The Correlation between Changes in Serum Sodium and Hospital Mortality in Critically Ill Children with Acute Kidney Injury

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Abstract: For critically ill patients with acute kidney injury (AKI), early warning and intervention can be achieved through monitoring serum sodium levels. However, the normal range of serum sodium levels in AKI patients in the intensive care unit (ICU) is not yet clear. This article aims to use statistical methods to study the relationship between changes in serum sodium and hospital mortality in critically ill children with acute kidney injury. This article conducted a retrospective observational cohort study on multiple critically ill children with acute kidney injury using the China Large Pediatric Intensive Care Database (PIC) to demonstrate that serum sodium levels as an independent predictor can be used to evaluate the hospital mortality rate of critically ill children with AKI. A total of 1505 eligible critically ill children with acute kidney injury were included in the study. The research results indicate that the normal range of serum sodium in children with AKI is approximately 135 to 142mmol/L. The increase or decrease in serum sodium concentration in critically ill children with acute kidney injury admitted to ICU is associated with in-hospital mortality. Therefore, in the ICU, it is necessary to continuously monitor and evaluate the serum sodium levels of children with acute kidney injury to prevent death due to the progression of the disease.

Keywords: Serum Sodium; Intensive Care Unit; Acute Kidney Injury; Mortality Rate; Children

1. Introduction

1.1 Research Background and Significance

It is common for patients to experience serum sodium abnormalities on admission or during hospitalization in the hospital or intensive care unit. In the ICU alone, the population of patients with hyponatremia and hypernatremia can reach 20% -30%. Generally speaking, the serum sodium level of adult patients with hyponatremia is lower than 135mmol/L, which is usually associated with critical diseases such as heart failure and cirrhosis. It is an indicator of disease severity and a risk factor for poor prognosis. The serum sodium level of adult patients with hypernatremia is more than 145 mmol/L. Like hyponatremia, no matter what the level is, rapid changes may cause abnormal disease symptoms, which is also an independent indicator of incidence rate and mortality. Acute kidney injury is a common clinical acute critical illness, where decreased function can lead to electrolyte disorders, and the kidney plays a central role in sodium homeostasis. Therefore, it is particularly important to confirm the impact of different serum sodium levels on mortality in AKI patients. Previous studies have described the impact of abnormal natremia on adult patients with AKI, emphasizing the issue of significant serum sodium imbalance during the development of AKI, which contributes to short-term risk prediction. However, the normal range of serum sodium suitable for children with AKI is not yet clear. Therefore, early identification of high-risk factors and accurate prediction of prognosis during ICU hospitalization may have greater benefits for children with acute kidney injury admitted to

the ICU. Therefore, determining the normal range of serum sodium with clinical significance is an important issue for clinical doctors to make decisions when AKI children experience electrolyte imbalance.

1.2 Current Research Status at Home and Abroad

There is a correlation between serum sodium ion fluctuations and the occurrence of acute kidney injury, with sodium imbalance occurring before kidney injury and to some extent indicating kidney injury [1,2]. Acute renal injury is a serious disease that has an acute impact on renal filtration function. Among hospitalized patients, the incidence rate of AKI in ICU is the highest. A meta-analysis shows that the incidence rate of AKI in ICU is 31.7%, higher than the incidence rate of other departments [3]. AKI not only increases the mortality rate of hospitalized patients, but also causes many complications, exacerbating the condition and medical burden of critically ill patients. Therefore, the health risks brought by AKI history to patients should not be underestimated. Due to the high risk of acute kidney injury in critically ill patients, there is a certain correlation between changes in serum sodium levels and acute kidney injury. A study on hospitalized individuals with hyponatremia showed that hyponatremia is independently associated with the development of AKI, and hyponatremia increases the risk of AKI by 30% [2]. Previous investigations showed that the incidence rate of AKI in adult inpatients was 0.7% -77% [4-6], and the mortality was 14% -60% [4, 7]. A recent cross-border cross-sectional study reported that more than half of patients treated in intensive care units (ICUs) experience AKI, while the proportion in critically ill children is 26.9% [8,9]. Previous studies have described the impact of adult and hospitalized AKI patients on mortality [10-12], but limited information is available on this result in critically ill AKI patients. At present, there is no direct evidence to prove that changes in serum sodium concentration are a risk factor for hospital mortality in critically ill children with AKI. Therefore, this article explores the relationship between serum sodium levels and mortality in critically ill children with AKI.

