The Egyptian Journal of Hospital Medicine (April 2025) Vol. 99, Page 1638-1643

Acute Kidney Injury in Children with Type 1 Diabetes Hospitalized for Diabetic Ketoacidosis: Incidence and Risk Factors

Mai Mohammed Mostafa*[1], Azza Omran[2]
Departments of [1]Pediatrics and [2]Clinical Pathology, Mataria Teaching Hospital, Cairo, Egypt
***Corresponding author:** Mai Mohammed Mostafa, **Mobile:** (+20) 01222295284, E-mail: maiyoi_dec2007@yahoo.com

ABSTRACT

Background: Diabetic ketoacidosis (DKA) is the most critical medical condition of type 1 diabetes mellitus (TIDM). Hospitalized children with DKA are more likely to develop acute kidney injury (AKI).

Objective: This study aimed to evaluate AKI risk factors and incidence in TIDM children hospitalized with DKA.

Methods: This one-year prospective observational study that was carried out at El-Mataria Teaching Hospital on 22 children ≤ 18 years old with T1DM hospitalized with DKA.

Results: Out of 22 T1DM children with DKA, 17 (77.27%) presented with AKI. 8 (47.06%) had mild, 7 (41.18%) had moderate, and 2 (11.76%) had severe AKI. Serum creatinine on admission was significantly increased in AKI contrasted to non-AKI patients (P value =0.019). Body mass index (BMI) was a significant risk factor for AKI (OR 0.653, 95% CI 0.439–0.971, P value =0.035). Platelets were a significantly greater in AKI cases (P value =0.027).

Conclusions: This study found AKI high incidence, with 77.27% of children developing AKI during their hospitalization. Most AKI cases were mild to moderate in severity. The eGFR was significantly less in AKI children, while platelets were a significantly greater in AKI children. The duration of diabetes (newly diagnosed vs. established) was not a significant risk factor for AKI. However, in the univariate regression analysis, BMI emerged as a significant risk factor for AKI, with a higher BMI associated with a lower AKI development risk.

Keywords: Acute Kidney Injury, Diabetic Ketoacidosis, Type 1 Diabetes Mellitus.

INTRODUCTION

Children who are hospitalized with Diabetic ketoacidosis (DKA) have an elevated progressing acute kidney injury (AKI) risk [1]. AKI assess during DKA is tricky, there is not yet an easily accessible test to assess AKI in real-time [2].

The diagnosis of AKI is predicated on elevated serum creatinine levels and decreased urine output after hospitalization. But hyperglycemia causes osmotic diuresis, which hides the decrease in urine until significant volume depletion develops [3]. This work aimed to evaluate the AKI incidence and risk factors in type 1 diabetes mellitus (T1DM) children hospitalized with DKA.

SUBJECTS AND METHODS

This prospective research was carried out between December 2022 and December 2023 in EL-Mataria Teaching Hospital, Pediatric Intensive care Unit (PICU).

Twenty-two T1DM patients hospitalized with DKA, identified according to the International Society for Pediatric and Adolescent Diabetes (ISPAD) clinical practice consensus guidelines 2018 [1]. Exclusion criteria were children with congenital, acquired kidney disorders and diabetic nephropathy.

Patients were undergone detailed history taking involving, sex, age, diabetes family history and disease duration before admission was recorded. Full clinical evaluation involving vital signs (heart rate (HR) and blood pressure), anthropometric measurements (Weight, BMI & height), Glasgow coma scale (GCS), dehydration degree, and urine output were assessed at admission.

Also, the degree of acidosis was used to classify the severity of DKA. 5 ml of blood were collected and centrifuged where serum sodium and potassium were estimated by electrochemical and optical techniques (GEM Premier 3000-Bioteck). Serum creatinine (Its level was assessed at admission, 24 and 48 hours after hospitalization), and random blood glucose were assessed by a chemistry autoanalyzer (VITROS-350). 2 ml was collected on EDTA for a complete blood count (CBC) using Sysmex XS-500I. 1 ml of blood was collected on a heparinized syringe for blood gas detection (Ph, PCO_2, and Hco_3^-) using Roche COBAS B221. Metabolic acidosis recovery time was recorded (The duration from the therapy start until blood gas HCO_3^- level > 15 mEq/L and Ph > 7.3). The acidosis degree was used to classify the severity of the DKA episode at hospitalization following ISPAD guidelines [1]. Metabolic acidosis recovery time (The duration from the start of therapy until blood gas test showing HCO_3^- level >15 mEq/L and Ph >7.3) was collected.

