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Hyperchloremia as an Indicator for Acute Kidney Injury in Critically Ill Septic Patients

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Abstract

Background: Crystalloid solution therapy, including normal saline, is recommended for the early therapy of sepsis and septic shock. Shock is a state of circulatory failure that appears with hypotension as well as other vital sign deterioration or the presence of increased serum lactate levels. With recently proposed adverse outcomes and accusations of acute kidney injury, normal saline, which contains 154mmol/L of both sodium and chloride, has been accused of causing hyperchloremic acidosis in the past.

Aim of Study: Defining whether there is a relationship between Cl level at three different time points of the ICU, at ICU admission (Cl0), 24 hours after the ICU admission (Cl24h), and 48 hours after the ICU admission (Cl48h) and the development of acute kidney injury in critically ill patients with sepsis resuscitat-ed with isotonic saline (NaCl 0.9%).

Patients and Methods: This study included 70 ICU adult septic patients screened for eligibility.

Results: There was not a significant relationship between chloride level at three-time points: Day 0 (CL0), day 1 (CL24), and day 2 (CL48) and AKI among the studied cases (p=0.11); we found that there was a significant relationship between severity scoring systems e.g., APACHE and qSOFA with AKI on admission and 48 hours later. Regarding kidney function tests, we concluded that maintenance fluid volumes (MFV), urine output (UOP), serum lactate level, and anion gap all showed a significant rela-tionship with AKI (p<0.001). Even though PH and HCO3 showed a significant relationship with AKI (p<0.001), meanwhile; PCO2 showed a non-significant relationship with AKI (p=0.021).

Conclusion: A non-significant relationship was found between chloride level and AKI, neither on admission nor 24 or 48 hours later; chloride level could not be used as a predictor of AKI among critically ill septic patients.

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Key Words: *Crystalloids – Hyperchloremia – Chloride load – Acute kidney injury – Hyperlactatemia – Meta-bolic acidosis – Sepsis – Septic shock.*

Introduction

CRYSTALLOID solutions are commonly recommended for the early resuscitation of sepsis and are typically given to patients with septic shock. Tachycardia, hypotension, fever, and leukocytosis are frequent symptoms in patients with sepsis, whether it is suspected or confirmed. Signs of shock (such as cyanosis and cold skin) and organ dysfunction (such as oliguria, acute renal failure, altered consciousness level, etc.) occur as the severity increases. It's important to note that the presentation is generic, which means that it could resemble the signs of many other disorders (such as pancreatitis or acute respiratory distress syndrome) **[1]**. The most common fluid administered in critically ill septic patients is normal saline (NaCl, 0.9%), which might cause hyperchloremic acidosis and other potential side effects due to its high content of salt and chloride (154mmol/L). Among these hazards, kidney injury is the most concerning **[2].** A slight increase in serum creatinine (s.cr.) (0.3mg/dl) in critically ill septic patients is linked to lower long-term outcomes, increased mortality, and lengthier hospital admissions. Understanding and preventing this serious consequence may therefore be made easier by recognizing risk factors for the emergence of AKI in sepsis **[3].** Typically, sodium reabsorption comes first, then chloride reabsorption. The amount of sodium in 1 liter of renal tubule filtrate is around 140mEq. It should also contain roughly the same number of anions, primarily chloride (110mEq) as well as bi-carbonate to maintain electroneutrality (24mEq). The percentage of filtered chloride that is reabsorbed ranges from 65 to 70 percent of the total amount, which is comparable to the fractional reabsorption of salt and water **[4].** Chloride is primarily transported across cellular membranes by

electroneutral cation-Cl cotransporters that enable Cl to constantly follow cations, primarily Na and K **[5].** Thiazide-sensitive Na-Cl cotransporters, loop diuretic-sensitive Na-K-Cl cotransporters, and K-Cl cotransporters are all examples of cation-Cl cotransporters, which are transmembrane proteins **[6].** The main purpose of the study is to ascertain if the development of AKI in critically ill septic patients and serum chloride (Cl) levels at three distinct time points are independently correlated. Three CL time points were evaluated: At ICU admission (Cl0), 24 hours after an ICU stay (Cl24h), and 48 hours after an ICU stay (Cl48h). We hypothesized that the Cl level would be individually linked with AKI.

