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Electrolyte affecting in-hospital cause mortality in patients with cardiac arrest within 30 days: a retrospective study based on the MIMIC-IV database

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Abstract

Background: Electrolyte imbalance is closely related to the occurrence and prognosis of cardiac arrest. However, current research mainly focuses on the three ions—sodium, potassium, and calcium—while lacking attention to magnesium ions, chloride ions, bicarbonate ions, and phosphate ions. Therefore, we plan to explore the effects of electrolytes on the 30-day in-hospital mortality rate of patients with cardiac arrest based on the Medical Information Mart for Intensive Care IV (MIMIC-IV) database.

Method: Data were collected from the MIMIC IV database version 3.0 (v3.0) on electrolyte levels and 30-day in-hospital mortality rates of hospitalized patients with "cardiac arrest" from 2008 to 2022. Cox regression analysis was used to identify variables that affect the 30-day mortality rate of patients. Finally, the Kaplan-Meier curve was used in this study to further explore the effects of electrolytes on the 30-day mortality rate of patients.

Result: A total of 1491 patients who experienced cardiac arrest were included in this study. Cox regression analysis showed a correlation between age, calcium ions, bicarbonate ions, chloride ions, phosphate, and the 30-day in-hospital mortality rate in patients. The Kaplan-Meier curve further revealed that patients with advanced age, low calcium ion concentration, low chloride ion concentration, low bicarbonate concentration, and high phosphate concentration had poor prognoses.

Conclusion: Levels of bicarbonate ions, chloride ions, and inorganic phosphate at admission were associated with mortality on day 30 of admission.

Keywords: Bicarbonate ions, Cardiac arrest, Chloride ions, MIMIC-IV, Phosphate

Background

Cardiac arrest occurs when the ejection function of the heart suddenly stops, resulting in an abrupt interruption of blood flow to the brain,[1] leading to brain damage in the patient. If circulation is not restored in time, the patient will die within minutes.[2] Sudden cardiac death accounts for 20% of deaths among hospitalized patients in Western society.[3] Therefore, it is of great significance to search for biomarkers associated with poor prognosis in patients with cardiac arrest.[4]

In recent years, research has shown that abnormal cellular and molecular mechanisms during myocardial remodeling after myocardial infarction are closely related to an increased risk of myocardial arrest, such as changes in ion channel function.[5–8] To date, significant progress has been made in research on electrolyte imbalance and cardiac arrest. For example, abnormal blood potassium levels can affect the excitability, autonomy, conductivity, and contractility of myocardial cells, leading to arrhythmia and, in severe cases, cardiac arrest.[9,10] Abnormal blood calcium levels can also cause changes in myocardial excitability and autonomy and may induce heart conduction block, potentially leading to cardiac arrest.[11] Serum chloride is also an important electrolyte for maintaining fluid balance and acid-base balance. Although chloride imbalance does not directly cause cardiac arrest, it may affect neurological function.[12]

It is evident that electrolytes play a crucial role in maintaining the normal physiological function of the heart, and electrolyte imbalance is closely related to cardiac arrest. However, current research mainly focuses on the three ions—sodium, potassium, and calcium—while neglecting bicarbonate ions, magnesium ions, chloride ions, and phosphate ions. Therefore, we plan to further investigate the effects of bicarbonate ions, magnesium ions, chloride ions, and phosphate on the prognosis of patients with cardiac arrest.

Methods

Database

The data for this study come from the Medical Information Mart for Intensive Care IV (MIMIC-IV) version 3.0, a publicly available, large-scale, single-center intensive care database. This database

The datasets generated during and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

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contains hospitalization information of adult patients at Beth Israel Deaconess Medical Center (BIDMC, Boston, MA, USA) from 2001 to 2022. It mainly includes records of demographics, vital signs, laboratory tests, medication treatments, and complications. Data are entered and extracted according to the 10th edition of the International Classification of Diseases Code codes.[13] One of the authors of this study (Tang J) completed the CITI "Data or Specimens Only Research" course (Record ID: 12467279) and gained access to the MIMIC database. Because the MIMIC database records patient information anonymously, there is no requirement for ethical consent.[14]

Study population

The subjects of this study are cardiac arrest patients in the MIMIC database. We queried the disease codes (I462, I468, I469, I9712, I97120, I97121, I9771, I97710, I97711, O0336, O0386, O0486, O0736, O0881, O2911, O29111, O29112, O29113, O29119) of patients with cardiac arrest in the 10th edition of the International Classification of Diseases Code. Cardiac arrest patients in the MIMIC database were identified using the above disease classification codes,[15] and patient information was acquired according to the following inclusion and exclusion criteria (Fig. 1).