2. Materials and Methods

2.1 Data Source and Introduction

The data was collected from the China Large Pediatric Intensive Care Database (PIC). PIC is an integrated and comprehensive clinical dataset that includes routine hospital care records from Zhejiang University School of Medicine Children's Hospital from 2010 to 2018, with a total of 13941 patients admitted to the ICU. This study included 1505 children with AKI in the intensive care unit, including 827 males and 678 females.

2.2 Study Population

Children who have been admitted to the ICU multiple times are only included in the first admission. According to the initial pROCK standard [13], patients with creatinine levels greater than 200 μ mol/L were excluded from the first measurement, patients with creatinine levels measured only once or less, patients under 1 month old, and patients who died within 7 days of hospitalization. Therefore, this paper does not include these patients. In addition, this paper excludes children who have been poisoned through the International Classification of Diseases 10 (ICD-10) code, which has been registered in the original database. The paper also excluded patients who did not have AKI. The standard diagram of nano discharge is shown in Figure 1.

2.3 Statistic Analysis

In continuous variables, normal distribution variables are represented by mean ± standard deviation (SD), while skewed distribution variables are represented by median and interquartile spacing (IQR). The categorical variables are represented by numbers and percentages. If it is a continuous variable, use Kruskal Wallis rank sum test to obtain it. If it is a categorical variable, use Fisher's exact probability test to obtain it. The Kaplan Meier curve was used to graphically display the survival rate. Use multivariate logistic regression analysis to evaluate whether biomarkers can predict mortality. Due to the potential bias caused by a lack of data, variables with missing values greater than 20% were excluded from further analysis. Multiple imputation methods were used to analyze other variables with less missing values. A p-value of<0.05 is considered statistically significant. This article conducted all statistical analysis

using the empowerstats software and R software.

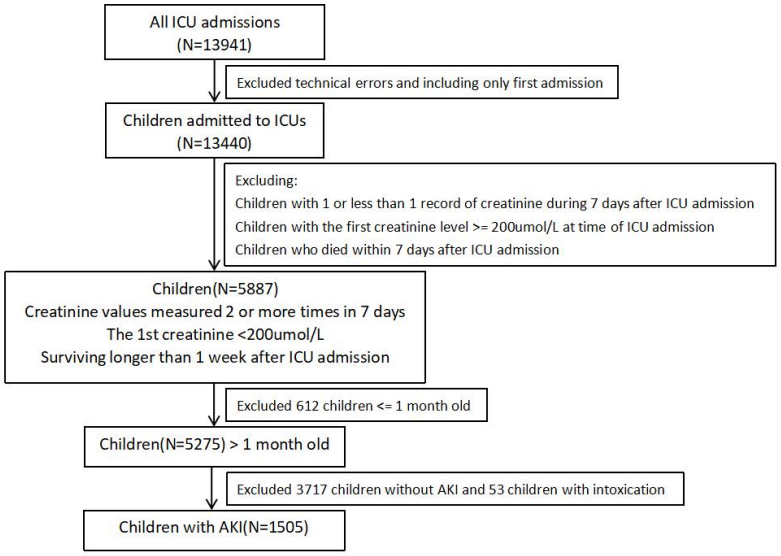

Figure 1. Patient selection flowchart. PIC, Pediatric Intensive Care database

3. Results

3.1 Smooth Curve Fitting

Based on data and statistical analysis, a smooth curve fitting, also known as a spline curve, was established as shown in Figure 2. This curve shows the nonlinear effect of serum sodium concentration on 28 day mortality in critically ill children with AKI.