Aim of the study:

Aim of this study is to determine how hyperchloremia affects critically ill septic individuals who develop acute kidney injury.

Patients admitted to the ICU with sepsis or septic shock frequently develop an increased chloride level.

Patients and Methods

Patients' recruitment: All patients were recruited randomly from surgical intensive care unit Ain Shams University Hospitals from September 2022 to February 2023, on condition that they meet our criteria of inclusion.

Ethics: Ethical approval for this study (FMASU MS 366/2022) was provided by the Ethics committee of Ain Shams University Hospital, Abbasia, Cairo, Egypt (Chairperson Prof F. Tash) on 25/5/2022. Written informed consent was obtained from all subjects or their caregivers.

Inclusion criteria:

We enrolled 70 ICU patients admitted with severe sepsis or septic shock with age of 18 or older, a diagnosis of severe sepsis (described as both two of the four Systemic Inflammatory Response Syn-drome (SIRS) requirements plus a suspected or verified infection source with one organ dysfunction at least or a lactate level higher than 4mmol/L), or septic shock (described as hypotension induced by sepsis, tissue hypoperfusion, or a need for vasopressors).

Exclusion criteria:

Patients with hypernatremia, underlying cardiac conditions, any other cause of shock, pre-existing renal impairment, chronic use of dialysis, evidence of AKI preceding resuscitation with normal saline infusion, and patients with any of these conditions.

Procedure and technique:

All 70 adult septic Patients were resuscitated with normal saline infusion, and then developed AKI based on RIFLE criteria (where injury means an increase in serum creatinine *2 of baseline or urine output (UOP) <0.5ml/kg/h for 12 hours and were subjected to the following:

Full history including: Age, gender, body weight, diabetes mellitus, hypertension, hypothyroidism, cerebrovascular stroke (CVS), HCV, and bronchial asthma (BA).

Laboratory Work-up: The blood samples were taken for 3 consecutive points (days), all patients were sampled for; serum creatinine, serum urea level, glomerular filtration rate (GFR) a test used to estimate how much blood passes through the glomeruli each minute. The kidneys' glomeruli are microscopic filters that remove the blood's waste, chloride level, arterial blood gases to determine; pH; the ratio of bases to acids. Typically, between 7.35 and 7.45. PCO2; the partial pressure of carbon di-oxide. HCO3; the basic chemical generated from carbon dioxide is computed using the values obtained of pH and PaCO2.

Clinical assessment: Daily maintenance fluid volume (MFV), According to the Surviving Sepsis Campaign (SSC) recommendations, 25-30ml/kg were rapidly infused to maintain mean arterial pressure (MAP) of at least 65mmHg or maintained by continuous infusion of vasopressors. Shock is defined as either (1) Plasma lactate of at least 4mmol/L, (2) MAP below 65mmHg, (3) Capillary refill time defined as less than or equal to three seconds, or (4) Oliguria. The threshold for lactate was set based on the Surviving Sepsis Campaign (SSC) recommendations and evidence showing that mortality increases noticeably at lactate levels above 4mmol; nevertheless, meeting any one of these requirements was not a requirement for fluid bolus treatment. The four hypoperfusion criteria listed above were to be reassessed before a subsequent fluid bolus or at least 30 minutes later to determine the effect of the fluid bolus. urine produced (UOP).

Severity scores: A general measure of disease severity based on age, prior health conditions, and cur-rent physiological data is the acute physiological and chronic health evaluation (APACHE II) score with the lowest score is 0 and the highest score is 71; a higher score indicates a higher risk of hospital mortality. A bedside prompt called the rapid sequential organ failure assessment (qSOFA) score commonly referred to as the quick SOFA, may help identify patients with a suspected infection who are more likely to have a bad outcome outside of the intensive care unit (ICU). The scores can range from 0 to 3 points and are based on three criteria: Low blood pressure (SBP >100mmHg), increased respiratory rate (>22 breaths per minute), or disturbed mental status (>15 on the Glasgow Coma Scale). A higher risk of death or a longer stay in the intensive care unit was correlated with the presence of two or more qSOFA points close to the commencement of infection. Severity scoring systems were done on day 0 and day 2.

Patients were divided into two groups according to chloride level to determine the relation between Chloride level and AKI:

- Group (1): Hyperchloremic (cl >110).
- Group (2): Normochloemic (cl ≤110).