Inclusion criteria are as follows: (1) patients diagnosed with "cardiac arrest"; (2) patients admitted for the first time; (3) patients over 18 years old; and (4) patients with electrolyte reexamination on the first day of admission.

Exclusion criteria are as follows: (1) patients who died outside the hospital; (2) patients with a history of cardiac arrest; and (3) patients with no 30-day follow-up records.

Data extraction and processing

This study used the Navacity tool for data extraction and management. Patient information for cardiac arrest was screened based on the above exclusion criteria. Electrolyte data (serum sodium, serum potassium, serum magnesium, serum calcium, serum chloride, and phosphate) and the 30-day mortality rate after admission were extracted for analysis. The data were imported into IBM SPSS statistical software version 25.0 (IBM Corp, Armonk, NY, USA). Missing electrolyte values (<10%) were replaced with the mean values corresponding to each sequence.

The data were cleaned and coded, and electrolyte data were divided into three groups according to the quartiles. The 25th and 75th percentiles were used as cutoff points to classify the data into low-, middle-, and high-value groups. These groups were encoded as "1," "2," and "3." This grouping ensured that the three groups were numerically similar and clinically relevant. For example, patients can be categorized as normal, elevated, or reduced based on electrolyte levels. Finally, the coded data were reimported into SPSS for analysis.

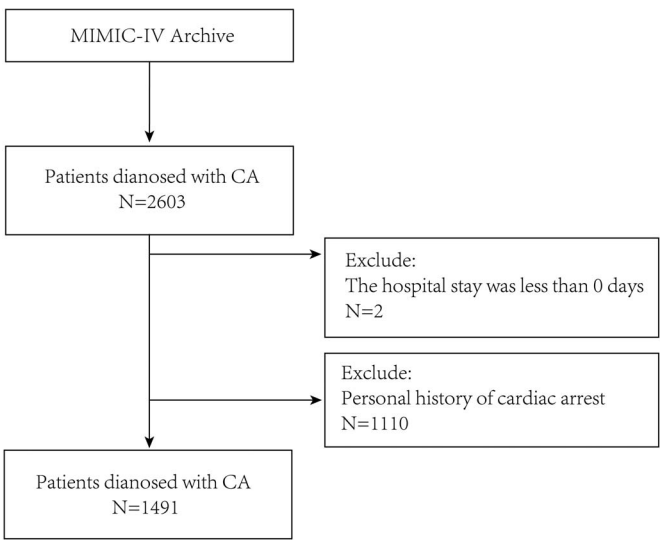

Figure 1. Extraction, inclusion, and exclusion of cardiac arrest data. CA, cardiac arrest; MIMIC-IV, Medical Information Mart for Intensive Care IV.

Statistical analysis

If the measurement data among groups followed a normal distribution, the mean ± standard deviation ($x \pm s$) was used for expression, and the independent sample t test was used for analysis between groups. If the data did not follow a normal distribution, it was expressed as the median (quartile) [M (Q25, Q75)], and the nonparametric rank sum test was used for intergroup comparison. Categorical data were expressed as the number of cases (%) [n (%)], and the χ^2 test was used for comparison between groups.

To investigate which electrolytes impact the prognosis of patients with cardiac arrest, Cox regression analysis was conducted. A two-tailed $P < 0.05$ indicated statistical significance. Finally, the Kaplan-Meier curve was used to investigate the effect of electrolytes on 30-day survival in patients with cardiac arrest.

Results

Population characteristics

A total of 1491 patients with cardiac arrest were included in this study, of which 589 (39.5%) were female. Regarding complications, 690 (46.3%) patients had congestive heart failure, and 495 (33.2%) had myocardial infarction. Cerebrovascular disease was present in 251 patients (16.8%), and 311 patients (20.9%) had chronic lung disease (Table 1). Additionally, 507 patients (34.0%) had kidney disease.

