health Evaluation)-3, which has been previously validated in the cardiac arrest population, was used to define illness severity.[19] Temperature points were excluded from the APS as 78% of the cohort received targeted temperature management, so temperature likely did not reflect true physiologic abnormality. Ventilator and arterial blood gas parameters were abstracted at times zero (first recorded values after hospital arrival), 12 h, and 24 h; these time points were selected a priori.

Statistical analysis

Baseline demographic characteristics, clinical variables, and outcomes were compared according to ARDS status using two-sample t-tests for means, Mann–Whitney tests for medians, and Chi-squared tests for categorical variables.

Using multivariable logistic regression, we fit models to evaluate the associations between ARDS and hospital mortality and ARDS neurologic outcome at hospital discharge. We adjusted the models for age, Charlson index (coded as binary with a cutoff of 2), and the presence of shockable rhythm. Analyses were performed using SAS 9.4 (SAS Institute Inc., Cary, NC, USA).

Sensitivity analyses

We performed a sensitivity analysis to elucidate the relative contribution of cardiac dysfunction to patients' hypoxemic respiratory failure. We excluded patients with the following markers of cardiac dysfunction: (1) a mechanical circulatory support device (most likely to have the highest severity of heart failure), (2) left ventricular ejection fraction <40% within the first 48 h after ROSC, and (3) percutaneous coronary intervention within the first 24 h after ROSC. We also performed a separate sensitivity analysis excluding patients lacking a traditional ARDS risk factor, as specified by the Berlin definition.[8] We examined how excluding these patients would affect ARDS incidence.

Results

Patient characteristics and cardiac arrest care

A total of 978 OHCA patients were transported to the study hospitals from 2007 to 2014 (Fig. 1). Of these, 600 met eligibility criteria and were included. Mean age was 56 ± 16 years, 74% were male, and 69% were white (Table 1). Overall survival to hospital discharge was 60% (95% CI 55–64%), with 328 patients (55%, 95% CI 51–59%) surviving with complete neurologic recovery or mild impairment (CPC 1 or 2).

A total of 45% had an initial shockable cardiac arrest rhythm (ventricular fibrillation or pulseless ventricular tachycardia), and the majority of events were witnessed (Table 2). Most patients received prehospital bag valve mask ventilation (98%) and endotracheal intubation (95%). The majority (78%) of patients received targeted temperature management (TTM) (Table 2). Patients who developed ARDS received TTM more frequently than those who did not (86 vs 70%, $p < 0.05$).

Ventilator parameters and arterial blood gas tensions

Nearly all patients (96%) received assist-control, volume-cycled mechanical ventilation initially. (Supplemental Table 1) Within 24 h, a small number of patients were breathing spontaneously on a pressure support mode or following extubation. Patients in the ARDS group were ventilated initially with higher median tidal volumes, respiratory frequency, and PEEP. By 12 h, there was no difference in tidal volumes, and by 24 h, the ARDS group was ventilated with lower tidal volumes when compared with the non-ARDS group, on average. Static compliance of the respiratory system was lower at all time points in the ARDS group. Patients who developed ARDS received significantly higher median initial F_IO_2 (1.0, IQR 0.5–1) compared with patients without ARDS (0.5, IQR 0.5–1.), $p < 0.05$. Patients in the ARDS group had lower P_aO_2 values across all time points. Mean P_aO_2:F_IO_2 at zero, 12, and 24 h was 155, 240 and 220 in the ARDS

