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ORIGINAL ARTICLE

Acute Kidney Injury in Children with Type 1 Diabetes Mellitus Hospitalized for Diabetic Ketoacidosis: A Retrospective Study

Shaila Pachapure,[1] Jasmine Kandagal,[2] Manjunath Revanasiddappa,[2] Kavita Konded[2]

[1]*Jawaharlal Nehru Medical College (KAHER - KLE Academy of Higher Education and Research), Belagavi, Karnataka, India*
[2]*Shri Dharmasthala Manjunatheshwara College of Medical Sciences and Hospital, Shri Dharmasthala Manjunatheshwara University, Dharwad, Karnataka, India*

Abstract

Objectives. Diabetic ketoacidosis (DKA) is the most common initial presentation in children with newly diagnosed type 1 diabetes. Severe dehydration/acidosis, shock at admission, and hyperchloremia contribute to acute kidney injury (AKI). This retrospective study was done to determine the proportion of children hospitalized for DKA who had AKI and to compare clinical parameters between DKA children with AKI and without AKI to identify the risk factors associated with AKI.

Methodology. A retrospective review of all DKA admissions with type 1 diabetes was done. AKI was diagnosed as per KDIGO-2012 criteria. The analysis was done using a Chi-square test to assess the association between the status of AKI and other parameters. The Independent t-test was applied for comparison with the mean score between the No AKI / AKI group for numerical variables with normal distribution. A multivariate logistic regression analysis was performed to compare clinical parameters between both groups.

Results. Out of 32 children with DKA, 13 (40.63%) developed AKI. Among them, 9 had AKI at admission and 4 children developed AKI within the first 48 hours of admission. Optimum fluid management resolved AKI in 10 patients, but 3 of them required dialysis. Parameters like higher heart rate (p = 0.0390), higher respiratory rate (p = 0.0402), high leukocyte count (p = 0.0005), severe hyperglycemia (p = 0.0204), severe acidosis (p = 0.0001), hyperchloremia (p = 0.016) and shock at admission (p = 0.0001) were present in children with DKA and AKI.

Conclusion. In our study, a high proportion of children with DKA had AKI, which causes prolonged acidosis and hospital stay. Hence, comparing clinical parameters between both groups helps in identifying risk factors associated with AKI in persons with type 1 diabetes with DKA.

Key words: diabetic ketoacidosis, acute kidney injury, ketone bodies, hyperchloremia

INTRODUCTION

Diabetic ketoacidosis (DKA) is the most common initial presentation in new-onset type 1 diabetes in children with a frequency of around 15-70% across various populations.[1] Recurrence of DKA occurs in those with poor control, mismanagement during sick days, or interruption of delivery of insulin in insulin pumps. The leading cause of morbidity and mortality in DKA is due to cerebral injury. Cerebral edema accounts for about 60–90% of deaths, others being electrolyte imbalance, venous thrombosis, sepsis, renal injury, etc.[1] Acute kidney injury is associated with prolonged hospital stay and it can predispose to hypertension and chronic kidney disease (CKD). DKA with complications leads to prolonged hospital stay and higher treatment costs. Ketoacidosis resolves with correction by fluid and insulin therapy. However, the presence of AKI leads to the persistence of acidosis as renal function gets compromised in severe and prolonged DKA. Several risk factors contribute to AKI in DKA. The most common being shock at admission, severe dehydration, severe acidosis, and hyperchloremia. There are no validated pharmacological interventions for AKI, hence prevention and early detection are key in management. Studies pertaining to the occurrence of AKI are less studied in DKA patients. Interpretation of creatinine varies in DKA as ketones interfere in creatinine measurement.[2] Even osmotic diuresis in DKA delays early diagnosis of oliguria. This retrospective study aimed to determine the proportion of children who develop AKI and to compare clinical parameters between children with DKA and AKI and those without AKI to identify the risk factors associated with AKI.

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Corresponding author: Shaila Pachapure, MD
Consultant Pediatric Endocrinologist and Assistant Professor,
Department of Pediatrics, KAHERs – Jawaharlal Nehru Medical College,
JNMC KLE University Campus, Nehru Nagar, Belagavi, Karnataka 590010, India
E-mail: drshailams@gmail.com
ORCiD: https://orcid.org/0000-0002-9328-3024