The expected baseline creatinine (EBC) and the indicated eGFR were measured utilizing Schwartz formula [4]. We detected stage 3 AKI (Severe AKI) as creatinine level greater than 3 times the EBC, stage 2 AKI (Moderate AKI) as creatinine level between 2 and 3 times the EBC, stage 1 AKI (Mild AKI) as creatinine level 1.5 to 2 times the EBC, and stage 0 AKI (no AKI) as a creatinine level < 1.5 times the EBC.

The primary outcome was to detect the incidence and stage of AKI between T1DM children who were admitted to the hospital with DKA in accordance with kidney disease/Improving Global Outcomes criteria [5].
The secondary outcome was to find the AKI risk factors for example, age, severity of acidosis, GCS

Received: 10/11/2024
Accepted: 09/01/2025

score, degree of dehydration, recovery time from metabolic acidosis, serum sodium, corrected sodium, and potassium levels on admission.

Ethical approval: Institutional Review Board of General Organization of Teaching Hospitals and Institutes approved the study. Informed consents were signed by all participants guardian as all of them were under 18 years. The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis

SPSS version 26.0 was done to perform the statistical analysis. The data distribution normality was assessed utilizing the histograms and Shapiro-Wilks test. The unpaired student t-test was done to analyse the quantitative parametric data, which were presented as Mean ± SD. Qualitative data were analysed utilizing the Fisher's exact or X^2-test when appropriate and were presented as frequency and percentage (%). Statistical significance was definite as a two-tailed with P value ≤ 0.05.

RESULTS

A total of 22 children were hospitalized for DKA, from December 2022 to December 2023. 10 (45.45%) were females, and 12 (54.55%) were males. Their mean age was 8.57 ± 3.28 years and ranged from 1.3 to 12 years. Of all 22 studied children, 12 (54.55%) were newly diagnosed with T1DM, while the other 10 (45.45%) had pre-existing T1DM. Out of 22 T1DM children with DKA, 17 (77.27%) presented with AKI as illustrated in figure (1).

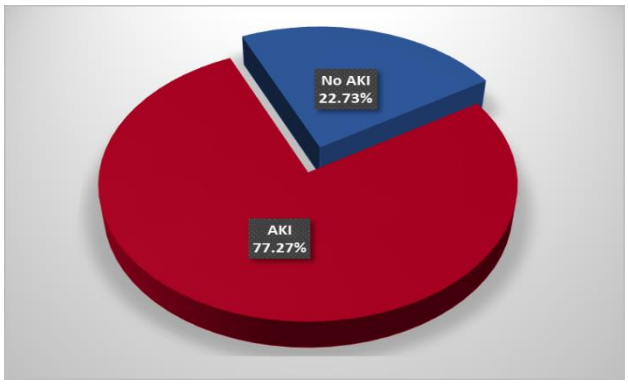

Figure (1): The incidence of AKI among T1DM children with DKA.

Regarding AKI patients, 8 (47.06%) had mild AKI, 7 (41.18%) had moderate AKI, and 2 (11.76%) children had severe AKI (Table 1).

Table (1): AKI stage

	n=17
Mild	8 (47.06%)
Moderate	7 (41.18%)
Severe	2 (11.76%)

Data are presented as frequency (%). AKI: acute kidney injury.

Table (2) highlighted the entire study population clinical characteristics. Overall, age, and BMI were significantly lower in AKI than in non-AKI children (P value <0.05). Gender, family history of DM, newly diagnosed diabetic patients, tachycardia, HTN, DKA stage, GCS, degree of dehydration, HR, SBP, DBP, urine output, recovery time from acidosis and hospital stay length were insignificantly different between non-AKI and AKI cases.

