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Factors affecting the return of spontaneous circulation in cardiac arrest patients

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Abstract

The aim of this study was to determine the factors affecting the return of spontaneous circulation (ROSC) in cardiac arrest patients who underwent quality chest compressions as recommended by international guidelines. In this retrospective observational study, the data of nontraumatic out-of-hospital cardiac arrest (OHCA) patients (n = 784) brought by an ambulance to emergency between January 2018 and December 2019 were extracted from the validated hospital automation system. About 452 patients met inclusion criteria. All eligible patients for analysis were treated with an automatic cardiopulmonary resuscitation (CPR) device for chest compression.. Significance threshold for *P*-value was < 0.05. Logistic regression analysis was used to determine the factors affecting mortality. 61.7% (n = 279) of the study population was male and 65.0% of patients (n = 294) had OHCA. 88 patients (19.5%) had a shockable rhythm and were defibrillated. There was a 0.5-fold increase in mortality rate in patients with thrombocyte count < 199 × 10^9/L (OR: 0.482, 95% CI: 0.280–0.828) and CPR duration longer than 42 minutes led to a 6.2-fold increase in the probability of ROSC (OR: 6.232, 95% CI: 3.551–10.936) (*P* < .05). There is no clear consensus on the ideal resuscitation duration; however, our study suggests that it should last at least 42 minutes.

Abbreviations: ALT = alanine aminotransferase, AST = aspartate aminotransferase, CFIO = continuous flow insufflation of oxygen, CPR = cardiopulmonary resuscitation, ECG = electrocardiographic, Hb = hemoglobin, HCO_3 = bicarbonate, hs-TnI = high-sensitive-troponin-I, IHCA = in-hospital cardiac arrest, OHCA = out-of-hospital cardiac arrest, $PaCO_2$ = partial pressure of carbon dioxide, PaO_2 = partial pressure of oxygen, ROC = receiver operating characteristic, ROSC = return of spontaneous circulation, SpO_2 = peripheral saturation of oxygen.

Keywords: cardiac arrest, chest compression, resuscitation

1. Introduction

Various international guidelines detail the management of sudden cardiac arrest and these have been updated yearly. Although the mortality rate is high, early initiation of cardiopulmonary resuscitation (CPR) improves the return of spontaneous circulation (ROSC).[1] In 1960, Kouwenhoven et al[2] reported about 14 patients who survived sudden cardiac arrest with close chest compression for the first time in literature. In accordance with the improvements and updates in CPR all over the world, a chain-of-survival has been developed.

CPR consists of standard chest compressions and artificial ventilation. This procedure helps to decrease anoxic cell damage, and protects the cardiac, pulmonary, and cerebral circulation, thereby enabling these 3 organs to continue functioning. A high-quality CPR aims to restore the pre-arrest quality of life and maintain functional recovery.

Updates on CPR guidelines made further recommendations to ameliorate the outcomes of CPR but the improvement has been limited. Standard chest compression and excessive ventilation have been especially emphasized to increase the awareness in the last decade.[3–5] Appropriate depth and rate of chest compressions have been noted as determining factors for quality, yet adequacy parameters for high-quality chest compressions to maintain normal physiological processes have not been fully determined.[6–8]

Each chain-of-survival step should be performed correctly to achieve a successful CPR. Various studies revealed that in

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developed countries, the majority of errors during CPR are preventable rescuer-dependent errors.[9] Besides, determining the weak chain-of-survival circles and preventable reasons may increase the CPR success rate.

Many parameters have been studied to predict survival after cardiac arrest. Among these are witnessed events, bystander CPR, shockable initial rhythms, bystander defibrillation, older age, cardiac arrest occurring at home and prolonged duration of CPR.[10–12] There are also studies showing that biochemical parameters may be useful in predicting ROSC.[13] However, studies are still ongoing for clear and definitive evidence. According to our best knowledge, there is no clear, well defined, and significant evidence about the factors that improve ROSC in patients who received standard and quality chest compressions. The aim of this study was to determine the factors affecting ROSC in patients who received quality chest compressions.

2. Materials and methods

2.1. Study protocol and participants

After the ethics committee approval (Health Science University, Adana City Research and Training Hospital Ethics Committee Meeting Number: 47, Decision Number: 671, Date: January 8, 2020), this retrospective observational study was conducted in Health Science University, Adana City Research and Education Hospital Emergency Department (ED). Our hospital is a tertiary center and approximately 300,000 patients present to our ED annually. Patients with cardiac arrest who were admitted to the ED between January 1, 2018, and December 21, 2019, were extracted from the hospital's database.

