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Clinical paper

The early change in pH values after out-of-hospital cardiac arrest is not associated with neurological outcome at hospital discharge

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Abstract

Background: The association between pH values and outcome for patients after out-of-hospital cardiac arrest (OHCA) was not fully elucidated; besides, the relationship of change in pH values and neurological outcome was unknown. The aim was to explore the association of pH values as well as change in pH values and neurological outcome for OHCA cardiac patients.

Methods: The adult patients with non-traumatic out-of-hospital cardiac arrest, shock-refractory ventricular fibrillation or pulseless ventricular tachycardia, and at least two arterial blood gases analysis recorded after admission were included. The change in pH values is calculated as the difference between the second and first pH value, and divided by time interval got the rate of change in pH values. The primary outcome was modified Rankin Score (mRS), dichotomized to good (mRS 0–3) and poor (mRS 4–6) outcomes at hospital discharge. The independent relationship of the first pH value, second pH value, and changes in pH values with neurological outcome was investigated with multivariable logistic regression models, respectively.

Results: A total of 1388 adult patients were included for analysis, of which 514 (37%) had good neurological outcome. The median first pH value and second pH value after admission were 7.21 (interquartile range [IQR] 7.09–7.29) and 7.28 (IQR 7.20–7.36), respectively. The median absolute, relative change, and rate of changes in pH values were 0.08 (IQR 0.01–0.16), 1.10% (IQR 0.11–2.22%), and 0.02 (IQR 0–0.06) per hour, respectively. After adjusting for confounders, the higher first pH value (odds ratio [OR] 3.81, confidence interval [CI] 1.60–9.24, $P = 0.003$) and higher second pH value (OR 9.54, CI 3.45–26.87, $P < 0.001$) after admission were associated with good neurological outcome, respectively. The absolute (OR 1.58, CI 0.58–4.30, $P = 0.368$) and relative (OR 1.03, CI 0.96–1.11, $P = 0.399$) change as well as the rate of change (OR 0.98, CI 0.33–2.71, $P = 974$) in pH values were not associated with neurological outcome.

Conclusions: For OHCA patients, abnormality in pH values was very common, with a more acidic pH value indicating poor neurological outcome. However, the change in pH values was not associated with outcomes.

Keywords: Out-of-hospital cardiac arrest, Post cardiac arrest care, pH value, Neurological outcome, Mortality

Introduction

For out-of-hospital cardiac arrest (OHCA) patients after achieving return of spontaneous circulation (ROSC), acid-base disorder is common due to hypoventilation and impaired tissue perfusion.[1–5] The decrease in pH value could affect cellular function via different mechanisms.[6] Several studies had reported that the pH value was independently associated with neurological outcome and was one of the important predictors of prognosis models for outcomes of OHCA patients.[7–12]

Abbreviations: ABG, arterial blood gas, CI, confidence interval, CKD, chronic kidney disease, CPR, cardiac pulmonary resuscitation, IQR, interquartile range, LOWESS, local weighted scatter smoothing, mRS, modified Rankin Score, OHCA, out-of-hospital cardiac arrest, OR, odds ratio, $PaCO_2$, partial pressure of carbon dioxide, PaO2, partial pressure of oxygen, PEA, pulseless electrical activity, ROC ALPS, resuscitation outcomes consortium amiodarone, lidocaine, and placebo, ROSC, return of spontaneous circulation, SB, sodium bicarbonate, SD, standard deviation, VF, ventricular fibrillation, VT, ventricular tachycardia

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According to the Henderson-Hasselbalch equation, the pH value is dependent on the ratio of HCO_3^- and $PaCO_2$.[13] The guidelines recommend that it is preferable to avoid hypotension and maintain the $PaCO_2$ in a normal physiological range for the post cardiac arrest care,[14,15] which could alleviate acidosis and improve pH value. However, the relationship between the dynamic course or rate of change in pH values and neurological outcome for OHCA patients with ROSC was unclear.

Therefore, the main primary objective of this present study was to investigate the relationship of pH values and neurological outcome as well as the association between the early change in pH values and neurological outcome for OHCA patients with ROSC. In addition, the second objective was to evaluate the association between changes in pH values and hospital mortality.

Methods

Study design and patients

This study was conducted as a retrospective cohort study with prospective data collection. The study included patients from the randomized clinical trial of "Resuscitation Outcomes Consortium amiodarone, lidocaine, and placebo" (ROC ALPS). The ROC ALPS trial was designed to compare the effects of amiodarone, lidocaine, and placebo on survival after OHCA.[16]

The data were available in the Biologic Specimen and Data Repository Information Coordinating Center (https://biolincc.nhlbi.nih.gov). The institutional review board (IRB) approved the protocol and informed consent documents at each participating site. The ethics for secondary analysis of the data was exempted due to the retrospective design and de-identification.

