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Original Research Article

STUDY OF ACUTE KIDNEY INJURY IN CHILDREN WITH DIABETIC KETOACIDOSIS

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ABSTRACT

Background: Diabetic ketoacidosis is a relatively common acute complication of Type 1 diabetes mellitus. Children hospitalized with DKA have an increased risk for the development of acute kidney injury secondary to severe water and electrolytes loss from intracellular and extracellular fluid compartment. AKI in children with DKA is an independent risk factor associated with prolonged hospital stay and higher mortality rate in children. The objective is to evaluate the association between acute kidney injury in children with type one diabetes with diabetic ketoacidosis.

Materials and Methods: This is a prospective observational study conducted over a period of one and a half year in children with confirmed type one diabetes with diabetic ketoacidosis according to International Society of Paediatric and Adolescent Diabetic clinical guidelines admitted in paediatric intensive care of Indira Gandhi Institute of Child Health, Bengaluru.

Results: The study included 50 children with T1DM with DKA. AKI occurred in 20(40%) children with T1DM with DKA. Mild AKI was noted in 11(22%) children and moderate to severe AKI occurred in 9(18%) children with DKA. Serum chloride level at 12 hours were the most significant factor associated with moderate to severe AKI development. Children with AKI had prolonged acidosis, longer PICU stay, and higher mortality.

Conclusion: AKI frequently occurred in T1DM children hospitalized with DKA. Hyperchloremia at 12 hours after hospital admission was associated with increased risk for development moderate to severe AKI.

Keywords: Acute Kidney Injury, Children, Diabetic Ketoacidosis.

INTRODUCTION

Diabetes mellitus (DM) is a multifactorial metabolic disease characterized by hyperglycaemia that stems from deficiencies in either insulin production, insulin function, or both. Type 1 diabetes mellitus (T1DM) is due to immune depletion of β cells. Diabetic ketoacidosis (DKA) results from absolute or relative deficiency of circulating insulin and the combined effects of increased levels of the counter regulatory hormones: glucagon, catecholamines, growth hormone and cortisol.[1,2]

Acute kidney injury (AKI) is characterized by rapid decline in Glomerular Filtration Rate (GFR) in hours to days because of retention of nitrogenous waste products and perturbation of extracellular volume and fluid, alteration in electrolyte & acid base homeostasis. A patient may have AKI without injury to renal parenchyma.[3]

The clinical manifestations of paediatric AKI range from a mild increase in serum creatinine to anuric renal failure requiring dialysis. The most common type of paediatric AKI is prerenal or volume-responsive AKI, which is typically caused by hypovolemia and reduced renal perfusion. If a prerenal insult is severe or prolonged, it can lead to structural damage to the renal parenchyma, known as acute tubular necrosis (ATN).

In cases of DKA, presenting with severe AKI there will be significant intravascular volume depletion leading to intrinsic tubular injury rather than a milder prerenal or volume-responsive injury.

Our study aims to assess the incidence of AKI in children with DKA and examine the association between the severity of acidosis and volume depletion and the risk of developing AKI. Despite the importance of fluid-rehydration strategies in managing DKA, AKI in these patients has not been systematically studied.[4]

MATERIALS AND METHODS

This prospective observational study was conducted among children with type one diabetes with DKA diagnosed using ISPAD guidelines admitted at T1DM with DKA at Indira Gandhi Institute of Child Health, Bangalore. Duration of study was one and half year.

Inclusion Criteria
Children aged < 18 years, diagnosed with type I diabetes with DKA as per International Society for Paediatric and Adolescent Diabetes (ISPAD) criteria in PICU, and are willing to give consent.

Exclusion Criteria
Children diagnosed with congenital or acquired kidney diseases or chronic kidney disease, including patients with diabetic nephropathy and those with persistent microalbuminuria.

Method of Data Collection
Children diagnosed with T1DM aged between 1month to 18years admitted in paediatric intensive unit of Indira Gandhi Institute of Child Health Bangalore were enrolled after obtaining informed consent.
Detailed history and clinical examination was done. DKA was defined and the severity classified as per ISPAD guidelines. Acute kidney injury was defined using KIDIGO criteria.
Demographic data, onset, duration of DKA and presence any associated complications were collected. Vital signs and Glasgow Coma Scale were assessed at admission. Laboratory parameters including blood glucose, blood gas analysis, serum electrolytes, urine ketone bodies, glycated hemoglobin (HbA1c) and renal function test were measured on admission.