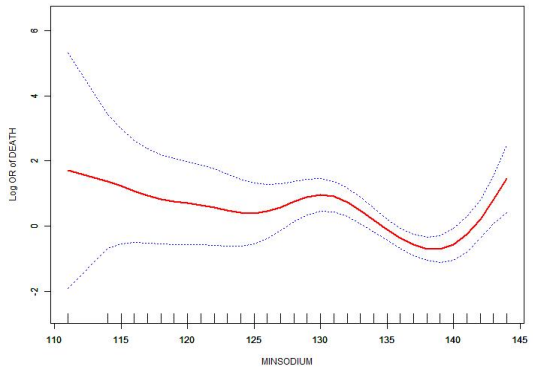

Figure 2. Smooth Curve Fitting

From the graph, it can be seen that the horizontal axis represents the serum sodium concentration, and the vertical axis represents the logarithmic mortality rate. The two blue dashed lines refer to the 95% confidence interval, while the red lines represent smooth curve fitting. When making a parallel line at zero on the vertical axis, it can be clearly seen that the range of serum sodium concentration in critically ill children is 135-142mmol/l, which is a protective factor. The hospital mortality rate is significantly reduced, while the mortality rate increases when the serum sodium concentration is below or above this range, which is a risk factor. Therefore, this paper found the normal range of serum sodium concentration in critically ill children with AKI based on the spline curve, which is 135-142mmol/l.

3.2 Study Population Description

A total of 1505 children with AKI were included in the survey, with serum sodium concentration as the independent variable and 28 day hospital mortality as the dependent variable. According to the smooth curve fitting in section 3.1, the serum sodium concentration was divided into three different groups: the group of children with normal serum sodium concentration range (135mmol/L<[Na]<=142mmol/L), the group of children with hyponatremia concentration ([Na]<=135mmol/L), and the group of children with hypernatremia concentration ([Na]>142mmol/L). Based on this, the study population description is presented in Table 1 using EasyTok statistical software. From Table 1, it can be seen that the longer a patient stays in the ICU and inpatient department, the higher the mortality rate, which is statistically significant. In terms of experimental data, the following experimental data indicate that if the serum sodium concentration is lower or exceeds the normal concentration range, the mortality rate will be higher and have statistical significance, including: MAXCL ($p<0.001$), MAXLACTATE ($p=0.002$),

MAXPT (p<0.017), MAXURA (p<0.001), MINALB (p<0.001), MINBASEEXCESS (p<0.001), MINBICARBONATE (p<0.001), MINHEMOGLOBIN (p=0.007), MINPH (p=0.003), MINPLTE (p<0.001), MINODODIUM (p<0.001), HOSPDEATH (p<0.001), GENDER (p=0.029), DEATH (p<0.001), INFE (p<0.001), PRIBLE (p<0.001), KDIGO (p<0.001), PROCK (p<0.001), DIAGNOSIS (p<0.001)