Cox regression analysis

Using univariate Cox regression, we found that variables associated with 30-day mortality in patients with cardiac arrest included age, calcium, chloride, phosphate and bicarbonate, and a two-tailed P value < 0.05 was considered statistically significant (Table 2, Table 3). Additionally, Cox regression analysis was used to investigate whether the above complications affected the 30-day mortality of patients with cardiac arrest. The results showed that complications (cardiogenic shock, nephropathy, cerebrovascular disease, chronic obstructive pulmonary disease, myocardial infarction, congestive heart failure) were not statistically associated with the mortality rate within 30 days of admission in patients with cardiac arrest ($P > 0.05$).

Kaplan-Meier curve

The survival rate predictions between different groups were compared by plotting Kaplan-Meier curves,[16] mainly using the log-rank test. A significant difference in survival rate was indicated by a two-tailed $P < 0.05$.

Previous studies have shown that age and calcium ions can affect the prognosis of patients with cardiac arrest.[17,18] We found that age was a risk factor for 30-day postadmission mortality in patients with cardiac arrest. There were statistically significant differences in 30-day mortality between group 3 (>79 years old) and group 2 (58–79 years old), as well as between group 3 and group 1 (<58 years old) ($P < 0.05$). However, there was no statistically

Table 1

Basic Characteristics and Difference Analysis

Variables	Total (N = 1491)	Survival Group (n = 756)	Death Group (n = 735)	χ^2	*P*
Gender, n (%)				0.36	0.550
Male	902 (60.50)	463 (61.24)	439 (59.73)		
Female	589 (39.50)	293 (38.76)	296 (40.27)		
Age, n (%)				20.20	<0.001
<58 years	393 (26.36)	228 (30.16)	165 (22.45)		
58–79 years	691 (46.34)	356 (47.09)	335 (5.58)		
>58 years	407 (27.30)	172 (22.75)	235 (31.97)		
Congestive heart failure, n (%)				0.41	0.523
0	801 (53.72)	400 (52.91)	401 (54.56)		
1	690 (46.28)	356 (47.09)	334 (45.44)		
Myocardial infarct, n (%)				0.05	0.827
0	996 (66.80)	507 (67.06)	489 (66.53)		
1	495 (33.20)	249 (32.94)	246 (33.47)		
Cerebrovascular disease, n (%)				4.47	0.035
0	1240 (83.17)	644 (85.19)	596 (81.09)		
1	251 (16.83)	112 (14.81)	139 (18.91)		
Chronic pulmonary disease, n (%)				2.22	0.136
0	1180 (79.14)	610 (80.69)	570 (77.55)		
1	311 (20.86)	146 (19.31)	165 (22.45)		
Renal disease, n (%)				1.74	0.187
0	984 (66.00)	511 (67.59)	473 (64.35)		
1	507 (34.00)	245 (32.41)	262 (35.65)		
Cardiogenic shock, n (%)				0.44	0.506
0	1275 (85.51)	651 (86.11)	624 (84.90)		
1	216 (14.49)	105 (13.89)	111 (15.10)		
Chloride, n (%)				13.69	0.001
<98	391 (26.22)	167 (22.09)	224 (30.48)		
98–106	706 (47.35)	375 (49.60)	331 (45.03)		
>106	394 (26.43)	214 (28.31)	180 (24.49)		
Bicarbonate, n (%)				24.86	<0.001
<18	408 (27.36)	164 (21.69)	244 (33.20)		
18–24	670 (44.94)	368 (48.68)	302 (41.09)		
>24	413 (27.70)	224 (29.63)	189 (25.71)		
Calcium, n (%)				7.19	0.027
<2	374 (25.08)	168 (22.22)	206 (28.03)		
2–2.25	732 (49.09)	391 (51.72)	341 (46.39)		
>2.25	385 (25.82)	197 (26.06)	188 (25.58)		
Potassium, n (%)				10.76	0.005
<3.2	390 (26.16)	190 (25.13)	200 (27.21)		
3.2–5.0	700 (46.95)	385 (50.93)	315 (42.86)		
>5.0	401 (26.89)	181 (23.94)	220 (29.93)		
Sodium, n (%)				16.07	<0.001
<136	453 (30.38)	202 (26.72)	251 (34.15)		
136–141	573 (38.43)	326 (43.12)	247 (33.61)		
>141	465 (31.19)	228 (30.16)	237 (32.24)		
Phosphate, n (%)				28.60	<0.001
<3.8	400 (26.83)	222 (29.37)	178 (24.22)		
3.8–4.7	702 (47.08)	382 (50.53)	320 (43.54)		
>4.7	389 (26.09)	152 (20.11)	237 (32.24)		
Magnesium, n (%)				7.28	0.026
<1.8	409 (27.43)	224 (29.63)	185 (25.17)		
1.8–2.3	700 (46.95)	359 (47.49)	341 (46.39)		
>2.3	382 (25.62)	173 (22.88)	209 (28.44)		