Patients underwent hourly monitoring of vital signs and capillary blood glucose, with blood gas analysis and serum electrolyte estimation performed every 12 hours until resolution of metabolic acidosis was done. Serum osmolality and corrected serum sodium was calculated accordingly and serum creatinine measured at admission, 24 hours and 48hours after admission.

Statistical Analysis
SPSS (Statistical Package for Social Sciences) version 20 [IBM SPASS statistics (IBM corp. Armonk, NY, USA released 2011)] was used to perform the statistical analysis. Data was entered in the excel spread sheet. Descriptive statistics of the explanatory and outcome variables was calculated by mean, standard deviation for quantitative variables. Inferential statistics like o Chi-square test was applied for categorical variables. The level of significance is set at 5.

RESULTS

In the present study among the 50 children with T1DM presenting with DKA, 22 (44%) were aged 11–15 years, 13 (26%) were 6–10 years, 10 (20%) were <5 years, and 5 (10%) were >15 years. The mean age was 9.3 years (range, 0.2–17 years) with male to female ratio of 0.78:1.

Thirty one (62%) children had established T1DM and 19 (38%) children were newly diagnosed with T1DM.Mild, moderate, severe DKA was noted in 1(3.2%), 5(16.1%), 25(80.6%) respectively in children with established T1DM. Among the newly diagnosed cohort,15(78.9%) children had severe DKA. Mean duration of symptoms of DKA for new onset and established T1DM was 2.26 days and 1.84 days respectively.
The mean HbA1c in 31(62%) children with established T1DM in our study was 13.25% and in 19(38%) children with newly diagnosed T1DM, the mean HbA1c was 13.04%.

Among 31(62%) children with established T1DM, 30(96.8%) children had poor control with HbA1c more than 9 and 1(3.2%) child had fair control with HbA1c between 7.5-9.
The mean BMI of 31(62%) children with established diabetes was 15.64 kg/m2 and in 19(38%) children with new onset diabetes was 12.75 kg/m2. Among 50(100%) children with T1DM with DKA, AKI was noted in 20(40%) children with DKA and no AKI was noted in 30(60%) children with T1DM with DKA.

Of the 50 children with DKA, 30 (60%) had no AKI, while 20 (40%) developed AKI, including 11 (22%) with mild and 9 (18%) with moderate-to-severe AKI. The mean age was 10.1 years in children without AKI and 8.1 years in those with AKI.

Among the 20(40%) children who had DKA with AKI, male: female ratio was 0.66:1 while the ratio of children with DKA without AKI was 0.87:1. Severe DKA was observed in 80% of children with AKI (16/20) and 80% without AKI (24/30).

Table 1: Clinical variables of children with DKA according to maximum stage of AKI during the hospital stay

Variables	Stage 0 No AKI (n=30)	Stage I Mild AKI (n=11)	Stage II &III Mod-Severe AKI (n=9)	P value
Age (years) Mean (SD)	10.12(4.12)	8.41(4.93)	7.66(4.92)	0.29
Gender	14 (46.6%)	5(45.5%)	3(33.3 %)	1.00
Male n (%)				
Female n (%)	16 (53.3%)	6(54.5%)	6(66.6%)	
BMI(Kg/m2) Mean (SD)	14.45 (3.27)	16.41(3.03)	12.31(3.04)	0.026
Pulse rate (beats /min) Median (IQR)	140(122.5-146.5)	134(121.5-143)	150(137.25-159.5)	0.21
Respiratory rate (cycle/min) Median (IQR)	40(36-42)	38(30.5-57.6)	45(32.5-64)	0.697
Systolic blood pressure (mm Hg) Median (IQR)	110(102.5-119)	108(105-122)	94(79.75-115.5)	0.2
Diastolic blood pressure (mmHg) Median (IQR)	72(55-86.5)	79(65-83.5)	64(50.2-70.5)	0.192
GCS Median (IQR)	13(12-15)	15(12-15)	12(11-14)	0.65

Children without AKI (n=30) had a mean BMI of 14.2 kg/m², mean pulse rate of 140 beats/min, respiratory rate of 40 breaths/min, blood pressure of 110/72 mmHg, and a median GCS score of 13. In contrast, children with moderate-to-severe AKI (n=9; 33.3% male) had a lower mean age (7.7 years) and BMI (12.3 kg/m²), higher median pulse rate (150 beats/min) and respiratory rate (45 breaths/min), lower median blood pressure (98.5/64 mmHg), and a lower median GCS score (12)