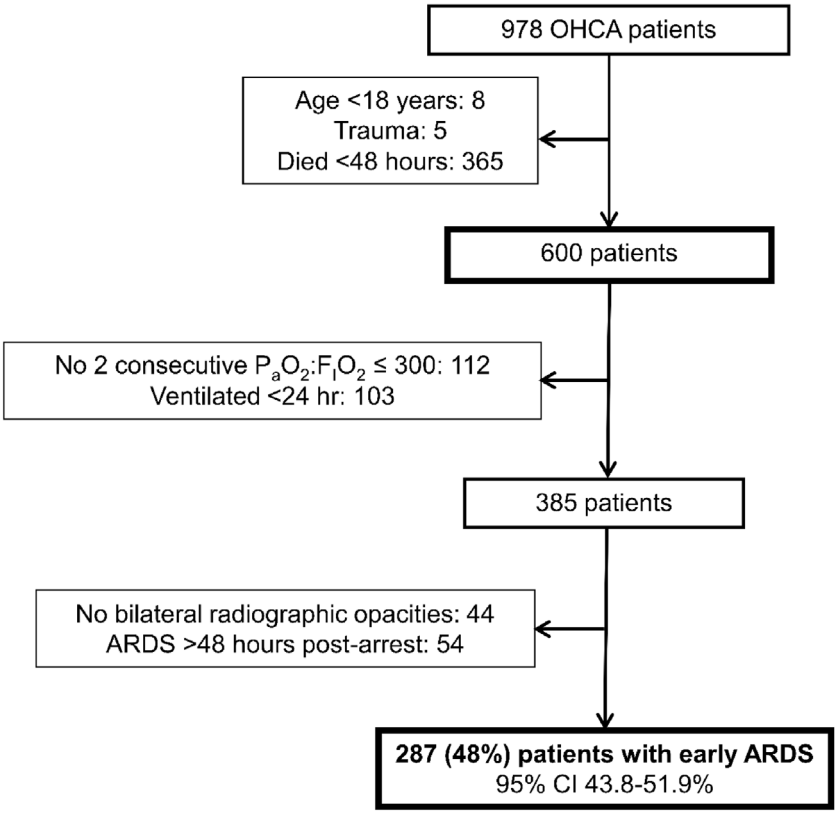

Fig. 1 – Patient flow diagram.
OHCA: out of hospital cardiac arrest, P_aO_2:F_IO_2: ratio of partial pressure of arterial oxygen to fraction of inspired oxygen, ARDS: acute respiratory distress syndrome.

Table 1 – Patient characteristics.

Characteristic	Cohort *N = 600*	No ARDS *N = 313*	ARDS *N = 287*	p-Value[a]
Age (mean ± SD)	56 ± 16	55 ± 16	57 ± 15	0.19
Male gender	443 (74%)	233 (74%)	219 (73%)	0.72
White race	413 (69%)	220 (70%)	193 (67%)	0.11
APS-3 (mean ± SD)	103 ± 32	90 ± 33	117 ± 24	<0.05
Independent prior to cardiac arrest	476 (79%)	253 (81%)	223 (78%)	0.33
Charlson comorbidity index (mean ± SD)	2.5 ± 2.5	2.1 ± 2.4	2.9 + 2.5	<0.05
Selected Charlson comorbidity categories				
Congestive heart failure	208 (35%)	85 (27%)	123 (43%)	<0.05
Chronic pulmonary disease	159 (27%)	74 (24%)	85 (30%)	0.09
Myocardial infarction	204 (34%)	94 (30%)	110 (38%)	<0.05
Moderate to severe chronic kidney disease	97 (16%)	41 (13%)	56 (20%)	<0.05
Traditional ARDS risk factor[b]				
Any	444 (74%)	206 (66%)	238 (83%)	<0.05
Shock	199 (33%)	72 (23%)	127 (44%)	<0.05
Aspiration	205 (34%)	90 (29%)	115 (40%)	<0.05
Overdose	135 (22%)	80 (26%)	55 (19%)	0.06
Pancreatitis	13 (2%)	5 (2%)	8 (3%)	0.32
Pneumonia	217 (36%)	98 (31%)	119 (42%)	<0.05
Pulmonary contusion	20 (3%)	8 (3%)	12 (4%)	0.27
Sepsis, no pneumonia	37 (6%)	13 (4%)	24 (8%)	0.03
Transfusion	35 (6%)	16 (5%)	19 (7%)	0.43

SD: standard deviation, APS: Acute Physiology Score component of Acute Physiology and Chronic Health Evaluation-3 Score with temperature removed, ARDS: acute respiratory distress syndrome, AIDS: Acquired Immune Deficiency Syndrome.
[a] Comparing ARDS versus no ARDS.
[b] Not mutually exclusive.

Table 2 – Cardiac arrest characteristics and care.