significant difference in 30-day mortality between group 1 and group 2 (P = 0.073). The Kaplan-Meier curve indicated that age group 3 had the worst survival prognosis.

For calcium ions, there was a significant difference in 30-day mortality between group 1 (≤2 mmol/L) and group 2 (2–2.25 mmol/L) (P < 0.001), but no significant difference among other groups (P > 0.05). According to the survival analysis curve, group 1 had a much lower prognosis than groups 2 and 3.

Regarding chloride ions, 30-day mortality differed between group 1 (<98 mmol/L) and group 2 (98–106 mmol/L), and between group 1 and group 3 (>106 mmol/L). However, there was no significant difference between group 2 and group 3 (P = 0.849). Kaplan-Meier curve analysis showed that patients in group 1 had the worst survival prognosis.

For phosphate, the 30-day mortality of group 1 (≤0.8 mmol/L) and group 3 (>1.25 mmol/L), and between group 2 (0.8–1.25 mmol/L) and

Table 2

Independent Variable Univariate Cox Regression Analysis of 30-Day Mortality after Cardiac Arrest

Variables	*b*	SE	*Z*	*P*	HR (95% CI)
Gender					
Male					1.000 (Reference)
Female	0.068	0.075	0.900	0.368	1.070 (0.923–1.240)
Age					
<58 years					1.000 (Reference)
58–79 years	0.168	0.095	1.770	0.077	1.183 (0.982–1.426)
>79 years	0.414	0.102	4.076	<0.001	1.513 (1.240–1.847)
Magnesium					
<1.8					1.000 (Reference)
1.8–2.3	0.110	0.091	1.202	0.229	1.116 (0.933–1.335)
>2.3	0.291	0.101	2.885	0.004	1.338 (1.098–1.631)
Phosphate					
<3.8					1.000 (Reference)
3.8–4.7	0.040	0.094	0.430	0.667	1.041 (0.867–1.250)
>4.7	0.516	0.099	5.201	<0.001	1.676 (1.379–2.035)
Sodium					
<136					1.000 (Reference)
136–141	−0.324	0.090	−3.609	<0.001	0.724 (0.607–0.863)
>141	−0.093	0.091	−1.027	0.305	0.911 (0.763–1.088)
Potassium					
<3.2					1.000 (Reference)
3.2–5.0	−0.168	0.090	−1.854	0.064	0.846 (0.708–1.010)
>5.0	0.125	0.098	1.278	0.201	1.133 (0.936–1.372)
Calcium					
<2					1.000 (Reference)
2–2.25	−0.279	0.088	−3.158	0.002	0.757 (0.637–0.900)
>2.25	−0.193	0.101	−1.912	0.056	0.825 (0.677–1.005)
Bicarbonate					
<18					1.000 (Reference)
18–24	−0.469	0.086	−5.446	<0.001	0.626 (0.528–0.741)
>24	−0.503	0.097	−5.185	<0.001	0.605 (0.500–0.731)
Chloride					
<98					1.000 (Reference)
98–106	−0.263	0.087	−3.039	0.002	0.769 (0.649–0.911)
>106	−0.280	0.100	−2.798	0.005	0.756 (0.621–0.919)
Cardiogenic shock					
0					1.000 (Reference)
1	0.095	0.103	0.927	0.354	1.100 (0.899–1.346)
Renal disease					
0					1.000 (Reference)
1	0.048	0.077	0.629	0.529	1.050 (0.903–1.221)
Cerebrovascular disease					
0					1.000 (Reference)
1	0.142	0.094	1.511	0.131	1.153 (0.959–1.387)
Pulmonary disease					
0					1.000 (Reference)
1	0.109	0.088	1.235	0.217	1.115 (0.938–1.326)
Myocardial infarct					
0					1.000 (Reference)
1	−0.006	0.078	−0.075	0.940	0.994 (0.853–1.159)
Congestive heart failure					
0					1.000 (Reference)
1	−0.109	0.074	−1.477	0.140	0.896 (0.775–1.036)