All patients enrolled in the ROC ALPS trial were included. The excluding criteria were: (1) Death before admission; (2) Full recovery before admission; (3) Missing outcome assessment when hospital discharging; (4) Frequency of arterial blood pressure (ABG) less than 2.

Clinical variables and outcomes

Demographic variables including age and sex were extracted. Cardiac arrest related characteristics included bystander witness or not, the initial cardiac arrest rhythm, time to ROSC, and dose of epinephrine during CPR. The initial cardiac arrest rhythm included shockable and non-shockable rhythm. The shockable rhythm were ventricular fibrillation (VF) and tachycardia (VT). The non-shockable rhythm were asystole, bradycardia, and pulseless electrical activity (PEA). Time to ROSC was categorized as $\leq$15 min, >15 to $\leq$30 min, >30 min, and the missing category. The total doses of epinephrine during CPR was summarized and categorized as $\leq$2 mg, >2 to $\leq$4 mg, >4 mg, and the missing category. The use of sodium bicarbonate (SB) during CPR or within 24 h after admission was recorded.

The vital signs including temperature, systolic blood pressure, and heart rate upon admission were recorded. The arterial blood gases (ABG) including pH values, partial pressure of oxygen (PaO_2), partial pressure of carbon dioxide ($PaCO_2$), and bicarbonate (HCO_3^-) during the first 24 h after admission were extracted. The absolute change of pH values was calculated as the difference between the first and second ABG data. The relative change of pH values was calculated as the absolute change of pH values divided by the first pH value. The rate of change between the first and second pH value was calculated as absolute change of pH values divided by the time interval. The rates of change in $PaCO_2$ and HCO_3^- were calculated as absolute change of $PaCO_2$ and HCO_3^- divided by the time interval, respectively. This method was derived from the way to calculate the lactate clearance.[17]

The primary outcome was modified Rankin Score (mRS) at hospital discharge, with higher mRS score indicating poorer neurological outcome (0: no symptoms, 1: no significant disability despite symptoms, 2: slight disability, 3: moderate disability, 4: moderately severe disability, 5: severe disability, 6: dead). According to the mRS score, the good and poor neurological outcomes were defined as mRS 0–3 and 4–6, respectively. The second outcome was hospital mortality, defined as death during this hospitalization.

Statistical analysis

Continuous variables were shown as mean and standard deviation (SD) or median and interquartile range (IQR) after assessing for normality with Shapiro-Wilk test. Categorical variables were reported as numbers and percentages. The difference between patients with good and poor outcome were compared by the chi-square test or Fisher's exact test for categorical variables and the nonparametric Mann-Whitney *U* test for continuous variables. As for variables (time to ROSC and doses of epinephrine) with missing data, the missing rate was reported. Patients with missing data were considered as a unique category for analyzing. The time course of pH value, $PaCO_2$, and HCO_3^- between patients with good and poor neurological outcome were plotted with locally weighted scatterplot smoothing (LOWESS) method.

The multivariable logistic regression models were used to explore the independent relationship of first pH value, second pH value, absolute change in pH values, relative change in pH values, and rate of change in pH values with the neurological outcome as well as the hospital mortality. The covariates were selected using the univariate analysis with a significance level of 0.2 and clinical significance. The final model was built by the stepwise backward elimination method based on likelihood ratio. The results of the univariate analysis and the final model were reported as odds ratio (ORs) with 95% confidence intervals (CIs). Potential multicollinearity between variables was assessed by the variance inflation factor (VIF) using "car" package of R software. The overall fit of the models was assessed by Hosmer-Lemeshow goodness-of-fit test.

The subgroup analysis was conducted as exploratory analysis. Age (<65 or $\geq$65 years), sex (male or female), bystander witness (yes or no), shockable rhythm (yes or no), use of SB, hypothermia (yes or no), and the first pH value (<7.2 or $\geq$7.2) after admission were selected as the factors for subgroup analysis. The interaction effect of the factors on the relationship between rate of change in pH values and neurological outcome were respectively explored by the multivariable model.

All analyses were completed using R software (version 4.1.2, R Foundation for Statistical Computing). A two-sided *P* value of <0.05 was considered statistically significant.