Statistical analysis: The Statistical Package for Social Science (SPSS) program for Windows (Standard Version 26) was used to evaluate the data. The one-sample Kolmogorov-Smirnov test was used to determine whether the data were normal, and the number and percentage were used to describe the qualitative data. Continuous variables were given as the mean SD (standard deviation) for data normality and repeated measurement. An ANOVA test was applied to compare means at various follow-up periods. Association among categorical variables was examined using the Chi-square test. Continuous data are correlated using Pearson's correlation.

The level of significance for all the aforementioned statistical tests is set at 5%. The results were deemed significant when the *p*-value was >0.05, and the more significant the results, the smaller the *p*-value that was achieved.

Results

Regarding APACHE and qSOFA scores in two points; day 0 and day 2 there was a significant relationship with AKI, APACHE and qSOFA scores were increased from day 1 to days 2 among studied patients as shown in (Tables 1,2).

According CL level in three points day 0 (CL0), day 1 (CL24), and day 2 (CL48) there was no significant relationship with AKI, increase in CL from Day 0 to Day 1 and Day 2 as shown in (Table 3).

Regarding maintenance fluid volumes (MFV) in three points day 0, day 1, and day 2 there was a sig-nificant relationship with AKI, also; there was a decrease in MFV from Day 0 to Day 1 and Day 2 as shown in (Table 4). Meanwhile, UOP in three points day 0 (on urinary catheter insertion), day 1, and day 2 showed a significant relationship with AKI, UOP increased from Day 0 to Day 1 and then de-creased from Day 1 to Day 2 as shown in (Table 4). Moreover, Serum Lactate levels in three points day 0, day 1, and day 2 showed a significant relationship with AKI, serum Lactate levels increased from Day 0 to Day 1 and Day 2, as shown in (Table 4).

According PH, and serum bicarbonate level (HCO3) in three points day 0, day 1, and day 2 there was a significant relationship with AKI, meanwhile; partial Pressure of Carbon Dioxide (PCO2) relation-ship with AKI in three points, it was not significant, as shown in (Table 5).

Table (1): APACHE among critically ill septic patients.

APACHE	Day 0	Day 2	Mean Difference (95%CI)
Mean ± SD	12.62±7.40 **ab**	21.45±6.85 **bc**	8.82
Range	4-38	9-42	(8.04-9.60)
Test of sig. *p*-value		F=354.5 *p*≤0.001*	

Table (2): qSOFA among critically ill septic patients.

CL	Day 0	Day 1	Mean Difference (95%CI)
Mean ± SD	1.74±0.97 **ab**	2.22±0.72 **bc**	0.48
Range	0-3	1-3	(0.35-0.61)
Test of sig. *p*-value		F=34.8 *p*≤0.001*	

Table (3): CL level among critically ill septic patients.

CL	Day 0	Day 1	Day2	Mean Difference (95%CI)
Mean ± SD	105.84±6.23	106.51±5.95	108.41±5.60	1.91 (0.02-3.8)
Range	88-108	94- 113	97-117	
High	0 (0%)	11 (15.7%)	16 (22.9%)	
Normal	70 (100%)	59 (84.3%)	54 (77.1%)	
Test of sig. *p*-value		F=2.61 *p*=0.11		

Table (4): MFV, UOP, and lactate among critically ill septic patients.

	Day 0	Day 1	Day2	Mean Difference (95%CI)	Test of sig. *p*-value
MFV:					
Mean ± SD	3155.7±586 **ab**	2524.57±469 **ac**	2017.25±388 **bc**	–1138.5 (-1200 – -1076)	F=1251
Range	2050-4150	1640-3320	1300-2650		$p \leq 0.001$*
UOP:					
Mean ± SD	1305.7±268 **a**	1465.7±299 **ab**	1191.4±569 **b**	-114.28 (-246.5-17.9)	F=12.58
Range	1000-2000	1100-2200	750-3850		$p \leq 0.001$*
Lactate:					
Mean ± SD	2.52±1.09 **ab**	4.26±2.02 **ac**	4.93±2.28 **bc**	2.41 (2.06-2.76)	F=162.9
Range	0.5-5	1.3-9	1.6-10.8		$p \leq 0.001$*

Table (5): ABG among critically ill septic patients.