Characteristic	Cohort *N = 600*	No ARDS *N = 313*	ARDS *N = 287*	*p-Value*[a]
Initial cardiac rhythm				0.26
Ventricular fibrillation/tachycardia	264 (44%)	139 (44%)	125 (45%)	
Pulseless electrical activity	209 (35%)	109 (35%)	100 (35%)	
Asystole	108 (18%)	51 (16%)	57 (20%)	
Public access defibrillator-no shock advised	1 (0.2%)	1 (0.3%)	0 (0%)	
Witnessed cardiac arrest	382 (64%)	207 (67%)	175 (61%)	0.13
Bystander CPR	275 (46%)	143 (46%)	132 (46%)	0.94
Suspected cardiac arrest etiology				0.51
Cardiac	277 (46%)	145 (46%)	132 (46%)	
Respiratory	136 (23%)	75 (24%)	61 (21%)	
Other	150 (25%)	76 (24%)	74 (26%)	
Unknown	37 (6%)	17 (5%)	20 (7%)	
Prehospital airway management				
Bag-mask ventilation	590 (98%)	303 (97%)	287 (100%)	<0.05
Endotracheal intubation	562 (95%)	282 (93%)	280 (98%)	<0.05
Cricothyrotomy	6 (1%)	3 (1%)	3 (1%)	0.92
Initial pH	*N = 576*	*N = 290*	*N = 286*	
Mean ± SD	7.35 ± 0.12	7.37 ± 0.11	7.32 ± 0.13	<0.05
Initial lactate	*N = 518*	*N = 249*	*N = 269*	
Mean ± SD	4.41	3.58	5.18	<0.05
Targeted temperature management	*N = 598*	*N = 312*	*N = 286*	
	465 (78%)	219 (70%)	246 (86%)	<0.05
Prehospital cold intravenous fluids	81 (14%)	35 (12%)	46 (17%)	0.08
Coronary angiography	219 (37%)	112 (36%)	107 (37%)	0.70
Percutaneous coronary intervention	82 (15%)	37 (13%)	45 (17%)	0.25
Coronary artery bypass grafting	23 (4%)	12 (4%)	11 (4%)	1.0
Intraaortic balloon pump	42 (7%)	13 (4%)	29 (10%)	<0.05
Implantable defibrillator	108 (18%)	70 (22%)	38 (13%)	<0.05

Abbreviations: CPRcardiopulmonary resuscitation, SDstandard deviation.

group, and 428, 340, 300 in the non-ARDS group. There was insufficient variation around tidal volume to evaluate the association between tidal volume and ARDS development or outcome.

Primary outcome: ARDS incidence

A total of 287 patients (48%, 95% CI 44–52%) met criteria for ARDS within 48 h (Fig. 1). Of these, mean initial P_aO_2:F_IO_2 was 155 ± 68 and 29% met hypoxemia criteria for severe ARDS (P_aO_2:$F_IO_2 \leq 100$).

Patients with who developed early ARDS had higher illness severity (mean APS 117 ± 24 vs. 90 ± 33) and more comorbidities (mean Charlson index 2.9 ± 2.5 vs. 2.1 ± 2.4) (Table 1). Notably, there was a significantly higher incidence of congestive heart failure, liver disease, and myocardial infarction among patients with ARDS. A total of 74% of patients had at least one traditional ARDS risk factor, the most common of which were shock (44%), pneumonia (42%), and aspiration (40%). A total of 13.8% of chest radiographs were randomly assigned for review by a critical care physician. The kappa statistic between blinded critical care physician review of chest radiographs and the radiology report query tool was 0.83 for a random sample of 13.8% of patients.

Outcomes of OHCA patients with ARDS

Patients with early ARDS were less likely to survive with full neurologic recovery to hospital discharge than those without ARDS (35% vs 54%, $p < 0.05$) (Tables 3 and 4). Patients with ARDS had longer median intensive care unit LOS, more days requiring mechanical ventilation, longer hospital LOS, and higher hospital mortality (50% vs 32%, $p < 0.05$). Patients with ARDS were less likely to be discharged home than patients without ARDS and were less likely to be independent at hospital discharge.

On multivariable analysis, ARDS within 48 h of hospital admission was associated with hospital mortality (OR 2.0, 95% CI 1.47–2.9) and unfavorable neurologic outcome (OR 2.8, 95% 1.6–3.2). Additionally, after adjustment, ARDS within 48 h was associated with higher odds of hospital discharge to a location other than home (OR 2.0, 95% CI 1.4–2.9) and do-not-attempt-resuscitation status during the hospitalization (OR 2.0, 95% CI 1.4–2.8).

Sensitivity analyses

After excluding patients with the following markers of cardiac dysfunction, the incidence of ARDS was unchanged: mechanical circulatory support device (excluded 42 patients, ARDS incidence 46%, 95% CI 42–51%), ejection fraction <40% within 48 h (excluded 133 patients, ARDS incidence 46%, 95% CI 41–50%), and PCI within 24 h (excluded 59 patients, ARDS incidence 46%, 95% CI 43–51%). Additionally, after excluding 156 patients without a traditional ARDS risk factor, the incidence of ARDS was 54% (95% CI 49–58%).