Results

The ROC ALPS trial included 4653 patients for the intention-to-treat analysis. After excluded patients with death (n = 2894) or full recovery (n = 1) before admission and patients without enough ABGs (n = 344) or with missing data (n = 25), a total of 1388 OHCA patients was included for analysis (Supplemental Fig. 1). The demographics

and baseline characteristics of the study patients were displayed in Table 1. Of these, the median age was 62 (IQR 53–71) years, 1053 (76%) was male, and 888 (64%) were witnessed by bystanders. The most common rhythm (84%) after cardiac arrest was shockable (VF or VT). For 514 patients with good neurological outcome (37%), the median age was younger (59 VS 64 years, $P < 0.001$), most were male (79% VS 74%, $P = 0.023$), and had higher proportion of bystander witness (72% VS 59%, $P < 0.001$) and shockable rhythm (94% VS 77%, $P < 0.001$). As expected, patients with poor neurological outcome had longer time to ROSC ($P < 0.001$) and more doses of epinephrine ($P < 0.001$) used in the CPR procedure. Patients with poor neurological outcome had higher proportion of SB usage (31% VS 21%, $P < 0.001$).

The distribution of frequency for ABG tests was shown in Supplemental Fig. 2, and the median frequency was 4 (IQR 3–6). The characteristics of the first and second ABG tests were displayed in Table 2. For the first ABG test after admission, the median time was 51 (IQR 21–145) minutes, and the median pH value was 7.21 (IQR 7.09–7.29). For the second ABG test, the median time was 277 (IQR 166–447) minutes, and the median pH value was 7.28 (IQR 7.20–7.36). The distribution of the first pH value, second pH value, absolute change in pH values, and rate of change in pH values were displayed in Fig. 1. The distribution of $PaCO_2$ and HCO_3^- were showed in Supplemental Fig. 3 and Fig. 4. Patients with good neurological outcome had higher first pH value (7.24 VS 7.19, $P < 0.001$), second pH value (7.31 VS 7.27, $P < 0.001$); However, the absolute (0.07 VS 0.08, $P = 0.549$), relative (−0.98% VS 1.11%, $P = 0.487$) change in pH values, and rate of change in pH values (0.02 VS 0.03 per hour, $P = 0.158$) were similar between patients with good and poor outcomes.

During the first 24 h after admission, the pH value was gradually increased, the $PaCO_2$ was gradually decreased, and the HCO_3^- showed increasing trend for both patients with poor or good neurological outcome, especially during the first 6 h (Fig. 2). The scatter plot of pH value, $PaCO_2$, and HCO_3^- was displayed in Supplemental Fig. 5. For each number of ABG tests, patients with good neurological outcome had higher pH values (Fig. 3). The $PaCO_2$ was similar between patients with good and poor neurological outcome, except for the first ABG test (Supplemental Fig. 6). Patients with good neurological outcome had higher HCO_3^- for the first 7 ABG tests (Supplemental Fig. 7).

After adjusted for covariates (age, sex, rhythm of cardiac arrest, bystander witness, time to ROSC, and doses of epinephrine), the first and second pH value were associated with neurological outcome, respectively; however, the absolute, relative change, and the rate of change in pH values were not associated with neurological outcome and hospital mortality (Table 3). For subgroup analysis, the interaction effect ($P = 0.014$) was found statistically significant for the use of SB on the relationship between rate of change in pH values and neurological outcome (Fig. 4).

Table 1 – Comparison of baseline characteristics of study patients.

Variables	Total (n = 1388)	Good (n = 514)	Poor (n = 874)	P value
Age, years	62 (53, 71)	59 (51, 67)	64 (54, 74)	<0.001
Sex				0.023
Female	335 (24)	106 (21)	229 (26)	
Male	1053 (76)	408 (79)	645 (74)	
Cardiac arrest witnessed	888 (64)	369 (72)	519 (59)	<0.001
Cardiac arrest rhythm				<0.001
Shockable	1159 (84)	483 (94)	676 (77)	
Non-shockable	229 (16)	31 (6)	198 (23)	
Time to ROSC				<0.001
≤15 min	163 (12)	99 (19)	64 (7)	
>15 to ≤30 min	815 (59)	317 (62)	498 (57)	
>30 min	260 (19)	66 (13)	194 (22)	
Missing	150 (11)	32 (6)	118 (14)	
Doses of epinephrine				<0.001
≤2	550 (40)	270 (53)	280 (32)	
>2 to ≤4	488 (35)	134 (26)	354 (41)	
>4	297 (21)	67 (13)	230 (26)	
Missing	53 (4)	43 (8)	10 (1)	
Temperature upon admission,	35.9 (35.0, 36.5)	36.0 (35.2, 36.5)	35.8 (34.8, 36.4)	0.002
Systolic blood pressure upon admission, mmHg	124 ± 32	127 ± 30	122 ± 33	0.006
Heart rate upon admission, bpm	98 (79, 116)	100 (82, 118)	95 (76, 115)	0.005
ICU length of stay, days	6 (3, 10)	8 (5, 12)	4 (2, 9)	<0.001
Duration of mechanical ventilation, days	4 (2, 7)	3 (2, 6)	4 (2, 8)	0.048
Sodium bicarbonate	377 (27)	106 (21)	271 (31)	<0.001
Treatment group				0.256
Placebo	434 (31)	169 (33)	265 (30)	
Lidocaine	486 (35)	165 (32)	321 (37)	
Amiodarone	468 (34)	180 (35)	288 (33)	

