ORIGINAL ARTICLE

Impact of Hyperchloremia and its Relationship with Serum Cystatin C Level as a Marker of Early Acute Renal Injury During the Treatment of Diabetic Ketoacidosis

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Abstract

Background: ***There has been growing recognition of hyperchloremic metabolic acidosis as a cause of in-hospital acute kidney injury (AKI) and increased serum cystatin C as a sensitive and early indicator of renal dysfunction. This acidosis typically occurs after the administration of 0.9% sodium chloride to treat diabetic ketoacidosis (DKA).***

Aim of the work: ***Whether hyperchloremia is connected with increased rates of in-hospital AKI development, how long it takes for DKA to resolve, and how long patients spend in the intensive care unit (LOS), as well as to look at the correlation between high blood cystatin C concentrations and increased serum chloride levels, which could indicate the beginning of acute kidney injury.***

Subjects and methods: ***This prospective observational study was carried out on 60-patients with DKA admitted to Al-Azhar University Hospitals, from May 2023 till January 2025. Patients were divided randomly into: Group-A hyperchloremia Group-I (n=30) and Normochloremia Group-II (n=30).***

Results: ***Group I with hyperchloremia had a much longer time to first and final DKA resolution compared to Group II with sustained normochloremia. Compared to Group-II, which maintained normochloremia, Group-I hyperchloremia patients were more likely to experience in-hospital AKI, intensive care unit admission, and length of stay. Group I, which had hyperchloremia, had a considerably greater serum cystatin C level compared to Group II, which maintained normochloremia.***

Conclusion: ***It is commonly believed that the administration of a 0.9% sodium chloride solution during the management of DKA is the cause of hyperchloremic metabolic acidosis, which is related with the development of in-hospital AKI in patients with DKA. The duration of hospital stay and the time it takes for DKA to resolve have both been linked to this acid-base imbalance.***

Keywords: Hyperchloremic metabolic acidosis; diabetic ketoacidosis; Acute kidney injury

1. Introduction

Hyperglycemia with high anion gap metabolic acidosis owing to excessive generation of ketoacids at the price of diminished serum bicarbonate concentration characterizes diabetic ketoacidosis (DKA), a potentially fatal consequence of diabetes mellitus.[1] In patients with DKA, electrolyte imbalances, such as hyperchloremia, may be exacerbated by volume resuscitation, which typically uses saline. Hyperchloremia has not been thoroughly studied in DKA; however, there is evidence that it increases the risk of mortality and morbidity.[2] More and more people are becoming worried about using 0.9% saline since it contains sodium and chloride at levels that are over what the body can normally handle. Several potential side effects of 0.9% saline have been suggested by preclinical and early clinical studies. There is some evidence that hyperchloremia may cause acute kidney damage (AKI), according to several studies.[3] The protein cystatin C inhibits the activity of the body's own cysteine protease. It has a low molecular weight (Mw 13,343 Da).

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The management of scaphoid waist fractures needs careful evaluation and planning before surgery, and the aim of the management is to confirm reduction and a stable fixation without compromising blood supply.[6]

Undisplaced stable fractures are managed conservatively with a scaphoid cast for eight to twelve weeks, until the fracture unites. Nevertheless, this might be rejected by the young, active population wanting to participate in professional or sports-related activities.[7]

Our research aimed to compare the outcomes of management of recent scaphoidal waist fracture using kershiner wires versus screw fixation regarding: radiological and functional outcome.

2. Patients and methods

From May 2023 through January 2025, sixty patients admitted to Al-Azhar University Hospitals with DKA were the subjects of this prospective observational study. An informed permission was obtained from each patient's legal guardian, and the study was approved by the Ethics Committee of the Faculty of Medicine, Al-Azhar University, Egypt.

Inclusion criteria:

Individuals meeting the following diagnostic criteria for diabetic ketoacidosis (DKA): plasma glucose >250 mg/dL, arterial pH 7.3 or below, serum bicarbonate less than 18 mEq/L, positive urine or serum ketones, and anion gap greater than 10 mEq/L.

Exclusion criteria:

Exclusion criteria included the following: renal replacement treatment for chronic kidney disease (CKD) or end-stage renal disease (ESRD), septic shock (within the past six months), acute pancreatitis, liver illness, a history of nonanion gap acidosis, and fluids administered prior to referral.