Table 3 – Primary outcome: incidence of ARDS.

Outcome	N = 600
ARDS, N (%)	287 (48%)
95% CI	44–52%
Mean initial qualifying P_aO_2:F_IO_2	155
95% CI	147–163
ARDS severity, P_aO_2:F_IO_2 category	
Severe ARDS ≤100	82 (29%)
Moderate ARDS 101–200	121 (42%)
Mild ARDS-300	84 (29%)

ARDS: acute respiratory distress syndrome, F_IO_2: fraction of inspired oxygen, P_aCO_2: partial pressure of arterial carbon dioxide, P_aO_2: partial pressure of arterial oxygen.

Discussion

We found that nearly half of OHCA patients surviving for at least 48 h following resuscitation develop ARDS, and that early ARDS is associated with poor outcome and increased healthcare resource utilization. The study results advance our growing understanding of an association between respiratory complications and outcome after cardiac arrest.[11,20–24] Early recognition of ARDS after OHCA may allow for prompt application of therapies known to improve outcome in ARDS such as low tidal volume ventilation, prone positioning, continuous neuromuscular blockade, and conservative fluid management.[25–28]

We described an incidence of ARDS after OHCA that is higher than previously reported. Sutherasan et al. performed a secondary analysis of three observational studies and identified 812 mechanically ventilated patients after cardiac arrest.[12] They described a much lower incidence of ARDS (5%), though it is not clear how ARDS was defined. It is likely that the older American European Consensus Definition was used, which required exclusion of left atrial hypertension as an etiology, and may have significantly reduced the number of post-arrest patients eligible for ARDS diagnosis.[29] A study by Elmer et al. examined the interaction between F_IO_2, lung injury, and neurologic outcome in post-cardiac arrest patients. In a mixed in-hospital and out-of-hospital cohort, they described a mean initial P_aO_2:F_IO_2 of 241, with 65% of patients having a value ≤ 300, which are consistent with our findings. They did not test the association between lung injury and outcome.

We also found that ARDS was associated with hospital mortality and poor neurologic outcome. In our cohort, mortality was 50% among patients who developed ARDS, compared with 32% in patients who did not. The recent LUNG SAFE study, which described the epidemiology and outcomes of patients with ARDS in 50 countries, documented an overall hospital mortality for ARDS of 40%.[13] It should be noted, however, that the mean P_aO_2:F_IO_2 in our cohort at 24 h was 220, implying mild ARDS severity according to the Berlin definition. In the mild ARDS cohort in LUNG SAFE, mortality was substantially lower at 36%, suggesting another driver for high mortality in post-arrest ARDS. After OHCA, ARDS may simply be a marker for illness severity or underlying comorbidities. It is also possible patients with PCAS are particularly sensitive to perturbations in oxygenation and ventilation.[30] A recent propensity-matched study demonstrated an association between time-weighted tidal volume in the first 48 h of hospitalization and neurologic outcome after OHCA.[31] Numerous other studies have demonstrated associations between abnormal in oxygen and carbon dioxide tensions and poor outcome after OHCA.[20,24,40–43,32–39]

Patients may develop hypoxemic respiratory failure after cardiac arrest due to ARDS or due to cardiogenic pulmonary edema in the setting of left ventricular dysfunction.[44] In our cohort, three-quarters of patients had imaging findings consistent with ARDS, but ARDS and cardiogenic pulmonary edema are difficult to distinguish radiographically. The Berlin Definition allows for some flexibility

Table 4 – Outcomes of patients with and without post-arrest ARDS.