Data are mean ± standard deviation, median (interquartile range), or no. (%).
bpm beat per minute, ICU intensive care unit, ROSC return of spontaneous circulation.

Table 2 – ABG between patients with good and poor functional outcome.

Variables	Total (n = 1388)	Good (n = 514)	Poor (n = 874)	P value
Frequency of ABG	4 (3, 6)	4 (3, 6)	5 (3, 6)	0.272
The first ABG after admission				
Time of the first ABG, minutes	51 (21, 145)	50 (21, 150)	52 (21, 140)	0.983
$PaCO_2$, mmHg	46 (39, 57)	46 (39, 53)	47 (39, 60)	0.018
PaO_2, mmHg	124 (79, 241)	116 (81, 215)	129 (77, 261)	0.109
pH	7.21 (7.09, 7.29)	7.24 (7.14, 7.3)	7.19 (7.06, 7.28)	<0.001
HCO_3^-, mmol/L	18 (15, 21)	19 (16, 21)	17 (15, 20)	<0.001
The second ABG after admission				
Time of the second ABG, minutes	277 (166, 447)	256 (154, 412)	277 (166, 447)	0.067
$PaCO_2$, mmHg	41 (34, 48)	40 (34, 47)	41 (34, 50)	0.137
PaO_2, mmHg	117 (79, 184)	123 (85, 179)	112 (76, 185)	0.023
pH	7.28 (7.20, 7.36)	7.31 (7.24, 7.37)	7.27 (7.17, 7.35)	<0.001
HCO_3^-, mmol/L	19 (16, 22)	20 (17, 22)	18 (15, 22)	<0.001
Differences between the first and second ABG				
Time interval, minutes	177 (101, 285)	186 (112, 298)	174 (96, 279)	0.065
Absolute change in pH	0.08 (0.01, 0.16)	0.07 (0.01, 0.16)	0.08 (0, 0.16)	0.549
Relative change in pH, %	1.10 (0.11, 2.22)	0.98 (0.14, 2.14)	1.11 (0.01, 2.3)	0.487
Rate of change in pH per hour	0.02 (0, 0.06)	0.02 (0, 0.06)	0.03 (0, 0.07)	0.158
Rate of change in $PaCO_2$ per hour	−1.64 (−5.38, 0.44)	−1.39 (−4.64, 0.33)	−1.75 (−6.28, 0.5)	0.207
Rate of change in HCO_3^- per hour	0.23 (−0.27, 1.22)	0.28 (−0.18, 1.22)	0.2 (−0.33, 1.21)	0.107

Data are mean ± standard deviation, median (interquartile range), or no. (%).

ABG arterial blood gas, $PaCO_2$ partial pressure of carbon dioxide, PaO_2 partial pressure of oxygen.