Table 2: Clinical variables of children with DKA according to maximum stage of AKI during the hospital stay

DKA		Stage 0 No AKI (n=30)	Stage I Mild AKI (n=11)	Stage II &III Mod-Severe AKI (n=9)	P value
State of diabetes	New onset 19 (38%)	9(30%)	4(36.4%)	6(66.6%)	0.84
	Established 31(62%)	21(70%)	7(63.6%)	3(33.4%)	
DKA severity at admission	Mild DKA n 3(6%)	1(3.3%)	2(18.2%)	0(-)	0.396
	Moderate DKA 7 (14%)	5(16.6%)	2(18.2%)	0(-)	
	Severe DKA 40(80%)	24(80%)	7(63.6%)	9(100%)	

Stage 3 AKI occurred in 9 (18%), including 6 (66.7%) with new-onset T1DM and 3 (33.3%) with established T1DM. Mild AKI was observed in 11 (22%) children, of whom 7 (63.6%) had severe DKA, 2 (18.2%) had moderate DKA, and 2 (18.2%) had mild DKA. All children with moderate-to-severe AKI (9/50, 18%) had severe DKA.

Table 3: Biochemical variables in diabetic ketoacidosis episodes at hospital admission in children with AKI and without AKI

Variables at admission Mean (SD)	No AKI Stage=0 (n=30)	Mild AKI Stage I (n=11)	Mod-Severe AKI Stage II & III (n=9)	P value
pH	7.08 ± 0.13	7.12 ± 0.17	7.01 ± 0.12	0.195
S. Bicarbonate (mEq/L)	4.87 ± 2.87	6.49 ± 3.78	3.78 ± 1.20	0.421
Anion Gap (mEq/L)	28.78 ± 6.37	29.22 ± 5.92	26.80 ± 6.93	0.893
corrected. Serum Sodium (mEq/L)	137.50 ± 5.39	140.31 ± 5.42	142.82 ± 11.47	0.133
S. Potassium (mEq/L)	4.89 ± 0.92	5.46 ± 0.90	4.81 ± 1.02	0.258
S. Chloride (mEq/L)	96.62 ± 5.63	100.15 ± 5.67	106.03 ± 12.98	0.029
RBS (mg/dL)	507.15 ±149.16	550.64 ±156.32	537.25 ± 159.69	0.748
S. Osmolality (mOsm/L)	290.61 ±12.71	300.95 ±11.42	302.00 ± 24.05	0.051
Blood Urea (mg/dL)	29.37 ± 13.77	41.90 ± 10.00	63.55 ± 32.92	<0.001
S. Creatinine (mg/dL)	0.45 ± 0.13	0.63 ± 0.17	1.11 ± 1.07	0.002
eGFR (ml/L)	120.83 ±27.75	78.75 ± 12.10	59.75 ± 37.14	<0.001

The table summarizes admission biochemical parameters across three groups: no AKI, mild AKI, and moderate-to-severe AKI. Children with moderate-to-severe AKI had a mean (SD) pH of 7.01 (0.12), serum bicarbonate of 3.78 (1.20) mEq/L, corrected sodium of 142.82 (11.47) mEq/L, and serum chloride of 106.03 (12.98) mEq/L. Among the eight children with available data, the mean blood urea was 63.55 (32.92) mg/dL and the mean eGFR was 59.75 (37.14) mL/min.

Table 4: Biochemical variables in diabetic ketoacidosis episodes at 12 hours after hospital admission in children with AKI and without AKI

Variables at 12 hours of admission mean (SD)	No AKI stage=0 (n=30)	Mild AKI Stage I (n=11)	Mod-Severe AKI Stage II & III (n=9)	P value
pH	7.21 ± 0.13	7.20 ± 0.18	7.13 ± 0.12	0.147
S. Bicarbonate (mEq/L)	9.29 ± 4.40	8.96 ± 4.98	6.62 ± 2.69	0.335
Anion Gap (mEq/L)	18.97 ± 4.85	22.19 ± 5.76	18.69 ± 6.79	0.280
corrected. Sodium (mEq/L)	137.08 ± 6.65	141.64 ± 9.16	147.16 ± 11.73	0.065