CI, confidence interval; HR, hazard ratio; SE, standard error.

group 3 was statistically significant ($P < 0.05$). However, there was no significant difference between groups 1 and 2 ($P = 0.661$).

For bicarbonate, 30-day mortality was significantly different between group 1 (<18 mmol/L) and group 2 (18–24 mmol/L), and between group 1 and group 3 (>24 mmol/L) ($P < 0.001$). No significant difference was observed between group 2 and group 3 ($P = 0.681$). Kaplan-Meier curve analysis revealed that patients in group 1 had the worst survival prognosis (Table 4, Figs. 2, 3).

Discussion

Many previous studies have shown a close relationship between electrolyte imbalance and the occurrence of cardiac arrest. For example, abnormal blood potassium can cause arrhythmia and, in severe cases, can even lead to cardiac arrest. Additionally, electrolyte disorders are often symptoms of other diseases or can induce other comorbidities. For example, acute kidney injury is often accompanied by electrolyte disturbances. Previous researchers have explored whether certain electrolyte disturbances can trigger cardiac arrest, but our study takes a different approach by investigating whether electrolytes affect the prognosis of patients with cardiac arrest.

Our results showed that age, calcium ions, chloride ions, bicarbonate ions, and phosphate ions were associated with 30-day in-hospital mortality in patients with cardiac arrest. The median age was 69 years, which is close to a survey conducted in the United States (median age of 66 years).[19] Through layered regression analysis, we further validated these findings. By drawing Kaplan-Meier curves, we compared the impact of different levels of the same electrolyte on patient survival prognosis.

Regarding age, the results of this study show that the older the patient, the worse the prognosis. This is consistent with the results of a single-center cohort study.[17] Because of exhaustion, reduced systemic organ function, and diminished compensatory ability, elderly people have lower tolerance to diseases, leading to poorer prognoses.

Table 3

Multivariate Cox Regression Analysis of Independent Variables for 30-Day Mortality after Cardiac Arrest

Variables	*b*	SE	*Z*	*P*	HR (95% CI)
Age					
<58 years					1.000 (Reference)
58–79 years	0.218	0.096	2.262	0.024	1.244 (1.030–1.502)
>79 years	0.474	0.103	4.592	<0.001	1.607 (1.312–1.967)
Magnesium					
<1.8					1.000 (Reference)
1.8–2.3	0.171	0.095	1.802	0.071	1.187 (0.985–1.430)
>2.3	0.192	0.107	1.788	0.074	1.211 (0.982–1.494)
Phosphate					
<3.8					1.000 (Reference)
3.8–4.7	−0.013	0.096	−0.136	0.892	0.987 (0.818–1.191)
>4.7	0.229	0.113	2.032	0.042	1.257 (1.008–1.568)
Sodium					
<136					1.000 (Reference)
136–141	−0.117	0.103	−1.133	0.257	0.890 (0.727–1.089)
>141	0.116	0.118	0.982	0.326	1.123 (0.891–1.414)
Calcium					
<2					1.000 (Reference)
2–2.25	−0.237	0.095	−2.508	0.012	0.789 (0.655–0.949)
>2.25	−0.243	0.107	−2.276	0.023	0.784 (0.636–0.967)
Bicarbonate					
<18					1.000 (Reference)
18–24	−0.371	0.095	−3.918	<0.001	0.690 (0.573–0.831)
>24	−0.484	0.114	−4.253	<0.001	0.617 (0.493–0.771)
Chloride					
<98					1.000 (Reference)
98–106	−0.194	0.103	−1.883	0.060	0.823 (0.672–1.008)
>106	−0.449	0.138	−3.253	0.001	0.638 (0.487–0.837)

CI, confidence interval; HR, hazard ratio; SE, standard error.