Table (2): Baseline characteristics of T1DM children with DKA regarding AKI incidence

	Total (n=22)	No AKI (n=5)	AKI (n=17)	P value
Age (years)	8.57 ± 3.28	11.6 ± 0.89	7.68 ± 3.2	0.015*
BMI (kg/m^2)	15.3 ± 3.35	18.25 ± 3.77	14.43 ± 2.77	0.021*
Gender Male	12 (54.55%)	2 (40%)	10 (58.82%)	0.624
Female	10 (45.45%)	3 (60%)	7 (41.18%)	
Family history of DM	15 (68.18%)	4 (80%)	11 (64.71%)	1.000
T1DM				
Newly diagnosed DM	12 (54.55%)	2 (40%)	10 (58.82%)	0.624
Pre-existing DM	10 (45.45%)	3 (60%)	7 (41.18%)	
Tachycardia	14 (63.64%)	2 (40%)	12 (70.59%)	0.309
HTN	8 (36.36%)	0 (0%)	8 (47.06%)	0.115
DKA stage Mild	2 (9.09%)	1 (20%)	1 (5.88%)	0.407
Moderate	6 (27.27%)	2 (40%)	4 (23.53%)	
Severe	14 (63.64%)	2 (40%)	12 (70.59%)	
GCS	13.73 ± 1.61	14.8 ± 0.45	13.41 ± 1.7	0.090
Degree of dehydration (%)	4.1 (1.16 – 10.7)	0 (0 – 3.5)	4.8 (1.9 – 10.7)	0.085
Vital signs				
HR (beats/min)	120.5 ± 24.96	105.2 ± 12.77	125 ± 26.12	0.121
SBP (mmHg)	108.64 ± 15.96	107.4 ± 10.76	109 ± 17.45	0.849
DBP (mmHg)	69.82 ± 9.27	69.4 ± 5.59	69.94 ± 10.24	0.912
Urine output (ml/kg/h)	1.4 (1.1 – 2.1)	1.4 (1.2 – 1.4)	1.4 (1.1 – 2.15)	0.719
Recovery time from metabolic acidosis (h)	29.5 (24.25 – 35)	21 (13 – 24)	31 (27 – 36)	0.085

Data are presented as mean ± SD, median (IQR), or frequency (%).DM: diabetes mellitus, AKI: acute kidney injury, BMI: body mass index, HTN: hypertension, DKA: diabetic ketoacidosis, GCS: Glasgow coma scale, HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, *: significant as P value ≤ 0.05.

Table (3) showed laboratory data in which EBC and eGFR were significantly less in AKI than in non-AKI patients (P value < 0.05). While, platelets were found to be significantly more in AKI than in non-AKI cases (P value = 0.027). Hb, HCT, MCV, MCHC, TLC, pH, $PaCO_2$, HCO_3^-, RBS, Na, corrected Na, and K were insignificantly different between AKI and non-AKI cases.

Table (3): Laboratory data of T1DM children with DKA regarding AKI incidence

Parameters	Total (n=22)	No AKI (n=5)	AKI (n=17)	P value
Hb (g/dl)	12.74 ± 1.93	12.72 ± 1.81	12.75 ± 2.02	0.979
HCT (%)	39.38 ± 5.23	38.96 ± 5.72	39.55 ± 5.26	0.839
MCV (fl)	70.43 ± 16.06	74.34 ± 4.81	69.28 ± 17.02	0.549
MCHC (g/dl)	33.89 ± 5.98	38.06 ± 9.4	32.66 ± 3.8	0.075
Platelets ($x10^9$/L)	383.32 ± 9.71	299.8 ± 73.91	407.88 ± 85.78	0.027*
TLC ($x10^9$/L)	11.4 (9.1 – 21.18)	10 (7.6 – 10)	13.2 (10.3 – 24.4)	0.071
pH	7.04 ± 0.15	7.13 ± 0.11	7.01 ± 0.15	0.085
$PaCO_2$ (mmHg)	15.6 (14 – 25)	14 (14 – 19)	16.2 (14.2 – 27)	0.595
HCO_3 (mEq/L)	4 (3 – 8.4)	4.8 (4.8 – 4.9)	3.3 (3 – 8.9)	0.446
RBS (mg/dl)	501.76 ± 67.31	496.5 ± 54.85	503 ± 71.34	0.867
Na (mEq/L)	127.87 ± 26.39	132.8 ± 5.22	126.42 ± 29.96	0.646
Corrected Na (mEq/L)	138.72 ± 6.08	136.75 ± 2.33	139.29 ± 6.75	0.479
K (mEq/L)	4.6 ± 0.98	4.1 ± 0.82	4.74 ± 1	0.207
EBC (mg/dl)	0.57 ± 0.13	0.71 ± 0.08	0.52 ± 0.11	0.002*
eGFR (mL/min/1.73 m^2)	61.56 ± 23.86	92.89 ± 13.45	52.34 ± 17.47	<0.001*
Length of hospital stay (days)	8 (2 – 19.75)	8 (2 – 14)	8 (2 – 21)	0.880