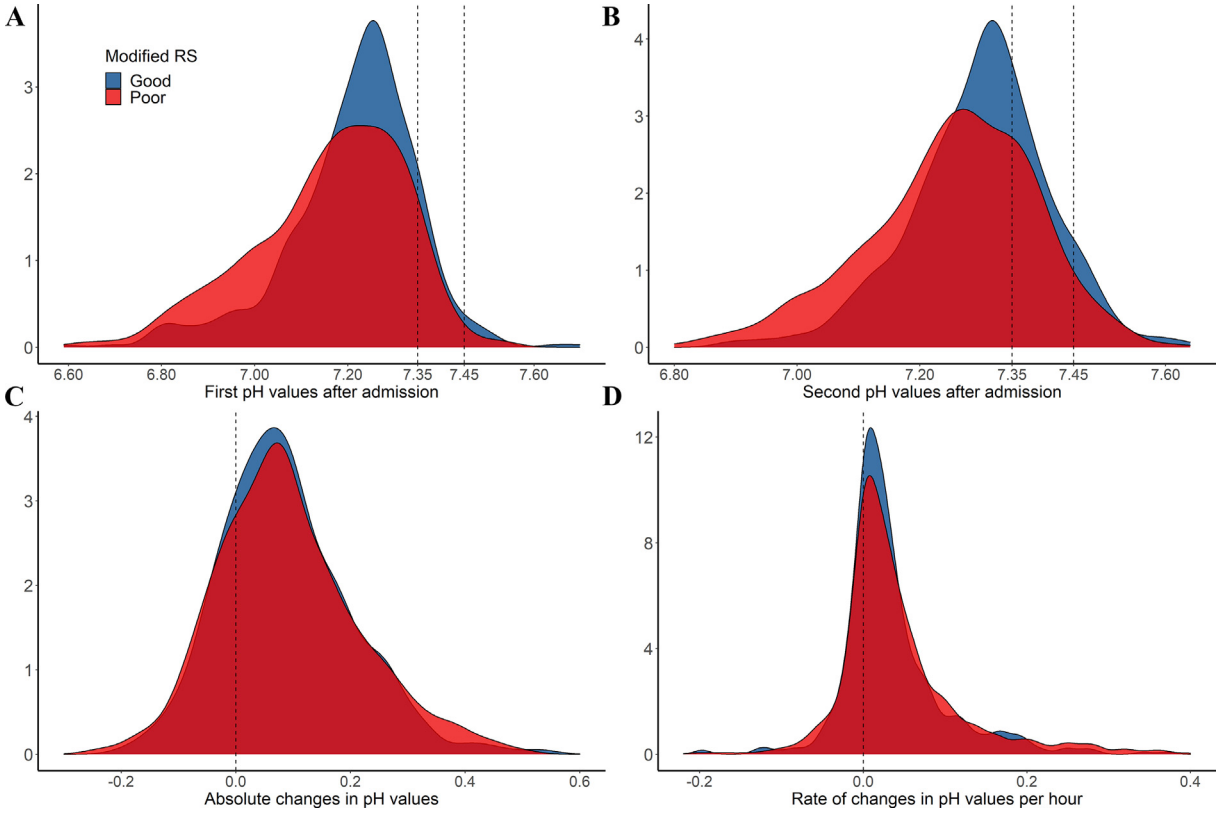


Fig. 1 – (A) Distribution of the first pH value after admission between patients with poor and good neurological outcome; (B) Distribution of the second pH value after admission between patients with poor and good neurological outcome; (C) Distribution of absolute change in pH values (the difference between the first and second pH value) between patients with poor and good neurological outcome; (D) Distribution of rate of changes in pH values (the difference between the first and second pH value divided by the time interval) between patients with poor and good neurological outcome.