The exclusion criteria included: history of trauma, age <18 years, and incomplete data. Patients who had nontraumatic cardiac arrest with complete data set were included in the study.

2.2. Data collection

Patients were classified into 2 groups according to where their cardiac arrest occurred: in-hospital cardiac arrest (IHCA) and out-of-hospital cardiac arrest (OHCA). Demographic data, physical examination, electrocardiographic (ECG) findings, and laboratory parameters were recorded. The CPR duration, whether defibrillation or drug treatment were used or not. The ROSC duration at the time of admission and dosage of drug treatment were also noted. We also analyzed risk factors of sudden cardiac death such as hypoxia, hypovolemia, pneumothorax, hyperkalemia, hypoglycemia, hypothermia, cardiac tamponade, pulmonary embolism, myocardial infarction, and intake of a toxic substance using history, physical examination, ECG, and laboratory findings of the patient. The patients in the study were divided into 2 groups. The patients with ROSC were classified as Group 1, and the others were classified as Group 2.

2.3. Laboratory

Blood samples collected and analyzed at admission and CPR starting moment were noted. In arterial blood gas analysis, the pH, partial pressure of oxygen (PaO_2), partial pressure of carbon dioxide ($PaCO_2$), bicarbonate (HCO_3), and lactate levels were analyzed. Low PaO_2 <60 mm Hg (normal range: 80–100 mm Hg) was termed hypoxemia and pH < 7.35 termed acidosis. Serum hemoglobin (Hb), leukocyte, thrombocyte, glucose, urea, alanine aminotransferase (ALT), aspartate aminotransferase (AST), sodium, potassium, calcium, ionized calcium, and high-sensitive-troponin-I (hs-TnI) levels were recorded.

2.4. Chest compression

We used an automated CPR device (LUCAS-2, Physio-Control, Inc./Jolife AB, Lund, Sweden) for chest compressions in all patients who had a cardiac arrest in our ED. This device has a rechargeable battery and consists of a big back layer and 2 wrap bands for the chest of the patient. The device gives 100 compressions per minute and active decompression via a piston system that can reach depths of 40 to 50 mm.

2.5. Statistical analyses

Statistical analyses were performed using the SPSS 25.0 statistical software package (SPSS Inc., Chicago). Definitive statistics were expressed as mean ± standard deviation and median (interquartile range, IQR). The Shapiro–Wilk test was used to control for normal distribution. The normally distributed variables were evaluated using independent sample *t* tests. Non-normally distributed variables were analyzed using the Mann–Whitney *U* test. The categorical variables were expressed in frequencies and percentages. The Chi-square, Fisher–Freeman–Halton Exact, and Fisher exact tests were used to compare the categorical data. *P*-value < .05 was considered statistically significant. Logistic regression analysis was used to determine the factors affecting mortality. Sensitivity and specificity values of significant factors affecting mortality were calculated. cutoff values were noted according to receiver operating characteristic curve.

3. Results

Four hundred and fifty two met the inclusion criteria among 784 cardiac arrest patients. The mean age of the eligible patients for analysis was 65.2 ± 15.2 years. Majority of these were male (61.7%) (n = 279). 294 (65.0%) had OHCA. 88 patients (19.5%) had a shockable rhythm and were defibrillated. There were 259 (57.3%) patients who required simultaneous drug administration during CPR. The mean CPR duration was 49.2 ± 7.6 minutes. The mean duration of hospitalization in surviving patients was 17.9 ± 56.1 days. The arterial blood gas analysis and laboratory values of all patients are summarized in Tables 1 and 2.

We achieved ROSC in 86 (19%) patients. The results of the statistical analysis comparing Groups 1 and 2 is summarized in Tables 3 and 4. The hypoxia ratio was higher in Group 2 (*P* = .008) than in Group 1. The age (*P* = .040), urea (*P* = .016), potassium level (*P* = .035) and CPR duration (*P* < .001) were higher in Group 1 than those in Group 2, but the thrombocyte count (*P* = .002) was lower in Group 1 than in Group 2. Other parameters were not statistically significantly different between both groups (*P* > .05) (Table 4).