Range and median: Non-parametric test. *: significant as P value ≤ 0.05. Hb, hemoglobin, HCT: hematocrit, MCV: mean corpuscular volume, MCHC: mean corpuscular hemoglobin concentration, TLC: total leucocytic count, RBS: random blood sugar, EBC: expected baseline creatinine, eGFR: estimated glomerular filtration rate.

Table (4) summarized the different groups serum creatinine levels. Serum creatinine on admission was significantly increased in AKI cases contrasted to non-AKI patients (P value = 0.019), while after 24 and 48 hours it was insignificantly different between non-AKI and AKI cases.

Table (4): Serum creatinine (mg/dl) among T1DM children with DKA

	No AKI (n=5)	AKI (n=17)	P value
On admission	0.88 ± 0.2	1.14 ± 0.2	0.019*
24 hours after hospitalization	0.8 ± 0.17	0.8 ± 0.32	0.998
48 hours after hospitalization	0.6 ± 0	0.66 ± 0.21	0.620
P value‡	0.135	<0.001*	

Data are presented as mean ± SD. DM: diabetes mellitus, AKI: acute kidney injury, *: significant as P value ≤ 0.05, ‡: ANOVA (F) test with post hoc test (Tukey) analysis for comparison between admission, 24, and 48 hours.

In AKI patients, serum creatinine was significantly lower 24 and 48 hours after hospitalization than on admission (P value <0.001), while in non-AKI group, it was insignificantly different between admission, 24 and 48 hours after hospitalization. In univariate regression analysis, BMI was considered a significant AKI risk factor (OR 0.653, 95% CI 0.439 – 0.971, P value = 0.035), while age, time from DM diagnosis, DKA, GCS, degree of dehydration, recovery time from metabolic acidosis, Na, corrected Na, and K were considered insignificant risk factors for AKI (Table 5).

Table (5): Univariate logistic regression analysis of risk factors for AKI in T1DM children with DKA

		OR	95% CI	P value
Age (years)		0.305	0.078 – 1.193	0.088
BMI (kg/m^2)		0.653	0.439 – 0.971	0.035*
Newly diagnosed DM		2.143	0.281 – 16.370	0.463
DKA stage	Mild	Reference	Reference	Reference
	Moderate	2.000	0.078 – 51.596	0.676
	Severe	6.000	0.257 – 140.053	0.265
	GCS	0.242	0.038 – 1.557	0.135
	Degree of dehydration (%)	1.172	0.887 – 1.549	0.264
	Recovery time from metabolic acidosis (h)	1.106	0.966 – 1.265	0.145
	Na (mEq/L)	0.984	0.916 – 1.058	0.662
	Corrected Na (mEq/L)	1.080	0.883 – 1.320	0.456
	K (mEq/L)	2.989	0.607 – 14.725	0.178

DM: diabetes mellitus, AKI: acute kidney injury, BMI: body mass index, DKA: diabetic ketoacidosis, GCS: Glasgow coma scale, OR: odds ratio, CI: confidence interval, *: significant as P value ≤ 0.05

DISCUSSION

This prospective observational study aimed to assess AKI among TIDM children hospitalized with DKA and potential risk factors for AKI. It was carried out at El-Mataria Teaching Hospital from December 2022 to December 2023 on 22 children under 18 years old with T1DM hospitalized with DKA, regarding the ISPAD Clinical Practice Consensus Guidelines 2018. About 17 (77.27%) of the DKA children had AKI on admission. Of the AKI children, 8 (47.06%) had mild AKI, 7 (41.18%) had moderate AKI, and 2 (11.76%) had severe AKI. In accordance, **Yang *et al.*** [6] reported that AKI was detected in 77.8% of 44 hospitalized children (70 total hospitalizations). Furthermore, 50 % of the cases were classed as severe AKI.