	Day 0	Day 1	Day2	Difference	Test of sig. *p*-value
PH:					
Mean ± SD	7.31±0.07 **ab**	7.28±0.08 **a**	7.28±0.06 **b**	-0.03 (-0.04 – -0.02)	F=14.75
Range	7.07-7.42	7.09-7.41	7.13-7.40		$p \leq 0.001$*
PCO2:					
Mean ± SD	30.35±7.83 **a**	29.91±7.31 **b**	28.62±5.66 **ab**	-1.72 (-3.1--0.33)	F=3.97
Range	2-49	16-42	18-42		p=0.021*
HCO3:					
Mean ± SD	16.20±5.5 **ab**	14.88±5.0 **ac**	14.13±3.9 **bc**	-2.07 (-3.01 – -1.12)	F=12.37
Range	1-29	4.5-24	7.4-24		$p \leq 0.001$*

Discussion

Shock is a life-threatening condition of circulatory failure that most commonly presents with hypoten-sion; other compromised vital signs, and high serum lactate levels. Initial signs of shock are reversible, but they can quickly turn fatal due to multiorgan failure (MOF), a state in which multiple organs fail at once. Therefore, it is crucial that the clinician quickly determine the etiology when a patient appears with unexplained hypotension and/ or is suspected of being in shock so that the cause of shock can be corrected and proper measures and therapy can be given to avoid MOF and death **[8].** A clinical syndrome known as sepsis is characterized by widespread infection-related inflammation. Severity levels vary considerably from sepsis to septic shock. Mortality rates have been estimated to range between 10% and 40% when the shock is present, though these numbers are wide-ranging and based on the group investigated **[9].**

Typically, the most common fluid used in adult ICU patients for the early resuscitation of sepsis was normal saline (0.9% NaCl) **[10].** Acute kidney injury (AKI), which has been reported to be the most concerning adverse event, can cause hyperchloremic acidosis when normal saline, which has 154mmol/L of both sodium and chloride, is administered **[11].**

Although chloride is the second-most significant contributor to plasma tonicity and the second-most abundant anion in extracellular fluid, playing a crucial role in a variety of body processes, including acid-base homeostasis, muscle activity, osmosis, and immunomodulation **[12]**, The current data suggests that resuscitation fluid-induced hyperchloremia is linked to mortality and acute kidney damage (AKI), especially in sepsis. Experimental studies suggest that organ functions could be impacted by hyperchloremia **[2].**

In the present study, there was a significant relationship at two-time points, day 0 and day 2, between the APACHE score and AKI ($p \leq 0.001$). This result makes sense because AKI represents one of the multiple adverse outcomes of sepsis and septic shock. Also, AKI scoring systems have been recently used to predict mortality **[13].**

Conceding these results, Yessayan et al. **[14]** study showed a significant relationship between the admission APACHE-II score and AKI ($p \leq 0.0001$), in Suetrong et al. **[3]** study, there was a signifi-cant relationship between the admission APACHE-II score and AKI ($p \leq 0.001$). Also, Neyra et al. **[15].**

agreed that a significant relationship was found between the admission APACHE-II score and AKI

(p<0.001) The present study revealed that qSOFA at two-time points, day 0 and day 2, showed a sig-nificant relationship with AKI (p<0.001). Yessayan et al. [14] confirmed our results that the SOFA score had a significant relationship with AKI (p<0.0001). Meanwhile, in Commereuc et al. [2] study, in the zero-hour SOFA score, there was no significant relationship with AKI (p<0.31); the SOFA score was not repeated later, contrary to our present study.

In the present study, we employed the qSOFA as a severity score instead of the SOFA score. Comparing the qSOFA score to the SOFA score has shown it to be a useful tool for predicting in-hospital mortality in patients with severe sepsis and septic shock at a tertiary hospital ED of lower socioeconomic countries [16].

In the present study, S.Cr., serum urea, and GFR at three-time points (day 0, day 1, and day 2) were all significant to AKI (p<0.001). Nevertheless, in Commereuc et al. [2] study, zero-hour S.Cr. was not significant with AKI (p<0.21). Also, Yessayan et al. [14] clarified that baseline S.Cr. and baseline eGFR were not significant with AKI with p-values of (0.0794 and 0.1032, respectively). In the Yessayan et al. [3] study, baseline S.Cr. was based on serum creatinine (S.Cr) measurement within three months before ICU admission. Patients with AKI on admission and patients with baseline eGFR<15 ml/min/1.73 ^{m2} were excluded. Suetrong et al. [3] showed that baseline S.Cr. was not significant with AKI (p<0.49). Neyra et al. [15] revealed that baseline S.Cr. and baseline eGFR were not significant with AKI with p-values of (0.09 and 0.07, respectively) and patients with baseline eGFR <15ml/min/1.73m^2 were excluded.