Significant factors for mortality (age, hypoxia, urea, potassium, thrombocyte, CPR duration) were added into the model given in Table 5. According to our model, there was a 0.5-fold increase in mortality in those with thrombocyte count less than 199 × 10^9/L (OR: 0.482, 95% CI: 0.280–0.828); and CPR duration >42 minutes led to a 6.2-fold increase in the probability of ROSC (OR: 6.232, 95% CI: 3.551–10.936) (*P* < .05).

4. Discussion

In our study, we evaluated the parameters that could predict ROSC in cardiac arrest patients. We used a mechanical compression device for chest compression during resuscitation. Recently, most of the clinicians and healthcare providers have started to use mechanical compression devices in patients with cardiac arrest. These devices reduce the need for human effort but the benefits for the patients remain unclear.[14]

Systematic reviews conducted in recent years have shown that the use of mechanical devices during CPR improved CPR quality but not ROSC outcomes.[15,16]

Standard superior-quality manual chest compressions are not easy to achieve among healthcare providers. We believe that by applying mechanical CPR, we are able to provide this standard for patients. Mechanical compression devices may be useful during hypothermic cardiac arrest, patient transfer by an ambulance, selective coronary angiography, in preparation for extracorporeal CPR, and when a limited number of rescuers are available.[17]

The use of a variety of tests in patients with cardiac arrest are recommended by the guidelines. However, the prognostic utility of blood gases, 1 of the recommended tests, is still unclear. In a retrospective cohort study, it was found that blood gas pH values had a prognostic benefit in cases where ROSC was achieved in the ED in OHCA. Acidemia in the first blood gas analysis following ROSC was associated with a reduced probability of survival after hospital discharge. The optimal cutoff for estimation was highlighted as 7.04. A pH below 7.2 was found to be associated with a reduced probability of survival following hospital discharge in patients with OHCA. It was determined that each 0.1-unit increase in pH could be associated with an increased probability of survival.[18] In a simulation study, the dynamics of PaO_2, $PaCO_2$, and peripheral saturation of oxygen (SpO_2) during simulated CPR were monitored and evaluated at 3 different simulation periods; cardiac arrest, chest compression-only CPR, and chest compression-only CPR with continuous flow insufflation of oxygen (CFIO).[19] The values observed in the first stage remained constant. In the next stage, the $PaCO_2$ started to increase and reached its maximum value at 63.5 mm Hg. The $PaCO_2$ decreased slightly during the CFIO phase, while the PaO_2 and SpO_2 decreased only in the second phase to 44 mm Hg and 70%, respectively. In the final stage, the PaO_2 increased and reached 614 mm Hg. After 2 minutes of CFIO application, the SpO_2 increased above 94%.[19] In our study, the blood gas analysis was performed at the time of admission to the ED. We did not find any significant difference in the blood gas parameters (pH, pO_2, pCO_2, HCO_3) between patients with ROSC and those without.

The importance of complete blood count and biochemical parameters such as serum potassium level in the prognosis of patients with cardiac arrest is not well defined in the literature. There is limited information regarding the relationship between serum potassium level at hospital admission and the neurological outcome following OHCA. In a prospective study from Japan, Shida et al demonstrated that neurological outcomes detoriate with increased serum potassium levels. And Serum potassium levels at admission to ED would be 1 of the most effective prognostic indications for OHCA with spontaneous circulatory return.[20] On the other hand, the sampling site for the blood used for measuring serum potassium level is a challenge. In a study, individual changes in serum potassium values were compared in cardiac arrests due to hypothermia, depending on the sampling site and analytical technique.[21] They compared potassium values in samples taken from the femoral artery and vein, and from a peripheral vein in 15 patients. It was observed that the preferred site for a single potassium sampling should be central venous blood where the lowest level of potassium is measured. While there was a difference in the potassium values between the 2 groups in our study, the logistic regression analysis showed that the potassium value was not significant in determining mortality.

According to our study, thrombocyte count below 199 × 10^9/L increased mortality. Similar to potassium measurement, data is limited when thrombocyte count should be measured in cardiac arrest patients. Thrombocyte count may be helpful in predicting the outcome in critically ill patients. Thrombocyte count decreases over the first 2 days of ROSC. Cotoia et al could not show any difference on thrombocyte count in patients with unfavorable and favorable neurological outcomes.[22]

Whether long-term resuscitation will result in a successful outcome or not, has not been fully clarified yet. And a certain duration for continuing cardiac compressions has not been recommended until today. Clinicians however agree that prolonged resuscitation is sometimes important for nontraumatic cardiac arrest. In a previous study, 1316 adult OHCA patients were analyzed for the duration of ROSC.[23] The relationship

Table 1

Arterial blood gases analysis of all patients.