Variable	Cohort *N* = 600	No ARDS *N* = 313	ARDS *N* = 287	p-Value
Duration of ventilation				
Median	2.9	1.9	4.2	<0.05
IQR	1.6–6.3	0.7–4.7	2.3–8.5	
95% CI	2.6–3.4	1.7–2.2	3.7–4.9	
ICU length of stay				
Median	4.6	3.7	5.7	<0.05
IQR	2.9–8.1	2.2–6.0	3.5–11.1	
95% CI	4.1–4.9	3.3–4.1	5.0–7.0	
Hospital length of stay				
Median	9	8	10	<0.05
IQR	5–15	5–12	6–21	
95% CI	8–9	7–8	9–11	
DNAR during hospitalization	279 (47%)	123 (39%)	156 (54%)	<0.05
95% CI	42–51%	34–45%	48–60%	
Hospital mortality	243 (41%)	101 (32%)	142 (50%)	<0.05
95% CI	37–45%	27–38%	44–55%	
Discharge location				
Home	217 (36%)	137 (44%)	80 (28%)	<0.05
Care facility	140 (23%)	75 (24%)	65 (24%)	
Expired	243 (41%)	101 (32%)	142 (50%)	
Neurologic status at discharge				
Full recovery	270 (45%)	171 (55%)	99 (35%)	<0.05
Mildly impaired	58 (10%)	30 (10%)	28 (10%)	
Severely impaired	14 (2%)	6 (2%)	8 (3%)	
Comatose	15 (3%)	5 (2%)	10 (4%)	
Expired	243 (41%)	101 (32%)	142 (50%)	
Independence at discharge				
Independent	168 (28%)	110 (35%)	58 (20%)	<0.05
Semi-independent	95 (16%)	58 (19%)	37 (13%)	
Dependent	91 (15%)	42 (13%)	49 (17%)	
Unknown	3 (0.5%)	2 (0.6%)	1 (0.3%)	
Expired	243 (41%)	101 (32%)	142 (49%)	

IQR: interquartile range, CI: confidence interval, DNAR: do not attempt resuscitation.

when differentiating cardiogenic and non-cardiogenic edema: it states, “Respiratory failure not be fully explained by heart failure or volume overload”, implying that patients can have concomitant ARDS and cardiogenic pulmonary edema, and “Need objective assessment to exclude (e.g., echocardiography) to exclude hydrostatic edema only if no risk factor present”.[7] Nearly 75% of our cohort had a traditional ARDS risk factor, most commonly shock, pneumonia, and sepsis, which are known to be common after cardiac arrest.[7,8,45,46] Nonetheless, cardiac arrest itself may be an ARDS risk factor. There is substantial overlap between the pathophysiology of ARDS and PCAS. Following reperfusion after cardiac arrest, cytokines, activated leukocytes, and reactive oxygen species are distributed throughout the body, leading to a profound systemic inflammatory response.[10] Much like in sepsis, a well-characterized ARDS risk factor, blood concentrations of various cytokines, soluble receptors, and endotoxin increase; the magnitude of these changes is associated with patient outcomes.[4,7]

Furthermore, our findings were robust to sensitivity analyses excluding patients with markers of cardiac dysfunction.

Limitations

Previous studies in other disease states, such as subarachnoid hemorrhage, documented that longer ICU stay is associated with development of ARDS.[47] To reduce the effect of competing risk of death from other causes and to avoid including patients who develop ARDS for reasons other than those directly related to the initial OHCA event, such as ventilator-associated pneumonia, we screened for ARDS within 48 h of admission. It is therefore possible that we underestimate the true incidence of ARDS after OHCA. It is also possible that some patients had hypoxemia and radiographic opacities fully explained by cardiogenic pulmonary edema but were not captured in our sensitivity analyses, as patients with ARDS had higher incidence of pre-existing congestive heart failure, coronary artery disease, and chronic kidney disease. Because patients with ARDS had more comorbidities, and higher illness severity, it is also possible that ARDS is simply a marker of critical illness. Our sensitivity analysis included one element, coronary angiography within the first 24 h, that may not be a true indicator of underlying heart disease. Because of observational data suggesting an association with favorable outcome after cardiac arrest, many centers perform early coronary angiography for any OHCA patient without an identified alternative precipitating etiology.[48–50] We were unable to examine the association between tidal volume and development of ARDS or outcome as there was little variation around tidal volumes of 8 ml/kg of predicted body weight in our study cohort, did not stratify patients according to ARDS severity Although overall OHCA survival was relatively unchanged over the study period, there likely been other secular changes in post-arrest and critical care over time that were not accounted for.

Conclusions

Nearly half of all out-of-hospital cardiac arrest survivors develop ARDS within 48 h of hospital admission. Early ARDS is associated with lower survival, worse neurologic outcome, and increased resource utilization in this population. OHCA should be considered among the traditional risk factors for ARDS. Future studies should prospectively evaluate whether specific mechanical ventilation strategies, such as low tidal volume ventilation and avoidance of hyperoxia, might reduce the incidence of ARDS after OHCA.

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NJ conceived the study, drafted manuscript, and takes primary responsibility. EC collected and analyzed data. MP, DC, DG, TR, and CLH all reviewed and edited the manuscript. The authors have no conflicts of interest. The study was funded by the Medic One Foundation.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.resuscitation.2019.01.009.

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