A wide AKI frequencies range was reported in earlier retrospective studies that evaluated AKI among DKA children. Episodes of DKA in children became complicated by AKI in 43% of cases as demonstrated by **Myers *et al.*** [7]. AKI was developed in 64% of T1DM children who were hospitalized for DKA in a previous report [8]. At the admission time, 56.5% of children and adolescents with DKA progressed AKI according to **Huang *et al.*** [9]. It is important to mention that one-third of AKI patients have been categorized as having severe AKI. **Hegab *et al.*** [10] discovered that 41.5% of DKA incidents were linked to the AKI progression. In 15.5% of all DKA incidents, moderate to severe AKI was detected. Conversely, **Weissbach *et al.*** [11] and **Baalaaji *et al.*** [12] discovered that AKI was developed in 30% and 35.3% of DKA children respectively.

In 2020, a study of DKA children reported that the AKI risk through subsequent DKA episodes was elevated nine folds when AKI was developed through DKA [7]. These data indicates that AKI episodes may not only elevate the CKD risk but also render the kidneys more susceptible to future AKI.

It is crucial to consider that the method used for measuring blood creatinine levels may have an impact on the AKI incidence as identified by the kidney disease developing global outcomes. Serum creatinine is measured using both the enzymatic method and the Jaffe method. However, the latter is considerably more expensive.

The current study revealed that, DKA stage was insignificantly different between AKI and non-AKI patients. An earlier adult research found that AKI is related to severe DKA in ICU hospitalizations with DKA [13]. **Hegab *et al.*** [10] determined the severity of DKA at hospitalization and showed that severity was significantly increased among DKA episodes with moderate to severe AKI.

This study indicated that age was significantly less in AKI than in non-AKI patients (P value <0.05). This may be explained by the possibility that younger children, particularly those with new-onset diabetes, may not get an immediate diagnosis of DKA, which can lead to more severe dehydration at the hospital admission time. Similarly, **Hegab *et al.*** [10] found a relation between AKI development and younger age.

In our study, newly diagnosed diabetic patients were insignificantly different between AKI and non-AKI cases. This disagrees with **Myers *et al.*** [7] who indicated that AKI was more common among known diabetic contrasted with those with new onset (P<.001). Also, **Huang *et al.*** [9] discovered that the AKI proportion was lower in recently detected diabetes children than in those who were earlier diagnosed. The results may have been influenced by the different criteria and populations for diagnosing AKI in children.

In this study, eGFR was significantly less in AKI than in non-AKI cases (P value <0.05). This agrees with **Huang *et al.*** [9] who demonstrated a suspicious relationship between metabolic acidosis recovery time and eGFR in DKA patients with severe AKI. The more time required to recover from metabolic acidosis, the more decline in eGFR. Additionally, their findings revealed that children with DKA who had severe AKI had renal tubular injury, and that as their renal function decreased, it impacted their ability to correct for

metabolic acidosis. This also agrees with **Chen *et al.*** [13] who reported that DKA cases with AKI exhibited a greater than two-folds decrease in eGFR within one year of discharge when contrasted to non-AKI DKA.

Our study revealed that platelets were significantly increased in AKI children contrasted to non-AKI children (P value =0.027). On the contrary, **Yang *et al.*** [6] indicated that there was no significant difference between AKI and non-AKI cases as regards platelets.