Study Design and Participants:

Adults hospitalized with DKA were categorized into two groups according to their peak serum chloride levels throughout treatment: hyperchloremia Group I (n=30) and normochloremia Group II (n=30). The data for this study came from the patients' medical records. Serum chloride levels below or equal to 109 mEq/L were considered normochloremia, but levels above 109 mEq/L were considered hyperchloremia. Group I management using a standard fluid replacement regimen for an adult weighing 70 kg. A solution of isotonic sodium chloride (0.9% sodium chloride) is used as an initial fluid loss correction. The following is the suggested timetable for fluid restoration: In the first hour, give 1-3 liters. Give 1 liter at the two-hour mark. It is recommended to provide 1 liter over the next 2 hours, followed by 1 liter every 4 hours, or as the dehydration level and CVP readings dictate. And after 12 hours, you must reevaluate your cardiovascular health; you might need to drink more fluids. After administering more than 1 liter of sodium chloride to hypotensive patients, potassium chloride may be necessary for resuscitation. When blood sugar drops below 180 mg/dL, 5-10% dextrose with half an isotonic sodium chloride solution is used instead. Once the patient has stabilized with isotonic saline, the flow rate should be adjusted to 200-1000 mL/h with half-normal saline. This will help to counteract any losses caused by osmotic diuresis.6 Using a balanced crystalloid solution (lactated Ringer solution) in place of 0.9% sodium chloride, Group II was managed according to the same protocol.

The degree of DKA was categorized as mild when the pH ranged from 7.2 to 7.3, moderate when the pH ranged from 7.1 to 7.2, and severe when the pH was less than 7.1. Following the recommendations of the American Diabetes Association For this study, the following guidelines were considered and defined: not using bicarbonate when arterial pH is 6.9, using intravenous (IV) fluids containing dextrose within 2 hours of blood glucose levels reaching 200 to 250 mg/dL, keeping IV insulin going for 1 hour after starting subcutaneous insulin, and keeping serum potassium between 3.5 and 5.3 mEq/L with no two consecutive measurements outside of this range, separated by 2 hours.[7]

Methods:

Complete medical history (including symptoms such as fever, vomiting, diarrhea, and stomach discomfort) and insulin delivery, both correctly and incorrectly, were administered to all participants in the study. The following clinical and laboratory data were recorded at admission to the hospital: weight, heart rate, respiratory rate, neurological status as measured by the Glasgow Coma Score, urine output, mean blood pressure, and blood count, urea, creatinine, glucose, sodium, potassium, chloride, plasma bicarbonate, carbon dioxide, and the presence of ketonuria and glycosuria. These observations were made prior to treatment. An enzyme-linked immunosorbent test (ELISA) was used to determine the levels of serum cystatin C.

We measured and examined the serum cystatin C and creatinine readings all at once. After that, the following parameters were evaluated: ECG, chest x-ray, and time to recovery from DKA.

Sample size calculation:

Based on the following factors, the sample size was calculated using Epi Info STATCALC [version 7.2.5, Georgia, US]. The odds ratio is 1.04, and the two-sided confidence level is 95%. The power is 80%. Fifty people made up the final sample size determined by the Epi Info results.

Statistical analysis:

For the statistical study, we utilized SPSS v26, which was developed by IBM Inc. and is located in Chicago, IL, USA. The normality of the data distribution was verified by utilizing histograms and the Shapiro-Wilks test. Mean and standard deviation (SD) were utilized as quantitative parametric data, and a paired T-test was employed for comparison. The percentages or frequencies of the qualitative variables were then used to compare them using a Chi-square test. A two-tailed P-value<0.05 was used to determine a statistically significant result.

3. Results

Table 1. Demographic data of the studied groups.

		HYPERCHLOREMIA (N=30)	MAINTAINING NORMOCHLOREMIA (N=30)	P-VALUE
AGE (YEARS)	Mean ± SD	31.7±6.58	32.3±5.5	0.703
	Range	21-42	23-40	
SEX	Male	11(36.67%)	17(56.67%)	0.121
	Female	19(63.33%)	13 (43.33%)	
WEIGHT (KG)	Mean ± SD	81.2±7.63	79.9±7.74	0.515
	Range	71-96	63-94	
HEIGHT (CM)	Mean ± SD	164.2±5.43	166.17±5.92	0.185
	Range	156-174	155-175	
BMI (KG/M²)	Mean ± SD	30.16±2.99	29.02±3.25	0.162
	Range	25.2-35.7	22.4-34.3	

BMI: Body mass index, *: Significant as P-value≤0.05.