S. Potassium (mEq/L)	4.24 ± 1.17	4.58 ± 0.68	4.16 ± 1.29	0.422
S. Chloride (mEq/L)	103.08 ± 19.40	106.96 ± 9.03	118.08 ± 10.29	0.016
RBS (mg/dL)	307.68 ±117.09	348.27 ±115.16	339.25 ± 144.14	0.726
S. Osmolality (mOsm/kg)	285.23 ± 14.71	294.59 ± 20.14	306.32 ± 25.88	0.080

The above table summarizes biochemical variables in children with DKA after 12 hours of hospital stay into three groups, no AKI, mild AKI & moderate to severe AKI. The pH in moderate to severe AKI had mean of 7.13 with SD of 0.12, serum bicarbonate in moderate to severe AKI had mean of 6.62 mEq/L with SD 2.69 mEq/L, corrected sodium in moderate to severe had mean of 147.16 mEq/L with SD of 11.73 mEq/L, chloride levels in moderate to severe AKI had mean of 118.08 mEq/L with SD of 10.29 mEq/L.

Table 5: Biochemical variables in diabetic ketoacidosis episodes at first 24h after hospital admission in children with AKI and without AKI

Variables at 24 hours of admission mean (SD)	No AKI stage=0 n=30	Mild AKI Stage I n=11	Mod-severe AKI Stage II & III n=9	P value
pH	7.29 ± 0.08	7.26 ± 0.14	7.22 ± 0.10	0.265
S. Bicarbonate (mEq/L)	13.32 ± 3.34	12.15 ± 4.74	7.76 ± 3.45	0.003
Anion Gap (mEq/L)	14.50 ± 2.61	15.93 ± 3.24	17.65 ± 5.37	0.152
S. Sodium (mEq/L)	136.87 ± 5.05	138.48 ±10.87	144.51 ±11.29	0.265
S. Potassium (mEq/L)	3.78 ± 0.82	4.01 ± 0.59	3.92 ± 1.12	0.410
S. Chloride (mEq/L)	106.73 ± 5.62	108.47 ±12.28	117.09 ± 8.04	0.020
RBS (mg/dL)	250.89 ±109.21	283.34 ±95.76	241.88 ±68.38	0.659
S. Osmolality (mOsm/kg)	283.04 ±12.35	286.73 ±22.78	298.02 ±23.14	0.250
Blood Urea (mg/dL)	23.23 ± 14.35	46.50 ± 19.35	73.29 ± 39.79	<0.001
S. Creatinine (mg/dL)	0.42 ± 0.21	0.60 ± 0.20	1.35 ± 0.98	<0.001
eGFR (ml/L)	140.12 ±37.93	84.88 ± 14.31	51.67 ± 40.60	<0.001

The table summarizes biochemical parameters 24 hours after admission among children with DKA stratified by AKI severity. Children with moderate-to-severe AKI had a mean (SD) pH of 7.22 (0.10), serum bicarbonate of 7.76 (3.45) mEq/L, corrected sodium of 144.51 (11.29) mEq/L, and serum chloride of 117.09 (8.04) mEq/L. Among the six children with available data, the mean blood urea was 73.29 (39.79) mg/dL and mean eGFR was 51.67(40.60)mL/min.

Table 6: Biochemical variables in diabetic ketoacidosis episode at first 48h after hospital admission in children with AKI and without AKI

Variables at 48 hours of admission mean (SD)	No AKI stage=0 n=30	Mild AKI Stage I n=11	Mod-severe AKI Stage II & III n=9	P value
pH	7.35 ± 0.06	7.32 ± 0.06	7.24 ± 0.11	0.020
S. Bicarbonate (mEq/L)	17.72 ± 4.28	15.67 ± 4.19	10.10 ± 3.97	0.002
Anion Gap (mEq/L)	13.40 ± 3.81	13.20 ± 2.75	13.64 ± 2.50	0.915
corrected. Sodium mEq/L)	138.45 ± 4.54	138.91 ± 3.73	145.56 ± 7.25	0.018
S. Potassium (mEq/L)	12.46 ± 48.17	30.47 ± 88.23	3.39 ± 0.62	0.140
S. Chloride (mEq/L)	102.25 ±17.59	108.85 ± 5.99	120.10 ± 7.60	<0.001
RBS (mg/dL)	200.41 ±70.96	190.32 ±64.70	218.21 ±96.68	0.882
S. Osmolality (mOsm/kg)	282.67 ±11.01	283.18 ±14.81	299.29 ±15.91	0.008
Blood Urea (mg/dL)	18.54 ± 9.72	27.85 ± 15.66	64.93 ± 22.37	<0.001
S. Creatinine (mg/dL)	0.36 ± 0.13	0.47 ± 0.18	1.57 ± 0.98	<0.001
eGFR (ml/min)	158.56 ±42.19	112.50 ±28.31	43.75 ± 28.97	<0.001

In current study children with moderate to severe AKI (stage II and III), the mean length of PICU stay was 5.12 days and the mean length of hospital stay was 8.25.