It is difficult to correctly assess these patients' volume status, which is defined as a percentage of body weight loss. Yet, in this research, there was no a significant difference in the clinical assessment of dehydration (P = 0.264). Similarly, **Yang *et al.*** [6] reported that there was no significant difference in hydration state. These findings contradict the results of **Huang *et al.*** [9]**,** who stated that AKI children had higher initial HRs and more severe dehydration at hospital admission. While, HR, SBP, DBP, urine output, recovery time from acidosis, Hb, HCT, MCV, MCHC, TLC, PH, $PaCO_2$, HCO_3, RBS, Na, corrected Na, and K were insignificantly different between non-AKI and AKI patients. This is slightly in agreement with **Yang *et al.*** [6], who discovered no significant difference in serum sodium levels between cases without and with AKI (P=0.718). Nevertheless, Na level was significantly elevated in the severe AKI group contrasted to in the no/mild AKI group. The serum Na level in DKA cases is typically low upon admission due to the water osmotic flux from the intracellular to the extracellular space in the hyperglycemia presence. The clinical dehydration evaluation, which is a body weight loss percentage, did not exhibit a significant difference (P = 0.198). Additionally, the authors noted that children with AKI exhibited elevated white blood cell counts. In DKA, leukocytosis (WBC around 10,000–15,000/mm^3) is a frequent result at admission and may not necessarily suggest an infection. This is ascribed to pressure and could be associated with an elevation in stress hormone levels. Even though a definitive infectious process was not determined, the median WBC count was significantly lower in the no AKI than in the AKI group of DKA children.

In addition, **Huang *et al.*** [9] demonstrated that the severity of AKI was correlated with higher levels of blood glucose, corrected sodium, and HR. These results indicated that volume depletion is a critical factor in the AKI development in DKA children.

Hegab *et al.* [10] discovered no distinction in serum sodium levels between patients without and with AKI; although, sodium values in this study were not adjusted for serum glucose levels. A new pediatric study has demonstrated that AKI is linked to severe acidosis and volume depletion markers [10]. They presupposed that AKI in DKA children is the result of a prerenal mechanism. **Hursh *et al.*** [8] found that more severe AKI was associated with an elevated HR, corrected Na level (≥ 145 mEq/L), and reduced HCO_3^- level (< 10 mEq/L) at hospital admission. The presence of hyperglycemia in the presence of an elevated or even normal serum Na level suggests a significant degree of free water loss [15]. According to **Lopes *et al.*** [15], volume depletion is the primary AKI cause in DKA children.

In an earlier report, it was shown that a less Ph level at admission in adults were a strong predictor of the lengthier time needed for DKA resolution [16]. Furthermore, another report has demonstrated that metabolic acidosis recovery time is correlated with the insulin dosage and the DKA severity [17, 18].

In this study, serum creatinine on admission was significantly elevated in AKI than in non-AKI cases (P value =0.019), while after 24 and 48 hours it was insignificantly different between AKI and non-AKI cases. In AKI group, serum creatinine was significantly less 24 and 48 hours after hospitalization than on admission (P value <0.001). While, in non-AKI group, it was insignificantly different between admission, 24 and 48 hours after hospitalization. **Hursh *et al.*** [8] indicated that higher serum creatinine levels at admission were associated with an elevation developing AKI risk.

In our study, in univariate regression analysis, BMI was considered as a significant risk factor for AKI (95% CI 0.439 – 0.971, OR 0.653, P value =0.035). In harmony, **Huang *et al.*** [9] reported that BMI may be associated with the severity of AKI, where higher BMI seems to be associated with a lower risk or lower AKI severity.

LIMITATIONS

In pediatric individuals, baseline serum creatinine values were unavailable, necessitating the estimation of a baseline value. The optimal eGFR for establishing a baseline creatinine level was a topic of debate. In order to maintain consistency with other pediatric AKI studies, we implemented an eGFR of 120 mL/min/1.73 m^2.

CONCLUSION

This study found high AKI incidence with 77.27% of children developed AKI during their hospitalization. Most AKI cases were mild to moderate in severity. The estimated glomerular filtration rate (eGFR) was significantly less in AKI children, while platelets were significantly greater in AKI children. The duration of diabetes (newly diagnosed vs. established) was not a significant risk factor for AKI. However, in the univariate regression analysis, BMI emerged as a significant risk factor for AKI, with a higher BMI associated with a lower AKI development risk.

No funding.

No conflict of interest.