Table 1. Study Population Description

MINSODIUM categorical	<=135	>135, <=142	>142	P-value
N	673	729	103	
LOS	10.4 (15.2) 6.5 (2.0-12.8)	7.0 (9.5) 3.9 (1.7-8.0)	12.5 (18.4) 7.9 (4.8-15.7)	<0.001
AGEMONTH	43.7 (46.3) 24.6 (8.1-68.2)	43.3 (49.3) 19.5 (6.8-64.4)	37.5 (45.1) 19.5 (7.1-50.9)	0.459
TIMEDAY	13.3 (8.1) 11.6 (6.9-18.1)	12.2 (7.6) 10.5 (6.6-16.0)	13.1 (8.5) 12.1 (6.0-19.4)	0.030
MAXALT	203.8(811.6) 34.0(23.0-72.0)	130.8(737.8) 31.0 (23.0-44.0)	297.3(733.9)63.0 (34.0-191.0)	0.052
MAXAST	417.1(1331.3)67.0(37.0-180.0)	414.5(2048.3)82.0(45.0-145.0)	845.9(2621.1)150.0(71.0-405.5)	0.068
MAXTBIL	31.7 (66.1) 10.9 (6.5-24.0)	25.0 (48.9) 12.4 (7.9-21.3)	25.7 (59.7) 10.6 (6.2-20.9)	0.091
MAXCL	109.7 (6.7) 110.0 (106.0-114.0)	112.7 (5.6) 113.0 (109.0-116.0)	122.1(12.0) 119.0 (114.0-125.5)	<0.001
MAXCRE	120.6 (714.4) 51.0 (42.0-68.0)	77.0 (365.1) 49.0 (41.0-60.0)	112.0 (358.6) 66.0 (47.5-98.8)	0.322
MAXHEMATOCRIT	36.4 (6.8) 35.7 (32.0-40.0)	36.2 (6.6) 36.0 (32.3-40.0)	35.7 (6.3) 34.8 (31.9-39.7)	0.618
MAXLACTATE	3.7 (3.6) 2.6 (1.7-4.1)	3.4 (3.0) 2.6 (1.7-3.9)	4.6 (4.3) 3.3 (2.1-5.2)	0.002
MAXPT	16.8 (9.0) 14.1 (12.5-17.0)	16.2 (8.5) 14.4 (12.7-16.8)	18.9 (13.9) 15.6 (13.2-19.7)	0.017
MAXUREA	5.5 (4.3) 4.2 (3.0-6.3)	4.7 (2.7) 4.3 (3.3-5.7)	9.6 (8.3) 7.4 (4.6-11.2)	<0.001
MAXWBC	15.1 (25.8) 12.4 (8.1-17.5)	19.3 (56.9) 12.9 (9.4-17.4)	13.6 (7.0) 12.6 (8.0-17.4)	0.129
MINALB	36.0 (8.2) 36.9 (31.8-41.2)	38.2 (6.8) 39.2 (35.1-42.2)	37.5 (8.2) 38.8 (33.2-43.8)	<0.001
MINBASEEXCESS	-5.9 (5.7) -5.3 (-8.3--2.6)	-4.9 (4.7) -4.3 (-6.6--2.5)	-6.8 (6.5) -6.9 (-9.6--3.1)	<0.001
MINBICARBONATE	19.9 (4.3) 20.2 (17.9-22.4)	20.6 (3.6) 21.0 (19.2-22.5)	19.2 (5.0) 19.1 (16.8-21.9)	<0.001
MINHEMOGLOBIN	100.8 (22.5) 100.0 (88.0-114.0)	100.7 (20.5) 102.0 (89.0-114.0)	93.8 (21.5) 93.0 (81.0-107.0)	0.007
MINPH	7.3 (0.1) 7.3 (7.3-7.4)	7.3 (0.1) 7.3 (7.3-7.4)	7.3 (0.1) 7.3 (7.2-7.4)	0.003
MINPLT	220.4(145.0)213.0(120.0-305.0)	199.7(130.0)177.0(110.0-263.0)	162.5(132.8) 138.0 (65.0-228.0)	<0.001
MINPO2	90.6 (47.4) 82.0 (49.3-128.0)	96.0 (53.8) 85.7 (47.4-137.0)	90.5 (53.4) 73.2 (42.9-126.5)	0.121
MINPOTASSIUM	3.3 (0.6) 3.3 (3.0-3.7)	3.2 (0.5) 3.2 (2.9-3.6)	3.2 (0.6) 3.2 (2.7-3.6)	0.077
MINSODIUM	131.1 (4.8) 132.0 (130.0-134.0)	138.2 (1.8) 138.0 (137.0-140.0)	147.8 (7.2) 145.0 (143.0-148.0)	<0.001
HOSPDEATH				<0.001
0	620 (92.1%)	712 (97.7%)	92 (89.3%)	
1	53 (7.9%)	17 (2.3%)	11 (10.7%)	
GENDER				0.029
0	393 (58.4%)	375 (51.4%)	59 (57.3%)	
1	280 (41.6%)	354 (48.6%)	44 (42.7%)	
DEATH				<0.001
0	631 (93.8%)	716 (98.2%)	93 (90.3%)	
1	42 (6.2%)	13 (1.8%)	10 (9.7%)	
INFE				<0.001
0	469 (69.7%)	559 (76.7%)	64 (62.1%)	
1	204 (30.3%)	170 (23.3%)	39 (37.9%)	
PRIFLE				<0.001
0	1 (0.1%)	2 (0.3%)	0 (0.0%)	
1	250 (37.1%)	354 (48.6%)	26 (25.2%)	
2	340 (50.5%)	335 (46.0%)	65 (63.1%)	
3	82 (12.2%)	38 (5.2%)	12 (11.7%)	
KDIGO				<0.001
0	27 (4.0%)	29 (4.0%)	7 (6.8%)	
1	224 (33.3%)	327 (44.9%)	19 (18.4%)	
2	293 (43.5%)	301 (41.3%)	53 (51.5%)	
3	129 (19.2%)	72 (9.9%)	24 (23.3%)	
PROCK				<0.001
1	501 (74.4%)	629 (86.3%)	56 (54.4%)	
2	86 (12.8%)	58 (8.0%)	28 (27.2%)	
3	86 (12.8%)	42 (5.8%)	19 (18.4%)	