In the present study, we hypothesized that AKI was related to daily chloride levels. CL was calculated at three-time points: Day 0 (CL0), day 1 (CL24), and day 2 (CL48), but interestingly, our results showed that it was not significant with AKI (p=0.11); meanwhile, Commereuc et al. [2] confirmed our results that the relationship between (CL0) and AKI was not significant (p<0.97) among AKI and non-AKI groups. Furthermore, (CLmax), the maximal chloride level, was not significant with AKI (p<0.79). Delta chloride (ΔCl) was defined as the difference between the maximal level of chloride (CLmax) and serum chloride on ICU admission (Cl0) at least >5mmol/L among AKI and non-AKI groups, (ΔCl) was found to be not significant with AKI (p 0.72) among AKI and non-AKI groups. Also, Yessayan et al. [14] revealed that the relationship between (CL0) and AKI was not sig-nificant (p<0.81); furthermore, the delta chloride (ΔCl) was defined as the difference between serum chloride at 72h (Cl72) and serum chloride on ICU admission (Cl0); (ΔCl) during the first 72h of the ICU stay was also not associated with either AKI at 72h (p<0.27) which agrees with our results.

Furthermore, in Suetrong et al. [3] study (CL0), they deduced that the relationship between (CL0) and AKI was not significant (p<0.51), which confirms our results. However, the maximal chloride (CLmax) was defined as maximal Cl in 48 hours. The delta chloride (ΔCl) was defined as the difference between the level of serum chloride at 48h (Cl48) and the level of serum chloride on ICU admission (Cl0). Both (CLmax) and (ΔCl) were significant with AKI (p<0.001), which disagrees with our results. Also, in Neyra et al. [15] study, (CL0) was found to be significant with AKI and mortali-ty (p<0.25), which disagrees with our results, while both (CL72) and (ΔCl) were not significant with AKI and mortality with p values of (0.03 and 0.003, respectively), which agrees with our results.

In the present study, maintenance fluid volume (MFV) and UOP at three-time points (day 0, day 1, and day 2) showed a significant relationship with AKI (p<0.0001). Yessayan et al. [14] confirmed our results that cumulative fluid balance (CFB72) was in a significant relationship with AKI (p<0.0005). Also, in Neyra et al. [15] cumulative fluid balance at 72 hours was in a significant relation-ship with AKI (p<0.001), which agrees with our results.

Meanwhile, In Commereuc et al. [2] Cumulative volume of fluids before zero-hour was not significant with AKI (p<0.28), in contrast, the Cumulative volume of fluids after zero-hour there was a significant relationship with AKI (p<0.001), what convoys our results. Also, Suetrong et al [3]. Disagreed with our results that resuscitation volume and UOP were not significant with AKI, with p-values of (0.71 and 0.644, respectively).

In our present study, the serum lactate level at three-time points (day 0, day 1, and day 2) was significant with AKI (p<0.001); in the Commereuc et al. [2] study, 72 hours of metabolic data was recorded. Hyperchloremia (>110mmol/L) and hyperlactatemia (>2mmol/L) were investigated separately for their metabolic effects. The presence of hyperlactatemia was significantly more common than hyperchloremia (62% versus 71% of patients, respectively; p=0.006), and metabolic acidosis was significantly more common in patients with hyperchloremia regardless of the presence of hyperlactatemia (p=0.001). Cox models were constructed to evaluate the relationship between chloride parameters, day-28 mortality, and AKI. In terms of adjusted risk for AKI and mortality, serum chloride, hyper-chloremia, maximum chloremia, and delta chloremia were not substantially related. Commereuc et al. [2] findings are consistent with several negative retrospective cohorts assessing the role of hyperchloremia in ICU patients. Meanwhile, in Suetrong et al. [3] study, serum lactate showed no significant relationship with AKI (p<0.29), which disagrees with our results.