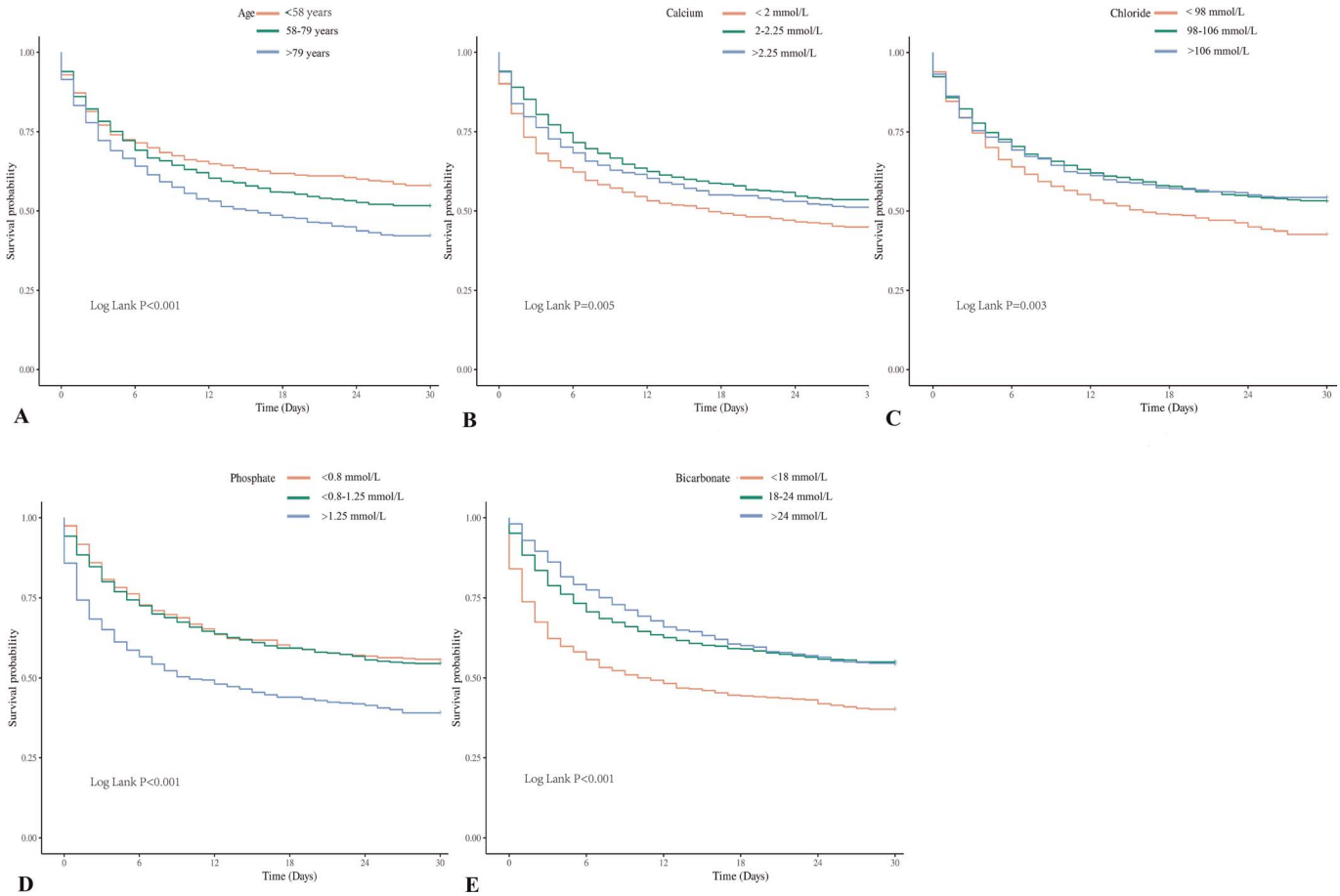

Figure 2. 30-Day survival by different levels of (A phosphate<) Age (1: <58 years, 2:58–79 years, 3: >79 years) < (B) calcium ions (1: <2 mmol/L, 2: 2–2.25 mmol/L, 3: >2.25 mmol/L) < (C) chloride ions (1: <98 mmol/L, 2: 98–106 mmol/L, 3: >106 mmol/L) < (D) phosphate (1: <0.8 mmol/L, 2: 0.8–1.25 mmol/L, 3: >1.25 mmol/L) < (E) bicarbonate (1: <18 mmol/L, 2: 18–24 mmol/L, 3: >24 mmol/L).

In terms of calcium ions, we found that the survival rate of the low-calcium group was lower than that of the medium-calcium and high-calcium groups. As is well known, calcium ions have a protective effect on myocardial cells, especially in patients with cardiac arrest, where this effect is particularly significant. For patients with hyperkalemia, clinicians often use calcium supplements or calcium gluconate to counteract the myocardial toxicity of potassium ions, thereby protecting myocardial cells.[20,21] Literature reports even suggest that hypocalcemia may be associated with hypoxic cardiac arrest.[22] Therefore, it is consistent with physiological mechanisms and clinical consensus that the survival rate of the low-calcium group is lower than that of the medium- and high-calcium groups.

Serum chloride is also an important electrolyte for maintaining fluid and acid-base balance. Our research shows that patients in the low-chloride group have the worst prognosis. This indicates that maintaining chloride ion balance is critical for patient prognosis. We believe that when sodium and potassium ions remain normal, a