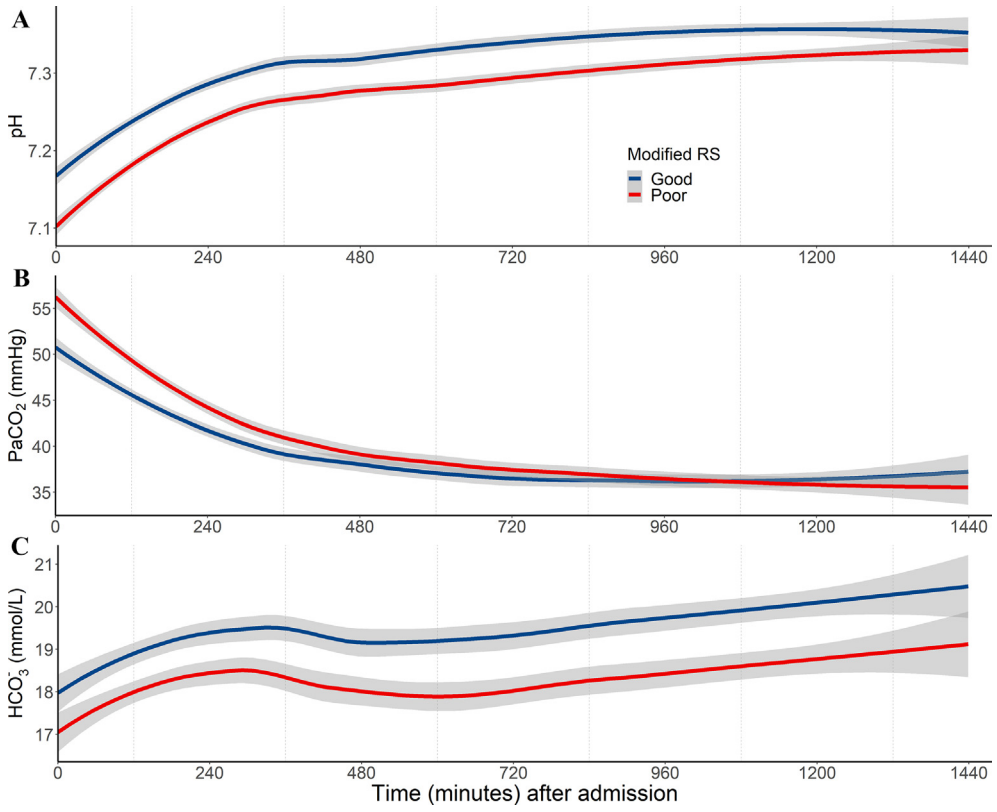
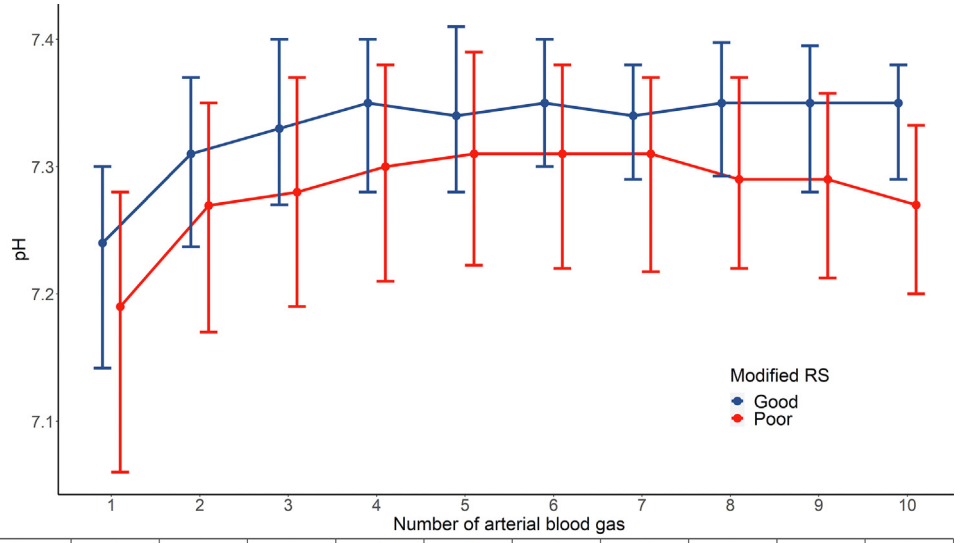


Fig. 2 – Local weighted scatter smoothing (LOWESS) method to detect the trend of pH values, $PaCO_2$, and HCO_3^- during the first 24 h after admission between patients with good and poor neurological outcomes.



Number	1	2	3	4	5	6	7	8	9	10
No. at risk	1388	1388	1177	889	610	417	254	155	85	57
mRS ≤3	514	514	449	332	232	154	94	54	27	17
mRS >3	874	874	728	557	378	263	160	101	58	40
P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	0.023	0.005

Fig. 3 – Comparison of HCO_3^- for different numbers of arterial blood gas tests between patients with good (mRS $\leq$ 3) and poor (mRS > 3) neurological outcomes.

Table 3 – Adjusted odds ratio of pH values for good neurological outcome and hospital mortality.

Variables	Adjusted ORs (95% CI) for good functional outcome	*P* value	Adjusted ORs (95% CI) for hospital mortality	*P* value
The first pH after admission	3.81 [1.60, 9.24]	0.003	0.12 [0.05, 0.28]	<0.001
The second pH after admission	9.54 [3.45, 26.87]	<0.001	0.03 [0.01, 0.09]	<0.001
Absolute changes between the first and second pH	1.58 [0.58, 4.30]	0.368	0.60 [0.23, 1.55]	0.287
Relative changes between the first and second pH	1.03 [0.96, 1.11]	0.399	0.97 [0.91, 1.04]	0.351
Rate of changes in pH per hour	0.98 [0.33, 2.71]	0.974	1.61 [0.59, 4.71]	0.367

The covariates used for the multivariable models included age, sex, bystander witness, initial rhythm (shockable or non-shockable), time to ROSC, doses of epinephrine, and use of sodium bicarbonate.

CI confidence of interval, OR odds ratio, ROSC return of spontaneous circulation.