Parameters (normal range)	Frequency (n)	Percentage (%)
pH (7.35–7.45)		
<7.35	429	94.9
Normal	23	5.1
pO_2 (80–100 mm Hg)		
Low	397	87.8
Normal	55	12.2
pCO_2 (35–45 mm Hg)		
Low	72	15.9
Normal	119	26.3
High	261	57.7
HCO_3 (22–26 mEq/L)		
Low	354	78.3
Normal	74	16.4
High	24	5.3

Table 2

Laboratory values of all patients.

Laboratory parameters (normal range)	Mean ± SD	Med (IQR)
Glucose (74–106 mg/dL)	266.9 ± 166.1	231 (193)
Urea (10–50 mg/dL)	69.1 ± 53.8	50 (14)
Creatinine (0.8–1.3 mg/dL)	1.72 ± 1.3	1.27 (0)
Alanine aminotranspherase (8–40 U/L)	159.6 ± 361.5	50 (167)
Aspartate aminotranspherase (8–40 U/L)	230.7 ± 540.8	69 (210)
Sodium (132–146 mmol/L)	138.3 ± 6.9	138 (11)
Potassium (3.5–5.5 mmol/L)	5.2 ± 1.4	4.98 (1)
Calcium (8.4–10.6 mg/dL)	8.8 ± 0.9	8.9 (1)
Hs-Troponin-I (0–16 ng/L)	2602.5 ± 14205.4	125 (1243)
CK-MB (5–25 µg/L)	10.8 ± 22.8	4.4 (24)
Hemoglobin (10.9–14.3 g/dL)	12.0 ± 2.7	12.2 (2)
Platelets (179–408 10^3/µL)	214.5 ± 106.4	196 (123)
Ionised Ca (1.1–1.4 mmol/L)	1.2 ± 0.1	1.2 (0)
Lactate (9–29 mg/dL)	102.9 ± 46.7	99 (76)

Table 3

Comparison of laboratory data of the study groups.

	Group 1 (n = 86)	Group 2 (n = 366)	*P*
Alanine aminotranspherase (U/L)	54.5 (10–1644)	49 (4–3802)	.162†
Aspartate aminotranspherase (U/L)	77.5 (15–1569)	68 (13–5451)	.314†
Sodium (mmol/L)	138 (97–158)	138 (116–169)	.369†
Potassium (mmol/L)	4.52 (3–7.8)	5.0 (1.9–12.7)	.035*,†
Calcium (mg/dL)	8.8 (5.2–10.4)	8.9 (3.3–12.8)	.531†
Hs-Troponin-I (ng/L)	139 (4–244,289)	120 (3–82,315)	.659†
Hemoglobin (g/dL)	12.3 ± 2.6	11.9 ± 2.7	.255‡
Platelets (10^3/μL)	221 (32–617)	190 (10–700)	.002**,†
Ionised Ca (mmol/L)	1.15 (0.8–1.6)	1.17 (0.4–1.9)	.405†
Lactate (mg/dL)	95.3 ± 41.8	104.7 ± 47.7	.093‡
Glucose (mg/dL)	248 (45–990)	228 (20–1373)	.274†
Urea (mg/dL)	40 (17–401)	52 (11–281)	.016*,†
Creatinine (mg/dL)	1.21 (0.4–10.8)	1.29 (0.2–8.4)	.246†
pH			
<7.34	82 (95.3)	347 (94.8)	.838§
Normal	4 (4.7)	19 (5.2)	
pCO_2			
Low	10 (11.6)	62 (16.9)	.453§
Normal	25 (29.1)	94 (25.7)	
High	51 (59.3)	210 (57.4)	
HCO_3			
Low	72 (83.7)	282 (77.0)	.389§
Normal	11 (12.8)	63 (17.2)	
High	3 (3.5)	21 (5.7)	

Abbreviation: HCO_3 = bicarbonate.
†Mann–Whitney *U* test.
‡Independent student *t* test.
§Chi-square and fisher exact test.
*$P < .05$.
**$P < .001$.