DISCUSSION

A total of 50 children in the age group of 1month to 18 years with T1DM with DKA admitted to the PICU were enrolled in our study. Similar age group was noted in studies by Huang et al,[6] Yang et al,[7] whereas, Hegab et al,[5] conducted a study in children aged between 6 months to 12 Years. The most common age group in our study was 11 to 15 years, the median age noted in our study was 10 years which was similar to studies conducted by Hegab et al,[5] Huang et al,[6] and Yang et al,[7] who noted median age of 11.8 years and 12 years respectively which was higher compared to our study. Female predominance was noted in 28(56%) children in our study with male to female ratio of 0.8:1 and was comparable to Hegab et al,[5] Huang et al,[6] Yang et al,[7] The diagnosis of DKA was based on ISPAD guidelines in the present study and in Hegab et al,[5] Yang et al,[7] study. In our study new onset T1DM was noted in 19(38%) children and established T1DM in 31(62%) children comparable to Hegab et al,[5] study who noted new onset T1DM in 67(43%) and established T1DM in 88(56.8%) children. Among 50(100%) children with T1DM with DKA mild, moderate and severe DKA noted in 3(6%), 7(14%) and 40(80%) children

respectively. Severe DKA was noted in 40(80%) children with T1DM which was comparatively higher to Hegab et al,[5] Yang et al,[7] who noted severe DKA in 130(49%), 37(41%) respectively.

The current study prospectively assessed AKI among T1DM children hospitalized with DKA using the KDIGO criteria, similar guidelines were used in Hegab et al,[5] Huang et al,[6] Yang et al,[7] whereas Baalaaji et al used pRIFLE criteria for diagnosis of AKI in DKA children. Our study found 20(40%) of DKA episodes associated with AKI and no AKI was noted in 30(60%) children which was identical to Hegab et al,[5] who observed AKI in 110(41.5%) children with DKA whereas, Huang et al,[6] and Yang et al,[7] noted higher frequency of AKI in 170(56.5%) and 75(71.4%) children with DKA respectively. In our study frequency of AKI occurred in established T1DM and new onset T1DM was same whereas Haung et al,[6] noted higher proportion of AKI in established T1DM with DKA that could be because of poor compliance, treatment failure, variable dehydration status and biochemical parameters contributed for AKI.

In our study the median age noted in children with DKA with no AKI and moderate to severe AKI was 11 years and 8 years respectively with p value of 0.29 which was statistically not significant whereas Hegab et al,[5] noted median age of 10 years and 7 years in no AKI and moderate to severe AKI children respectively, that is younger age was associated with development of moderate to severe AKI with DKA. In the current study GCS at admission in children with no AKI, mild AKI and moderate to severe AKI was 13,15 and 12.5 respectively, whereas Hegab et al,[5] noted GCS <14 (p value of 0.004) in children with moderate to severe AKI that could be because of more proportion of children with moderate severe AKI had severe DKA at admission compared to no AKI group, however in our study low GCS was not statistically significant with P value of 0.6. In our study the median heart rate at admission was noted to be 140,134 ,150 beats per minute in children with no AKI, mild AKI and moderate to severe AKI. Heart rate was higher in children with moderate to severe AKI when compared to no AKI and mild AKI but was not statistically significant with p value of 0.21, similarly Hegab et al,[5] also noted higher heart rate in children with moderate to severe AKI compared to no AKI and mild AKI, which was not statistically significant. In the present children with moderate to severe AKI had increased serum chloride levels of 121 mEq/L at 12 hours after admission with p value of 0.016 which was higher compared to no AKI and mild AKI with children with DKA who had serum chloride of 102 mEq/L and 104 mEq/L at 12 hours after admission respectively. Our study was correlating with Hegab et al,[7] who noted serum chloride levels at 12 hours after admission was 112(106-116mEq/L) which was statically significant (p value 0.01) and baalaaji et al,[9] reported that serum chloride more than 112 mmol/L at 24 hours after hospital admission was significantly associated with AKI progression. According to baalaaji et al,[8] The cause for hyperchloremia at 12 hours after admission was attributed to iatrogenic element caused by the type of intravenous fluids received. However, study conducted by Bansal et al concluded that rather than type of fluid or volume, the cause of hyperchloremia appears to be a physiological response to bicarbonate loss over chloride with increased renal perfusion. In the present study corrected sodium at admission in children with DKA with moderate to severe AKI was