Age, sex, weight, height, BMI and were insignificantly different between both groups, (table 1).

Table 2. Presenting complaints of the studied groups.

		HYPERCHLOREMIA (N=30)	MAINTAINING NORMOCHLOREMIA (N=30)	PVALUE
FEVER	Yes	2(6.67%)	1(3.33%)	1
	No	28(93.33%)	29(96.67%)	
VOMITING	Yes	5(16.67%)	3(10%)	0.706
	No	25(83.33%)	27(90%)	
DIARRHEA	Yes	2(6.67%)	0(0%)	0.491
	No	28(93.33%)	30(100%)	
ABDOMINAL PAIN	Yes	4(13.33%)	3(10%)	1
	No	26(86.67%)	27(90%)	

Fever, vomiting, diarrhea and abdominal pain were insignificantly different between both groups, (table 2).

Table 3. Laboratory investigations of the studied groups.

		HYPERCHLOREMIA (N=30)	MAINTAINING NORMOCHLOREMIA (N=30)	P-VALUE
CHLORIDE (MEQ/L)	mean ± sd	104.97±5.44	88.3±5.38	<0.001*
	Range	96-115	80-98	
BICARBONATE (MEQ/L)	mean ± sd	9.8±3.36	12.3±2.93	0.003*
	Range	4-16	8-18	
CREATININE (MG/DL)	mean ± sd	0.73±0.15	0.7±0.13	0.383
	Range	0.51-0.97	0.42-0.9	
PH	mean ± sd	6.84±0.26	7.28±0.11	<0.001*
	Range	6.47-7.31	7.11-7.41	
SODIUM (MEQ/L)	mean ± sd	140.6±7.4	144.03±8.74	0.106
	Range	130-152	132-159	
POTASSIUM (MEQ/L)	mean ± sd	4.4±1.15	4.65±1.4	0.441
	Range	2.5-6.5	2.9-7.1	
TOTAL FLUID VOLUME (ML)	mean ± sd	5638.1±1358.83	3997.73±681.31	<0.001*
	Range	4196-7987	2914-5252	

*:Significant as p-value<0.05.

Chloride and total fluid volume were significantly higher in hyperchloremia group than maintaining normochloremia group, (Table 3; figures 1&2).

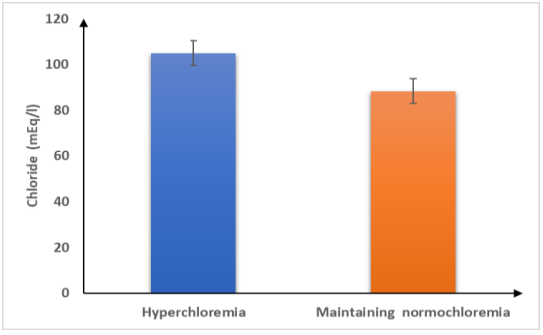

Figure 1. Chloride was significantly higher in hyperchloremia group than maintaining normochloremia group (P-value<0.05).

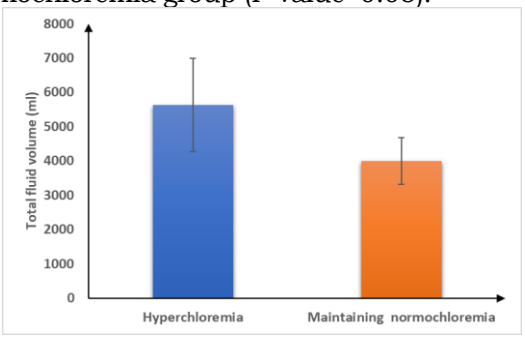

Figure 2. Total fluid volume was significantly higher in hyperchloremia group than maintaining normochloremia group (P-value<0.05).

Table 4. Anion gap and serum cystatin C level of the studied groups.