DIAGNOSIS				<0.001
0	32 (4.8%)	26 (3.6%)	2 (1.9%)	
1	59 (8.8%)	73 (10.0%)	14 (13.6%)	
2	133 (19.8%)	303 (41.6%)	14 (13.6%)	
3	45 (6.7%)	29 (4.0%)	3 (2.9%)	
4	18 (2.7%)	16 (2.2%)	5 (4.9%)	
5	14 (2.1%)	11 (1.5%)	2 (1.9%)	
6	7 (1.0%)	3 (0.4%)	0 (0.0%)	
7	8 (1.2%)	4 (0.5%)	1 (1.0%)	
8	8 (1.2%)	6 (0.8%)	1 (1.0%)	
9	85 (12.6%)	78 (10.7%)	10 (9.7%)	
10	264 (39.2%)	180 (24.7%)	51 (49.5%)	

Note: LOS: length of ICU stay; TIMEDAY: length of hospital stay; MAXALT: max alanine transaminase; MAXAST: max aspartate transaminase; MAXTBIL: max total bilirubin; MAXCL: max chloride; MAXCRE: max creatinine; MAXPT: prothrombin time; MAXWBC: white blood cell; MINALB: albumin; MINPLT: platalets; MINPO2: partial pressure of oxygen; INFE: infection etiology of AKI; PRIFLE: pediatric risk, injury, failure, loss, end stage renal disease; KDIGO: kidney disease improving global outcome; PROCK: pediatric reference change value optimizad for AKI in children.

Table results: Mean (SD) Median (Q1-Q3)/N (%)

P-value *: If it is a continuous variable, use Kruskal Wallis rank sum test to obtain it. If the counting variable has a theoretical number<10, use Fisher's exact probability test to obtain it.

This table was generated using EasyTok statistical software (www.empowerstats. com) and R software on July 26, 2023.

3.3 Kaplan Meier Curve

The Kaplan Meier method is currently the most commonly used method for survival analysis, proposed by Kaplan and Meier, commonly referred to as the KM method. Kaplan Meier survival analysis is a univariate survival analysis that combines the survival time and termination status of patients to compare and analyze the survival status of multiple groups of patients. The Kaplan Meier survival curve is commonly used, which can intuitively reflect the survival differences of patients under different conditions. This article uses the Kaplan Meier (KM) curve to explore the impact of different serum sodium levels on mortality, as shown in Figure 3. The vertical axis of the curve represents the probability of survival of critically ill children with AKI from the beginning of the experiment to a specific time point, and the horizontal axis represents the time axis representing 28 days. According to the previous smooth curve fitting, the serum sodium concentration was divided into three different groups. It can be clearly seen that when the blood sodium concentration range is between 135 and 142mmol/L, the mortality rate is lower than other blood sodium concentrations. When the serum sodium concentration is greater than 142mmol/L, the mortality rate is significantly higher than other blood sodium concentrations. When the serum sodium concentration is less than 135mmol/L, the mortality rate is also significantly increased, and the p-value is 0.00012, which is less than 0.05, It has statistical significance.

3.4 Univariate Analysis

Due to sample size limitations, when conducting multifactor analysis, it is often necessary to first conduct single factor screening, and then conduct multi factor analysis on the selected single factors, which is the "single before many" principle. Single factor analysis refers to the analysis of the degree of influence of a certain factor at different levels on an independent variable at a time point. The experimental treatment is only in one direction, aiming to determine the potential confounding factors of the model. The principle is to see which factors are related to mortality, and the p-value is less than 0.05, so as to make variable choices for later adjustment of the model, as shown in Table 2. From Table 2, it can be seen that LOS, GENDER, MAXLACTATE, MAXPT, MINBASEEXCESS, MINBICARBONATE, MINPH, MINPO2, MINSODIUM, PROCK can all be used for subsequent confounding factor adjustments.

3.5 Multivariate Analysis

Multivariate analysis is the analysis of whether an independent variable is influenced by one or more factors or variables, that is, the study of whether two or more control variables have a significant impact on outcome variables.

Before conducting multivariate analysis, in order to more accurately identify which adjustment variables are more suitable for evaluating the independent effect of risk factors on outcome variable mortality, a covariate screening is needed. The obtained variables include MAXCL, MAXLACTATE, MINALB, MINBICARBONATE, MINPH, MINPO2, PRILE, KDIGO, PROCK, DIAGNOSIS.