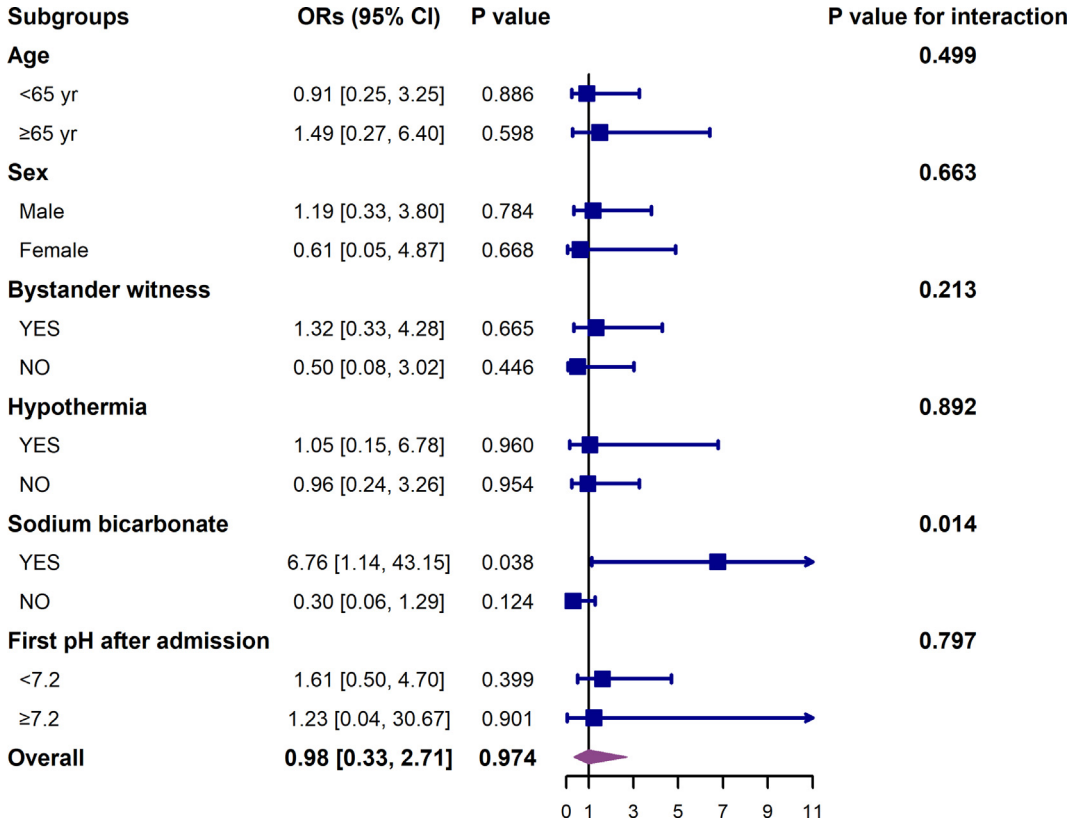


Fig. 4 – The association between the decreased rate in $PaCO_2$ and functional outcome in different subgroup of factors (age, sex, bystander witness, shockable rhythm, first $PaCO_2$, first pH value after admission, and $PaCO_2$ drop > 50%). The interaction effect of the different factors on the relationship of decreased rate in $PaCO_2$ and functional outcome were respectively explored by the logistic regression models.

Discussion

In this secondary analysis of a randomized clinical trial, we found that for OHCA patients: (1) the abnormalities of pH values, especially acidosis, were very common after ROSC; (2) the pH values were gradually increased after admission for patients with both good or poor neurological outcomes, especially during the first 6 h; (3) higher pH value during the first 24 h was associated with good neurological outcome; (4) the change in pH values was not associated with neurological outcome or hospital mortality; (5) for patients with use of SB, increase in pH value was associated with good neurological outcome. The present study suggested that the pH values had important roles for predicting outcomes for the whole-time course and should be monitored frequently.

Acid-base disturbance, especially acidosis, is very common in OHCA patients after ROSC due to hypoventilation and poor tissue perfusion.[18,19] Acidosis had been implicated in mitochondrial injury, free radical reactions, necrosis of microvessels in brain, and cerebral

edema.[20] Previous studies showed the pH value was independently associated with outcomes for OHCA patients.[7,9,21] The arterial pH was one of the important variables for risk scores to predict survival of OHCA patients both as categorial or continuous variables.[10–12,22] The present study suggested that the initial and second pH values during the first 24 h were both associated with neurological outcome, which was consistent with the study of Assil et al.[8] In addition, Shin et al. reported the pH level during CPR was also an independent factor related to neurological recovery.[9] However, the cut-off points of pH value to predict outcomes were inconsistent and remained unclear.[7,10,21] The pH value may have great change during CPR or after ROSC and time of ABG may modify the relationship of pH value and outcomes.[8]