		HYPERCHLOREMIA (N=30)	MAINTAINING NORMOCHLOREMIA (N=30)	P-VALUE
ANION GAP (MEQ/L)	Mean± SD	30.3±9	48.1±9.09	<0.001*
	Range	15-51	31-65	
SERUM CYSTATIN C LEVEL (MG/L)	Mean± SD	2.5±0.68	0.91±0.22	<0.001*
	Range	1.5-3.5	0.6-1.3	

*:Significant as p-value<0.05.

Anion gap and Serum cystatin C level were significantly lower in hyperchloremia group than maintaining normochloremia group (P-value<0.001), (Table 4; figures 3&4).

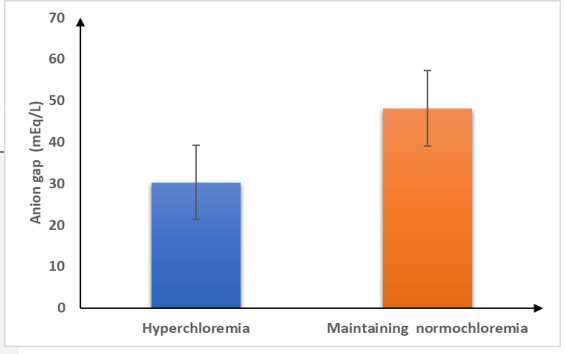

Figure 3. Anion gap was significantly lower in hyperchloremia group than maintaining

normochloremia group (P-value<0.001).

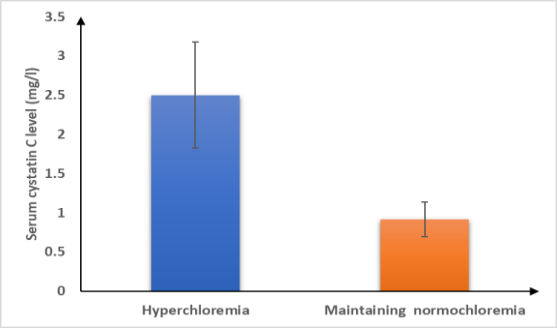

Figure 4. Serum cystatin C level was significantly higher in hyperchloremia group than maintaining normochloremia group (P-value<0.05).

Table 5. Time to initial and final DKA resolution of the studied groups.

		HYPERCHLOREMIA (N=30)	MAINTAINING NORMOCHLOREMIA (N=30)	P-VALUE
TIME TO INITIAL DKA RESOLUTION (HR)	Mean ±SD	21±7.67	10.67±3.63	<0.001*
	Range	8-32	5-16	
TIME TO FINAL DKA RESOLUTION (HR)	Mean ±SD	24.87±8.6	15.6±6.03	<0.001*
	Range	13-39	6-24	

*:Significant as p-value<0.05. DKA:Diabetic ketoacidosis.

Time to initial and: final DKA resolution were significantly delayed in hyperchloremia group than maintaining normochloremia group (P-value<0.001, (Table 5; figures 5&6).

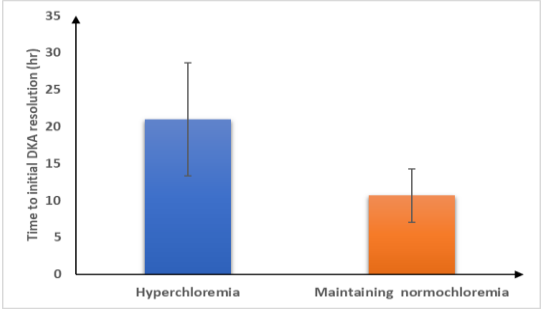

Figure 5. Time to initial was significantly delayed in hyperchloremia group than maintaining normochloremia group (P-value<0.001).

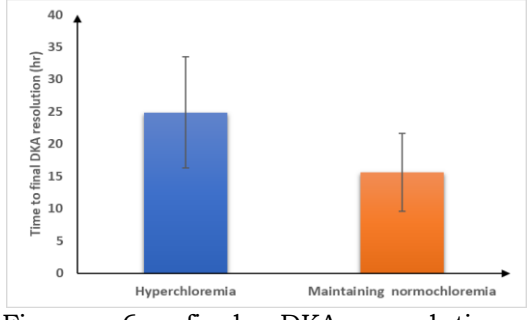

Figure 6. final DKA resolution was significantly delayed in hyperchloremia group than maintaining normochloremia group (P-value<0.001).

Table 6. Admission and in-hospital AKI, intensive care unit admission, and total hospital stay duration for the groups under study.