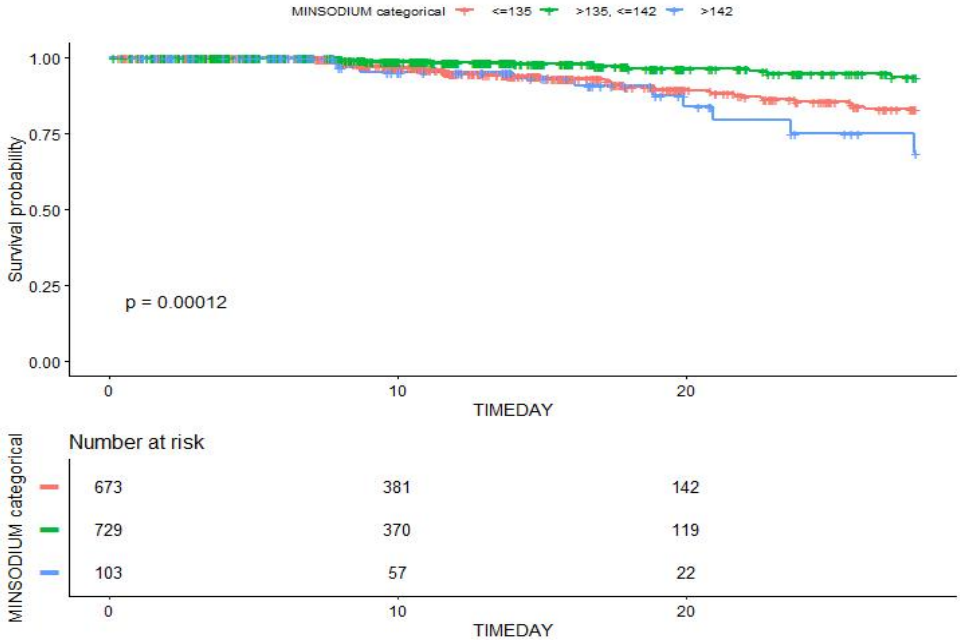

Figure 3. Kaplan Meier curve

Table 2. Univariate Analysis

Statistics	DEATH	P-value
LOS	8.9 ± 13.2	1.0 (1.0, 1.0) 0.004
GENDER		
0	827 (55.0%)	1.0
1	678 (45.0%)	0.3 (0.2, 0.6) <0.001
AGEMONTH	43.1 ± 47.7	1.0 (1.0, 1.0) 0.222
TIMEDAY	12.8 ± 7.9	1.0 (1.0, 1.1) 0.061
INFE		
0	1092 (72.6%)	1.0
1	413 (27.4%)	1.6 (0.9, 2.7) 0.082
MAXALT	174.9 ± 772.4	1.0 (1.0, 1.0) 0.107
MAXAST	445.2 ± 1816.7	1.0 (1.0, 1.0) 0.129
MAXTBIL	28.1 ± 58.0	1.0 (1.0, 1.0) 0.265
MAXCL	112.0 ± 7.4	1.0 (1.0, 1.0) 0.838
MAXCRE	98.9 ± 549.3	1.0 (1.0, 1.0) 0.745
MAXHEMATOCRIT	36.3 ± 6.6	1.0 (1.0, 1.0) 0.995
MAXLACTATE	3.6 ± 3.4	1.2 (1.1, 1.2) <0.001
MAXPT	16.6 ± 9.2	1.0 (1.0, 1.0) 0.002
MAXUREA	5.4 ± 4.2	1.0 (1.0, 1.1) 0.141
MAXWBC	17.0 ± 43.2	1.0 (1.0, 1.0) 0.786
MINALB	37.2 ± 7.6	1.0 (0.9, 1.0) 0.092
MINBASEEXCESS	-5.5 ± 5.3	0.9 (0.9, 0.9) <0.001
MINBICARBONATE	20.2 ± 4.1	0.9 (0.8, 0.9) <0.001
MINHEMOGLOBIN	100.3 ± 21.5	1.0 (1.0, 1.0) 0.373
MINPH	7.3 ± 0.1	0.0 (0.0, 0.1) <0.001
MINPLT	206.4 ± 137.9	1.0 (1.0, 1.0) 0.453
MINPO2	93.2 ± 51.1	1.0 (1.0, 1.0) 0.002
MINPOTASSIUM	3.3 ± 0.6	0.8 (0.5, 1.3) 0.344
MINSODIUM	135.7 ± 6.2	1.0 (0.9, 1.0) 0.029
PRIFLE		
0	3 (0.2%)	1.0
1	630 (41.9%)	55196.9 (0.0, Inf) 0.983