The time course of pH value showed relatively higher rate of increase at the beginning (during the first 6 h), and then gradually tended to be stable in the present study. The slope or form of curve variation between patients with good or poor outcome was similar. There was similar pattern in the time course of $PaCO_2$ and HCO_3^-. Hong et al. found that the change in arterial pH during CPR was not associated with rate of sustained ROSC in the late phase of OHCA.[23] However, arterial pH decreased during CPR while increased for post cardiac arrest care. Delta pH (final pH minus initial pH) was negatively correlated with initial pH but was not associated with neurological status at hospital discharge for patients with OHCA,[8] which was consistent with the result of our study. Cavayas et al. found change in $PaCO_2$ after ECMO initiation is independently associated with neurological complications.[24] The pH value is the reflection of $PaCO_2$ and HCO_3^-. The relationship between pH value and outcomes is independent or dependent on combination of $PaCO_2$ and HCO_3^- was unillustrated.

According to the Henderson-Hasselbalch equation ($pH = pK + \log [HCO_3^-/(S*PaCO_2)]$, where K indicating dissociation constant and S indicating solubility of CO_2), change in pH values is dependent on the ratio of HCO_3^- and $PaCO_2$.[13] Despite prehospital resuscitation, decreased ventilation and perfusion contributed to the disturbance of acid-base, including a combination of metabolic and respiratory acidosis. The main cause of metabolic acidosis is lactic acidosis, which could neutralize and decrease HCO_3^-. Both $PaCO_2$ and HCO_3^- were associated with outcomes for OHCA patients.[25] Patients with good or poor neurological outcome had similar $PaCO_2$ during the first 24 h except for the initial ABG result, which implicated that the pH value was mainly due to the difference of HCO_3^- after the early phase when hypercapnia may largely contribute to the acidosis.

SB had been recommended based on the theoretically correction of metabolic acidosis.[26] However, the guidelines reserved SB only for specific situations (hyperkalemia, pre-existing metabolic acidosis, and tricyclic antidepressant intoxication) due to the lack of beneficial evidence and adverse effects since 2005.[27–29] There was an interesting finding in the subgroup analysis that higher rate of change in pH values was associated with good neurological outcome in patients with SB infusion but not in patients without SB. Except for patients with SB had prolonged CPR duration and lower percentage of shockable rhythm, timely ABG analysis could also contribute to the interaction effect (S-Table 1). The timely ABG analysis could be benefit for management of post cardiac arrest patients. One double-blind, randomized, placebo-controlled pilot study found SB had significant effect on pH and bicarbonate levels.[30] However, it was a pity that the time and amount of SB were not recorded in our study, which limited the explanation of SB on change of pH values.

There were several obvious limitations to this study. First, the study was retrospectively designed. There were some potential unmeasured confounders. The time and frequency of ABG tests were different among patients. This study design could only show statistical association but not causality between pH values and outcomes for OHCA patients. Second, although the multivariable logistic regression models were used to adjust for potential confounders, many potential confounding factors could lead to biased results. For example, patients with history of chronic kidney disease (CKD) may had metabolic acidosis, which could influence the results. Third, patients without ABG or with one ABG test were excluded, which could affect outcomes. Fourth, the first and second ABG tests were used to calculate the rate of change in pH value, which may underestimate the real dynamic course of pH level. The monitor of pH value was not real-time, and the perturbation may exist between the first and second ABG time interval. Last, the dataset included patients with shock-refractory ventricular fibrillation or pulseless ventricular tachycardia, who had high percentage of shockable rhythm and good functional outcome. The external extrapolation of the present study should be cautious.

Conclusions

For OHCA patients with ROSC, abnormalities in pH values were very common, with higher pH values leaning to the reference area indicating good neurological outcome. However, the change in pH values was not associated with outcomes.

Availability of data and materials

The data were available in the Biologic Specimen and Data Repository Information Coordinating Center (https://biolincc.nhlbi.nih.gov).

Ethics approval and consent to participate

The institutional review board (IRB) approved the protocol and informed consent documents at each participating sites. The ethics of secondary analysis of the data was exempted due to the retrospective design and de-identification.

CRediT authorship contribution statement

Dawei Zhou: Writing – review & editing, Writing – original draft, Software, Resources, Methodology, Conceptualization. **Yi Lv:** Writing – original draft. **Chao Wang:** Writing – review & editing, Conceptualization. **Dan Li:** Methodology.

Declaration of competing interest

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

Appendix A. Supplementary material

Supplementary material to this article can be found online at https://doi.org/10.1016/j.resplu.2024.100650.

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