decrease in chloride ions leads to an increased anion gap (AG), suggesting the patient may have metabolic acidosis. Jamme et al. found that severe metabolic acidosis is common in patients with cardiac arrest and is associated with poor prognosis.[23] This may be one pathway through which serum chloride affects patient outcomes. Additionally, Kaplan-Meier curve analysis showed that patients with reduced bicarbonate levels had the worst prognosis. A decrease in bicarbonate also induces metabolic acidosis, which negatively impacts prognosis. In summary, we suggest that both chloride and bicarbonate ions affect the prognosis of cardiac arrest patients by inducing metabolic acidosis.

Table 4

Pairwise Comparisons of Survival Between Groups Using the Log-Rank Test

Variables	Comparisons	χ^2	*P*
Age (years)	<58 *vs.* 58–79	3.213	0.073
	<58 *vs.* >79	17.549	<0.001
	58–79 *vs.* >79	8.932	0.003
Calcium (mmol/L)	<2 *vs.* 2–2.25	10.576	<0.001
	<2 *vs.* >2.25	3.728	0.053
	2–2.25 *vs.* >2.25	0.945	0.331
Chloride (mmol/L)	<98 *vs.* 98–106	9.894	0.002
	<98 *vs.* >106	8.275	0.004
	98–106 *vs.* >106	0.036	0.849
Phosphate (mmol/L)	<0.8 *vs.* 0.8–1.25	0.193	0.661
	<0.8 *vs.* >1.25	28.674	<0.001
	0.8–1.25 *vs.* >1.25	32.106	<0.001
Bicarbonate (mmol/L)	<18 *vs.* 18–24	30.481	<0.001
	<18 *vs.* >24	28.243	<0.001
	18–24 *vs.* >24	0.169	0.681

P < 0.05 represents a statistically significant difference in survival between the two groups.

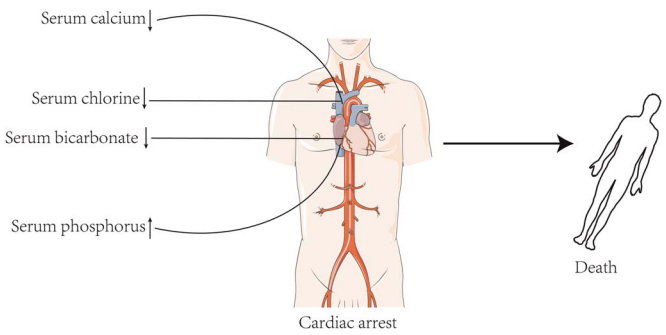

Figure 3. Effect of electrolyte changes on the prognosis of patients with cardiac arrest.