2	740 (49.2%)	95737.0 (0.0, Inf) 0.982
3	132 (8.8%)	313122.3 (0.0, Inf) 0.980
KDIGO		
0	63 (4.2%)	1.0
1	570 (37.9%)	0.3 (0.1, 1.0) 0.053
2	647 (43.0%)	0.5 (0.2, 1.6) 0.275
3	225 (15.0%)	1.9 (0.6, 5.7) 0.239
PROCK		
1	1186 (78.8%)	1.0
2	172 (11.4%)	3.4 (1.8, 6.6) <0.001
3	147 (9.8%)	6.4 (3.6, 11.6) <0.001
DIAGNOSIS		
0	60 (4.0%)	1.0
1	146 (9.7%)	0.7 (0.2, 2.9) 0.597
2	450 (29.9%)	0.2 (0.0, 0.8) 0.023
3	77 (5.1%)	1.0 (0.2, 4.8) 0.959
4	39 (2.6%)	1.0 (0.2, 6.4) 0.977
5	27 (1.8%)	0.7 (0.1, 7.4) 0.790
6	10 (0.7%)	2.1 (0.2, 22.6) 0.537
7	13 (0.9%)	3.5 (0.5, 23.1) 0.201
8	15 (1.0%)	1.4 (0.1, 14.1) 0.798
9	173 (11.5%)	1.0 (0.3, 4.0) 0.951
10	495 (32.9%)	1.4 (0.4, 4.6) 0.622

Note: Data in the table:β (95%CI) Pvalue / OR (95%CI) Pvalue
Result variable: DEATH
Exposure variables: LOS; GENDER; AGEMONTH; TIMEDAY; INFE; MAXALT; MAXAST; MAXTBIL; MAXCL; MAXCRE; MAXHEMATOCRIT; MAXLACTATE; MAXPT; MAXUREA; MAXWBC; MINALB; MINBASEEXCESS; MINBICARBONATE; MINHEMOGLOBIN; MINPH; MINPLT; MINPO2; MINPOTASSIUM; MINSODIUM; PRIFLE; KDIGO; PROCK; DIAGNOSIS.
This table was generated using EasyTok statistical software (www.empowerstats. com) and R software on July 26, 2023.

Table 3. Multivariate Analysis

Exposure	Non-adjusted	Adjust
MINSODIUM categorical recoded		
0	1.0	1.0
1	3.7 (2.0, 6.9) <0.001	2.6 (1.3, 5.2) 0.006
2	5.9 (2.5, 13.9) <0.001	2.8 (1.1, 7.6) 0.038

Note: Data in the table:β (95%CI) Pvalue / OR (95%CI) Pvalue
Result variable: DEATH
Exposure variables: MINSODIUM categorical recoded
Non-adjusted model adjust for: None
Adjust model adjust for: MAXCL; MAXLACTATE; MINALB; MINBICARBONATE; MINPH; MINPO2; PRIFLE; KDIGO; PROCK; DIAGNOSIS.
This table was generated using EasyTok statistical software (www.empowerstats.com) and R software on July 26, 2023.

As shown in Table 3, 0 represents the normal concentration range value, while 1 and 2 represent serum sodium concentrations below normal and above normal, respectively. From Table 3, it can be seen that whether the variable is adjusted or not, the hazard ratio of serum sodium concentrations below normal and above normal is greater than 1, and the p-value is less than 0.05, which verifies the accuracy and authenticity of our results.

4. Conclusion

The results of this study show a significant correlation between serum sodium levels and the 28 day hospital mortality rate in critically ill children with AKI, with a critical value of approximately 135-142 mmol/L. This indicates that early warning and intervention based on the increase or decrease of normal serum sodium levels in critically ill children with AKI can reduce the mortality rate. The limitations of this study lie in the small sample size and limited data included in the analysis. In the future, larger sample sizes and longer follow-up periods are needed for critically ill children in the ICU to further clarify the

relationship between serum sodium levels in children and hospital mortality, providing better guidance for clinical practice.

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