142.8 mEq/L with p value of 0.065 which correlates with Hursh et al,[9] study who noted corrected sodium of 143mEq/L in severe AKI, however Hegab et al,[5] noted sodium of 139 mEq/L in moderate to severe AKI which is lower compared to our study, the difference in serum sodium across different study was attributed to variability in severity of dehydration at admission.

The current study found that the length of PICU stay in children with moderate to severe AKI was 5 days compared to 3 days in no AKI children with p value of 0.09. The duration of hospital stay in children with DKA with moderate to severe AKI was 9 days compared to children with no AKI children had 5 days with p value of 0.36. The length of PICU stay and hospital stay was prolonged in children with moderate to severe AKI which was not statistically significant similar to study conducted by Hegab et al,[5] and Baalaaji et al,[8] who also noted longer duration of PICU and hospital stay in children with moderate to severe AKI when compared to no AKI children. The prolonged PICU stay in children with DKA with AKI secondary to the persistent metabolic acidosis. The strength of study is that it is a prospective study to find out the association between AKI and DKA in children and its risk factors. The study didn't asses the long-term effects of AKI on children T1DM. more studies are required to assess the long-term effects of AKI on development related renal complication.

CONCLUSION

AKI occurs frequently in T1DM children hospitalized with DKA. We found AKI in 40% children with T1DM with DKA. Increased serum chloride level at 12 hours during DKA management was associated with increased risk of development of moderate to severe AKI. Children with AKI had prolonged recovery time from metabolic acidosis and had longer duration of PICU stay. Careful monitoring and prompt management of children admitted with DKA who have elevated serum chloride levels during treatment may help prevent the progression of moderate to severe AKI.

REFERENCES

1. David R Weber, Nicholus Jospe. Diabetes mellitus: Classification of Diabetes, Neslson Textbook of Pediatrics, 21st edition vol 2, Philadelphia: Elsevier; 2020: p11814-11823.
2. Glaser N, Fritsch M, Priyambada L, Rewers A, Cherubini V, Estrada S, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. Pediatric Diabetes. 2022 Oct 17;23(7):835–56.
3. Khwaja A. KDIGO Clinical Practice Guidelines for Acute Kidney Injury. Nephron. 2012 Aug 7;120(4):c179–84
4. Kassebaum N, Kyu HH, Zoeckler L, Olsen HE, Thomas K, Pinho C, et al. Child and Adolescent Health From 1990 to 2015. JAMA Pediatrics [Internet]. 2017 Jun 1;171(6):573. Available from: https://jamanetwork.com/journals/jamapediatrics/fullarticle/2613463
5. Hegab AM, Khalil FF, Abosedera MM. Incidence and factors associated with acute kidney injury among children with type 1 diabetes hospitalized with diabetic ketoacidosis: A prospective study. Pediatric Diabetes. 2022 Jun 10;.
6. Huang SK, Huang CY, Lin CH, Cheng BW, Chiang YT, Lee YC, et al. Acute kidney injury is a common complication in children and adolescents hospitalized for diabetic ketoacidosis. Shimosawa T, editor. PLOS ONE. 2020 Oct 7;15(10):e0239160.
7. Yang EM, Lee HG, Oh KY, Kim CJ. Acute Kidney Injury in Pediatric Diabetic Ketoacidosis.Indian J Pediatr. 2021 Jun;88(6):568–73.
8. Baalaaji M, Jayashree M, Nallasamy K, Singhi S, Bansal A. Predictors and Outcome of Acute Kidney Injury in Children with Diabetic Ketoacidosis. Indian Pediatr. 2018 Apr 15;55(4):311–4.
9. Marzuillo P, Iafusco D, Zanfardino A, Guarino S, Piscopo A, Casaburo F, et al. Acute Kidney Injury and Renal Tubular Damage in Children With Type 1 Diabetes Mellitus Onset. J Clin Endocrinol Metab. 2021 Jun 16;106(7):e2720–37.