In the present study, PH, HCO3, and anion gap at three-time points (day 0, day 1, and day 2) were in a significant relationship with AKI (p 0.001), while PCO2 at three-time points (day 0, day 1 and day 2) was not significant with AKI (p=0.021). Yessayan et al. **[14]** results showed that the base deficit had a significant relationship with AKI (p< 0.0001). The baseline deficit was calculated by subtract-ing the blood HCO3 reading obtained during ICU admission from the standard value of 24mEq/L for serum HCO3. Also, Neyra et al. **[15]** found that the base deficit had a significant relationship with AKI (p< 0.001). Both studies agreed with our results.

Nonetheless, these results must be interpreted with caution, and many limitations should be born in mind according to the observational design; other treatments or physiological factors that cannot be controlled with this approach may have had an impact on the outcomes. The study's findings are further constrained by the limited number of patients analyzed; as a result, the findings' validity is constrained, and thus the study is underpowered to identify changes in mortality. Also, novel biomarkers for AKI are not included (e.g., NGAL, KIM-1, L-FABP, etc.) due to a lack of resources and documentation for baseline serum creatinine before the study started. Also, including all fluid volume used in maintenance fluid volume (MFV) may not reflect initial resuscitation volume, so further studies are needed in the future on large sample sizes with novel biomarkers for AKI.

Conclusion:

A non-significant relationship was found between chloride level and AKI, neither on admission nor 24 or 48 hours later; increased chloride levels could not be used as a predictor of AKI among critically ill septic patients.

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فرط الكلور بالدم كمؤشر لحدوث الفشل الكلوى الحاد فى مرضى انتان الدم والصدمة الانتانية

ان الصدمة وضعف التروية الدموية للجسم هى حالة تهدد الحياة والتى تحدث بشكل شائع مع انخفاض ضغط الدم، يمكن أيضًا أن تنذر بتغييرات أخرى فى العلامات الحيوية أو وجود مستويات مرتفعة من اللاكتات فى الدم، فيما يتم تعريف الإنتان بأنه متلازمة سريرية تتميز بارتفاع مؤشرات الالتهاب بسبب العدوى، من خلال سلسلة متصلة ومتفاوتة الشدة تتراوح من مجرد الإنتان إلى الصدمة الإنتانية، عادة ما يتم العلاج باستخدام السوائل والمحاليل الوريدية فى هؤلاء المرضى ويعد المحلول الملحى العادى هو السائل الأكثر استخدامًا فى مرضى وحدة العناية المركزة البالغين ويتم النظر اليه باعتباره المتسبب فى الإصابة بالفشل الكلوى الحاد ومضاعفات اخرى قد تصل إلى الوفاة.

كانت دراستنا هذه عبارة عن دراسة قائمة على الملاحظة، أجريت فى وحدة العناية المركزة الجراحية بمستشفيات جامعة عين شمس، من سبتمبر ٢٠٢٢ إلى فبراير ٢٠٢٣ وقد أجريت على سبعين مريضاً بالغاً تبلغ أعمارهم ثمانية عش عاماً على الأقل ولم يتم تشخيص مرض الكلى المزمن لديهم سابقًا مع تشخيص تعفن الدم أو الصدمة الإنتانية كما هو موضح فى حملة الإنتان الباقية، وتم إنعاشهم باستخدام المحلول الملحى الاعتيادى، والذين أصيبوا بالفشل الكلوى الحاد والمتابعة تم استبعاد مستوى الكلوريد لمدة يومين وتم تقسيم المرضى إلى مجموعتين وفقاً لمستوى الكلوريد لتحديد العلاقة بين مستوى الكلوريد والفشل الكلوى الحاد: المجموعة الاولى: فرط كلوريد الدم (>١١٠)، المجموعة الثانية: كلوريد الدم الطبيعى (<١١٠)، وقد خضع جميع المرضى لفحص تاريخهم المرضى الكامل للتحاليل المخبرية اللازمة، وقد استنتجنا من خلال الدراسة انه كانت هناك علاقة ذات دلالة إحصائية بين درجات الخطورة السريرية مثل؛ التقييم الصحى الفسيولوجى الحاد والمزمن، درجة التقييم السريع لفشل الأعضاء المتسلسل والفشل الكلوى الحاد، والمثير للاهتمام أنه لا توجد معنوية لكلوريد الدم فيما يتعلق بحدوث الفشل الكلوى الحاد.