		HYPERCHLOREMIA (N=30)	MAINTAINING NORMOCHLOREMIA (N=30)	P-VALUE
AKI ON ADMISSION	Yes	27(90%)	25(83.33%)	0.706
	No	3(10%)	5(16.67%)	
IN-HOSPITAL AKI	Yes	9(30%)	2(6.67%)	0.041*
	No	21(70%)	28(93.33%)	
ADMISSION TO ICU	Yes	19(63.33%)	7(23.33%)	0.002*
	No	11(36.67%)	23(76.67%)	
HOSPITAL LENGTH OF STAY (DAY)	Mean±SD	4.03±0.81	3.03±0.72	<0.001*
	Range	3-5	2-4	

*:Significant as p-value<0.05.

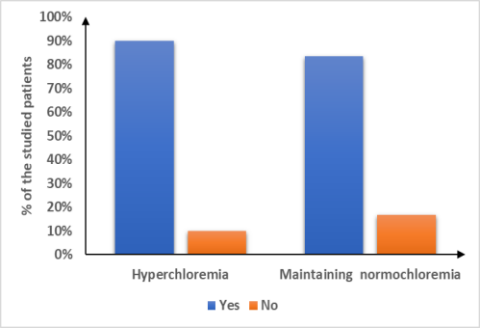

Figure 7. AKI on admission was significantly higher in hyperchloremia group than maintaining normochloremia group (P-value<0.05).

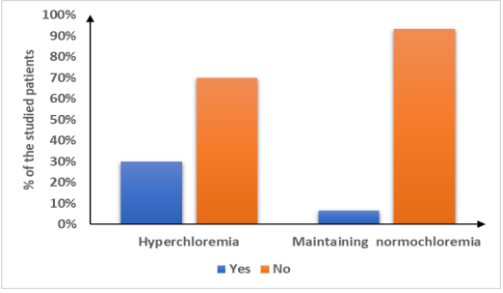

Figure 8. In-hospital AKI was significantly higher in hyperchloremia group than maintaining normochloremia group (P-value<0.05).

4. Discussion

When blood sugar levels are dangerously high, ketosis sets in, and anion gap metabolic acidosis develops, a condition known as diabetic ketoacidosis (DKA). Along with these abnormalities come changes in electrolytes and a decrease in blood volume.[8]

When administering conventional saline for resuscitation to individuals with DKA, electrolyte imbalances, such as hyperchloremia, might be worsened. ICU length of stay (LOS) and insulin infusion duration were both increased due to hyperchloremia-related acidosis. Acquired kidney injury (AKI) can occur in cases of severe hypovolemia. There is some debate on whether hyperchloremia causes AKI.[9]

Evidence linking hyperchloremia to AKI, metabolic acidosis, longer surgery delays, and

worsening morbidity and mortality is mounting among several populations of critically sick patients.[10]

In order to detect AKI at an early stage, a novel biomarker known as serum cystatin C has been utilized in recent years. Belonging to the cystatin superfamily of cysteine protease inhibitors, Cystatin C is a nonglycosylated 13-kDa protein. Every nucleated cell consistently produces it, and after passing freely through the glomerulus and being reabsorbed primarily through megalin-mediated endocytosis in the proximal tubule, it is subsequently catabolized. It is well-known that cystatin C is an excellent indicator of the onset of acute kidney injury in critically sick individuals. Age, gender, race, and muscle mass have less of an impact on serum cystatin C levels than serum creatinine.[11]

In order the purpose of this study was to identify the relationship between hyperchloremia and in-hospital AKI development rates, duration of stay (LOS) in the intensive care unit (ICU), and time to resolution of diabetic ketoacidosis (DKA) (by measuring cystatin-C level).

Neither group significantly differed from the other in terms of nausea, vomiting, diarrhea, and stomach pain.

Similarly, Toledo et al.,[12] enrolled 40 patients (all under the age of 18) with DKA in a cross-sectional and observational study. There were 22 people in the group with metabolic acidosis (MA) and a hyperchloremic component, and 18 people in the group with MA and a high anion gap alone. According to their findings, there was no discernible difference between the two groups in terms of fever, vomiting, and diarrhea.