Multiple surveys have shown that serum phosphate levels increase after cardiac arrest.[24–26] A single-center retrospective observational study found that higher phosphate levels are associated with poorer prognosis in patients with cardiac arrest.[27] Our research confirms this finding: the high-phosphate group had the worst 30-day survival prognosis. Given that phosphate are involved in the rate-limiting step of ATP synthesis, we believe that increased phosphate levels imply a lower adenosine triphosphate (ATP) level, reducing the energy supply to cardiomyocytes. Additionally, lower ATP levels mean higher adenylate levels. Previous studies have shown that plasma adenylate levels are significantly higher in patients who die after resuscitation compared to survivors.[28]

Mizuguchi et al. found that serum magnesium levels upon admission may serve as a potential surrogate indicator for predicting in-hospital mortality in patients with cardiac arrest accompanied by malignant ventricular arrhythmias.[29] Reports suggest that magnesium has a protective effect on nerves.[30] Another study indicates that magnesium levels at onset are inversely proportional to neurological prognosis in survivors of cardiac arrest who receive hypothermia therapy.[31,32] However, excessively high magnesium levels can paralyze the heart.[33] Our team found no association between magnesium levels and 30-day in-hospital mortality in patients with cardiac arrest.

Our study also found that gender, sodium ions, and potassium ions were not significantly associated with the 30-day in-hospital mortality rate. Although studies have shown that hyponatremia is associated with a poorer 6-month functional prognosis in patients with cardiac arrest,[34] we found no significant relationship between serum sodium and 30-day mortality. We believe this may be because sodium and potassium are the most closely monitored electrolytes, and clinicians prevent sustained imbalances in these ions. Therefore, we suggest that sodium and potassium imbalances may affect the short-term (3-day or 7-day) prognosis but do not significantly impact 30-day in-hospital mortality.

Electrolyte imbalance may be caused by various conditions, including kidney disease, gastrointestinal diseases,[35] and endocrine disorders.[36] Findings from a clinical trial show that targeted body temperature therapy in cardiac arrest patients can also cause electrolyte disturbances.[37] Therefore, monitoring and maintaining electrolyte balance is crucial, as imbalances may indicate other comorbidities that could impact prognosis. However, our study showed no correlation between kidney disease and 30-day patient prognosis. We believe that some kidney diseases are less severe and have minimal impact on electrolytes, resulting in no significant effect on prognosis. Only severe kidney disease, which causes substantial electrolyte fluctuations, can affect patient outcomes.

Limitations

Although we made every effort to improve our study design, certain limitations remain. First, since this is a retrospective clinical study, only statistical correlations can be identified, and specific mechanisms need to be further validated through experimental research. Additionally, as the database consists of single-center data from the United States, it remains unclear whether these findings apply to Asian and African populations. Future research should involve data collection from multiple centers to validate our conclusions.

Conclusion

This research confirmed a statistical association between chloride ions, calcium ions, phosphate, bicarbonate, and 30-day mortality in adult cardiac arrest patients. However, future clinical trials are still needed to validate our findings, along with further basic experiments to explore the specific mechanisms of how electrolyte disorders affect the prognosis of patients with cardiac arrest.

Conflict of interest statement

Hongke Zeng is an Editorial Board Member of *Emergency Critical Care Medicine*. The article was subject to the journal's standard procedures, with peer review handled independently of this Editorial Board Member and their research groups. The authors declare no conflict of interest.

Author contributions

Tang J participated in data acquisition and revisions of data analysis and interpretation. Zeng H participated in study conception and design, data interpretation, and manuscript revisions, and supervised the whole study. All authors provided their final approval for manuscript submission.

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Ethical approval of studies and informed consent

The study follows the principles of the Declaration of Helsinki, as amended in 2013. This study is based on the Critical Care Medical Information Market (MIMIC)-IV Critical Care dataset. It was approved by the institutional review boards at Beth Israel Deaconess Medical Center in Boston, Massachusetts (2001-P-001699/14) and the Massachusetts Institute of Technology (0403000206) for use in research without the need for individual patient informed consent because the data are de-identified and publicly available.

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