Table 4

Comparison of demographic data, cause of cardiac arrest, and treatment practices of the study groups.

	Group 1 (n = 86)	Group 2 (n = 366)	
	N (%)	N (%)	*P*†
Gender			
Male	57 (66.3)	222 (60.7)	.334
Female	29 (33.7)	144 (39.3)	
Arrest place			
Out-of-hospital	62 (72.1)	232 (63.4)	.313
In-hospital	24 (27.9)	134 (36.6)	.128
Cause of cardiac arrest			
Hypoxia	74 (86.0)	345 (94.3)	.008**
Hypovolemia	–	14 (3.8)	.050
Hyperkalemia	21 (24.4)	122 (33.3)	.110
Hypoglisemia	–	5 (1.4)	.276
Myocardial infarction	47 (54.7)	199 (54.4)	.963
Defibrillation requirement	16 (18.6)	72 (19.7)	.822
Drug administration	55 (64.0)	204 (55.7)	.166
Age (yr)	63 (24–89)	67 (20–97)	.040*,‡
CPR duration (min)	40 (30–90)	45 (30–150)	<.001**,‡

Abbreviation: CPR = cardiopulmonary resuscitation.
†Chi-square and fisher exact test.
‡Mann–Whitney *U* test (IQR [Percentage 25–75]).
*$P < .05$.
**$P < .001$.

between the prehospital resuscitation time and the functional outcome on the survival of OHCA patients, who achieved prehospital ROSC, was evaluated. According to that study, the median time from CPR commencement to ROSC was 12 minutes. Following more than 25 minutes of CPR, 20.4% of patients achieved ROSC. Consequently, prolonged resuscitation time had significant survival percentages. In study of Bruchfeld et al, they worked non-shockable IHCA and they have found that duration of CPR was the most important predictor of survival, followed by etiology.[24] Prolonged resuscitation has been correlated with poor functional result and survival in IHCAs.[25–27] According to a study by Goldberger et al, it is stated that patients are more likely to achieve ROSC and subsequently survive in hospitals with a median CPR duration of 25 minutes

Table 5

Factors affecting mortality.

	Odd ratio	95% CI	*P*
Age			
<65 yr old	1.000		.301
>65 yr old	1.361	0.759 to 2.441	
Hypoxia			
Yes	1.000		.403
No	1.735	0.477 to 6.305	
Urea			
<41	1.000		.120
41	1.618	0.882 to 2.968	
Potassium			
<4.74	1.000		.200
>4.74	1.447	0.823 to 2.543	
Platelets			
>199	1.000		.008*
<199	0.482	0.280 to 0.828	
CPR duration			
<42	1.000		<.001**
>42	6.232	3.551 to 10.936	

Logistic regression analysis was used to determine the factors affecting mortality. Values for urea, potassium, thrombocyte, and CPR time were grouped in the model by establishing cutoffs based on the patients' mortality results.

Abbreviation: CPR = cardiopulmonary resuscitation.

*$P < .05$.

**$P < .001$.

than in hospitals where CPR times are <25 minutes.[28] In our study, we found that CPR duration of longer than 42 minutes increased the ROSC.

The CPR duration may vary according to the etiology and the status of patients. Resuscitation guidelines are quite detailed for rescuers but recommendations on timing to terminate resuscitation are not clear.[29,30] According to the American Heart Association Guidelines, termination of resuscitation is recommended if emergency medical personnel have not witnessed the arrest, shock has not been delivered prior to hospital admission, and the patient has not reached the ROSC prior to hospital admission.[6] These recommendations have been validated in the literature.[27–38] Unfortunately, there are no certain rules for termination of resuscitation and the final decision to terminate resuscitation should be accompanied by full clinical evaluation, taking into account the very small probability of survival.

5. Limitation of the study

There are some limitations to our study. The first is that it is a retrospective study. Another limitation is that it is a single-centered study. Third, the starting time of CPR in cardiac arrests could not be clearly demonstrated.

6. Conclusion

According to our model, thrombocyte count < 199×10^9/L is associated with increased mortality. Our study also shows, that a resuscitation time of at least 42 minutes in cardiac arrest patients, increases the probability of ROSC. This duration supports that a long resuscitation attempt is not a futile effort. Prospective, multicenter, and large-series studies that clearly demonstrate the time to start CPR in cardiac arrest patients will provide stronger evidence in demonstrating the optimal duration for CPR.

Author contributions

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