The present research found that the hyperchloremia group had substantially greater chloride and total fluid volume than the normochloremia group that maintained the same level of chloride. The hyperchloremia group had noticeably lower bicarbonate and pH levels compared to the normochloremia group that maintained normal chloride levels. Sodium, potassium, and serum creatinine levels were not substantially different between the two groups.

A drop in bicarbonate concentrations results from hyperchloremia, which is characterized by a rise in Cl-concentrations that reduces the strong ion difference (SID).[13]

Supporting the findings of the current study, Goad et al.,[5] performed a cohort analysis looking back at 102 patients admitted with DKA who were 18 years old or older. Among the patients treated with DKA, 52 experienced hyperchloremia and 50 remained within the normal range. Results showed that compared to the normochloremia-maintaining group, the hyperchloremia group had much greater chloride and total fluid volume. The hyperchloremia group had noticeably lower bicarbonate and pH levels compared to the normochloremia group, which maintained normal chloride levels. The creatinine levels of the two groups were not significantly different.

Consistent with what was found in this study, Li et al.,[14] analyzed data from 5616 individuals to determine the effect of serum chloride on the risk of death from any cause in intensive care unit patients with serious illnesses. The hyperchloremia group had substantially greater chloride levels than the normochloremia group that was able to maintain normal levels, according to their findings.

The present investigation found that the anion gap was much reduced in the hyperchloremia group compared to the normochloremia group that maintained normal chloride levels.

This study's results are backed by Yıldırımçakar et al.,[15] investigated the risk of hyperchloremia and persistent acidosis in 153 patients treated for diabetic ketoacidosis in children. They discovered that 69.3% of the subjects had hyperchloremia and showed a small anion gap.

The risk of acute kidney injury (AKI), morbidity, and death has been linked to hyperchloremia in certain diseases and conditions in recent years. One such condition is diabetic ketoacidosis. Another possible explanation for the association between hyperchloremia and elevated serum cystatin C levels is the fact that these conditions are more common in intensive care unit (ICU) patients.[16,17]

In a consistent study, Hlemy et al.,[18] thirty critically sick patients with AKI and thirty control persons of the same age were included in a prospective cohort study. Specifically, they proved that cystatin C level is an excellent biomarker for AKI prediction and early identification. In addition, cystatin C level can be utilized to predict the outcome of AKI cases.

No statistically significant difference in AKI admission occurred between the two groups in our study. The hyperchloremia group had a far higher rate of in-hospital AKI, ICU admissions, and length of stay compared to the normochloremia group that maintained normal chloride levels. There were no changes in the prevalence of AKI at admission across the groups, even though more patients with hyperchloremia had AKI while hospitalized.

Multiple patient populations have shown that hyperchloremia is related to worse renal outcomes in observational studies.[19]

Supporting the findings of the present study, Patil and Vishwanath,[20] evaluated the prevalence of hyperchloremic metabolic acidosis in 32 patients diagnosed with diabetic ketoacidosis (DKA). Patients diagnosed with normochloremic metabolic acidosis were less likely to experience

AKI than those with hyperchloremic metabolic acidosis, according to their findings.

With the results of the present investigation in mind, Basnet et al.,[21] evaluated the impact of normal saline and half-normal saline on blood electrolytes during the recovery phase of DKA by conducting a retrospective chart review of all children admitted to the pediatric intensive care unit (PICU) with DKA, aged 1–18 years. In patients who were given normal saline as a post-bolus rehydration fluid, they discovered that hyperchloremia extended the duration of their PICU hospitalization.

The present study found that compared to the normochloremia-maintaining group, the hyperchloremia group required much more time to reach both the initial and final resolutions of DKA.

Consistent with what this study found, Desai et al.,[22] 140 patients, all 18 years or older, admitted with a diagnosis of DKA and given 2 liters or more of resuscitation fluid, were included in the retrospective chart analysis that was carried out at a single center. They were split into two groups: one with normochromic parameters (n=60) and another with hyperchloremic parameters (n=80). The researchers discovered that the hyperchloremic group required much more time than the normochromic group to reach the first DKA resolution.

4. Conclusion

Hyperchloremic metabolic acidosis, which frequently results from giving patients with DKA a 0.9% sodium chloride solution, is strongly linked to the development of in-hospital AKI. It has been suggested that this acid-base disruption adds time to the duration of hospital stays and the time it takes for DKA to resolve.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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