REFERENCES

1. **Wolfsdorf J, Glaser N, Agus M *et al.* (2018):** ISPAD Clinical Practice Consensus Guidelines 2018: Diabetic

ketoacidosis and the hyperglycemic hyperosmolar state. Pediatric Diabetes, 27: 155-177.

2. **Basu R (2020):** Dynamic biomarker assessment: a diagnostic paradigm to match the AKI syndrome. Frontiers in Pediatrics, 7: 535. https://doi.org/10.3389/fped.2019.00535
3. **Feldman-Kiss D, Li D, Cleve R *et al.* (2022):** Interference of ketone bodies on laboratory creatinine measurement in children with DKA: a call for change in testing practices. Pediatric Nephrology, 37 (5): 1347-53.
4. **Schwartz G, Haycock G, Edelmann Jr, C *et al.* (1976):** A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine. Pediatrics, 58 (2): 259-263.
5. **Kellum J, Lameire N, Aspelin P *et al.* (2012):** Kidney disease: improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. Kidney International Supplements, 2 (1): 1. https://doi.org/10.1038/kisup.2012.1
6. **Yang E, Lee H, Oh K *et al.* (2021):** Acute kidney injury in pediatric diabetic ketoacidosis. Indian Journal of Pediatrics, 88: 568-573.
7. **Myers S, Glaser N, Trainor J *et al.* (2020):** Frequency and risk factors of acute kidney injury during diabetic ketoacidosis in children and association with neurocognitive outcomes. JAMA Network Open, 3 (12): e2025481. doi: 10.1001/jamanetworkopen.2020.25481.
8. **Hursh B, Ronsley R, Islam N *et al.* (2017):** Acute kidney injury in children with type 1 diabetes hospitalized for diabetic ketoacidosis. JAMA Pediatrics, 171 (5): e170020. doi: 10.1001/jamapediatrics.2017.0020.
9. **Huang S, Huang C, Lin C *et al.* (2020):** Acute kidney injury is a common complication in children and adolescents hospitalized for diabetic ketoacidosis. Plos One, 15 (10): e0239160. doi: 10.1371/journal.pone.0239160.
10. **Hegab A, Khalil F, Abosedera M (2022):** Incidence and factors associated with acute kidney injury among children with type 1 diabetes hospitalized with diabetic ketoacidosis: a prospective study. Pediatric Diabetes, 23 (6): 783-791.
11. **Weissbach A, Zur N, Kaplan E *et al.* (2019):** Acute kidney injury in critically ill children admitted to the PICU for diabetic ketoacidosis. A retrospective study. Pediatric Critical Care Medicine, 20 (1): 10-14.
12. **Baalaaji M, Jayashree M, Nallasamy K *et al.* (2018):** Predictors and outcome of acute kidney injury in children with diabetic ketoacidosis. Indian Pediatrics, 55: 311-314.
13. **Chen J, Zeng H, Ouyang X *et al.* (2020):** The incidence, risk factors, and long-term outcomes of acute kidney injury in hospitalized diabetic ketoacidosis patients. BMC Nephrology, 21 (1): 48. doi: 10.1186/s12882-020-1709-z.
14. **Umpierrez G, Davis G, ElSayed N *et al.* (2024):** Hyperglycemic Crises in Adults with Diabetes: A Consensus Report. Diabetes Care, 67 (8): 1455-1479.
15. **Lopes C, Pinheiro P, Barberena L *et al.* (2017):** Diabetic ketoacidosis in a pediatric intensive care unit. Journal Pediatr., 93 (2): 179-184.
16. **Lee M, Calder G, Santamaria J *et al.* (2018):** Diabetic ketoacidosis in adult patients: an audit of factors influencing time to normalisation of metabolic parameters. Internal Medicine Journal, 48 (5): 529-534.
17. **von Oettingen J, Rhodes E, Wolfsdorf J (2018):** Resolution of ketoacidosis in children with new onset diabetes: evaluation of various definitions. Diabetes Research and Clinical Practice, 135: 76-84.
18. **Edge J, Nunney I, Dhatariya K (2016):** Diabetic ketoacidosis in an adolescent and young adult population in the UK in 2014: a national survey comparison of management in paediatric and adult settings. Diabetic Medicine, 33 (10): 1352-1359.