METHODOLOGY

A retrospective review of all case records of DKA admissions was done for children (<18 years) who were admitted from July 2018 to June 2021 at SDM College of Medical Sciences and Hospital, Dharwad. After ethical committee clearance (Ref. SDMIEC/2021/83), data of all the cases admitted from the above 3-year period were collected. Diagnosis of DKA and its severity as mild, moderate, and severe DKA was defined as per ISPAD guidelines. The initial demographic details, clinical features (including vital signs and urine output), and anthropometry values were retrieved from case records in a structured format. Laboratory investigations like complete hemogram, venous blood gas analysis, and serum electrolytes were recorded at admission and were repeated after 4 to 6 hours and also on a case-to-case basis until recovery. An initial bolus of 10 - 20 ml/kg of normal saline (NS) followed by an additional 10 ml/kg infusion was given based on the presence of shock. Fluids were calculated according to severity of dehydration and maintenance fluids were added to replace the deficit over 24 to 48 hours (as per ISPAD 2018 guidelines). Inotropes and bicarbonates were considered in special situations as per standard guidelines.[1,3] Intravenous insulin was used in all patients except in three children who were managed with subcutaneous insulin as they had mild DKA. Insulin infusion was started at 0.05 – 0.1 U/kg/hour after 1st hour of NS bolus. Intravenous fluids along with insulin infusion were adjusted as per serial serum electrolytes and blood sugar values during hospital stay. The duration of resolution of acidosis, duration of pediatric intensive care unit (PICU) and hospital stay were noted. Other associated complications like cerebral edema, deep vein thrombosis and foot drop were noted. Acute kidney injury was defined and staged as according to 2012 guidelines of Kidney Disease / Improving Global Outcomes Criteria (KDIGO). In the KDIGO criteria, AKI is defined as a creatinine level >1.5 times the expected baseline creatinine value.[4] As we did not have baseline creatinine data of the patients at admission, we calculated the expected baseline creatinine values using an estimated glomerular filtration rate of 120 ml/min/1.73 sq. m. and patients height using Schwartz formula.[5] Stage 0 AKI was defined as creatinine value <1.5 times, stage 1 AKI as creatinine 1.5-2 times, stage 2 AKI as 2-3 times and stage 3 AKI as creatinine >3 times the baseline creatinine. Those children who developed AKI during hospital stay were also taken into consideration and were staged based on progression. Creatinine estimation was done using the alkaline picrate-kinetic, International Federation of Clinical Chemistry-Isotope Dilution Mass Spectrometry (IFCC-IDMS) standardized method.

Statistical procedures

After the collection of data, the descriptive statistics with means/standard deviation were calculated for numerical variables; frequency, and percentages for categorical variables. The analysis was carried out by using Chi-square/ Chi-square with the Yates correction test for association between the status of AKI with other parameters. The Independent t-test was applied for comparison with mean score between with/without AKI group for numerical variables with normal distribution. The Kolmogorov-Smirnov test was performed and tested for the normality of all variables. Then the Mann-Whitney U test was used for numerical parameters with skewed distribution. The simple and multivariable logistic regression model was applied to compare clinical parameters between children with DKA and AKI and those without AKI to identify the risk factors associated with AKI. A statistical significance was set at a 5% level of significance (p <0.05). The statistical software used are SPSS 20 and STATA 16.0.

RESULTS

A total of 62 children with type 1 diabetes were admitted to our hospital during the study period, but only 32 episodes of DKA were noted. Among those children without DKA (n = 30), few were newly diagnosed without ketosis and others were referred from other hospitals for post-DKA management.

Out of the 32 DKA episodes, 24 (75%) were new-onset diabetes and 8 (25%) were known diabetes cases (Table 1). Out of these 8 episodes, only one child had two episodes of DKA. The median age among patients with DKA was 10.2 years (range 1.58 – 13.92 years), with 15 (46.88%) being male and 17 (53.13%) being female (Table 1). Four of the 32 were diagnosed in another institution and they received appropriate IV fluids but not intravenous insulin bolus. All those old patients were on a basal-bolus regimen. The causes for the precipitation of DKA in established diabetes cases are poor compliance to medication and missed insulin doses. The majority of the children presented with severe DKA i.e., 24 (75%), mild and moderate being 3 (9.38%) and 5 (15.63%) respectively.

Out of 32 children, a total of 13 (40.63%) children were diagnosed with AKI. Among these, AKI was noted mainly in 1 (20%) case out of 5 moderate DKA and 12 (50%) cases in 24 severe DKA categories. Out of 13, 9 children had AKI at onset, others showed increasing creatinine within 48 hours of admission. The number of patients categorized into stages 0, 1, 2, and 3 of AKI as per KDIGO guidelines at admission were as follows: 4 (30.7%), 7 (53.84%), 1 (7.69%), and 1 (7.69%). The same at 48 hours were as follows: 6 (46.15%), 0 (0%), 4 (30.7%) and 3 (23.08%) respectively. Four patients with normal creatinine at admission being 0.73, 0.88, 1.01, and 0.91 showed increasing trends up to a maximum of 2.11, 5.77, 1.71, and 2.87 at 54, 108, 90, and 48 hours respectively. Oliguria was noted in only 4 patients. A total of 14 out of 32 (43.75%) patients presented in shock; 12 of these developed AKI (Yates corrected Chi-square – 32.00) (p = 0.0001) (Table 2).

AKI was managed by optimization of volume status in ten patients. Three of them required dialysis. Indications were as follows: persistence of low pH (less than 6.9) at 24 hours

in the first case, increase in creatinine and recurrence of acidosis in another case, and increase in creatinine along with oliguria which developed at 24 hours followed by hyperkalemia at 60 hours in the last case. The number of dialysis sessions was 5, 1, and 2, respectively.

A significant difference was observed between AKI and non-AKI groups for the following parameters by independent t-test (Table 3): higher heart rate (p = 0.0390), higher respiratory rate (p = 0.0402), high leukocyte count (p = 0.0005), severe hyperglycemia (p = 0.0204), severe acidosis (p = 0.0001), hyperchloremia (p = 0.016), time for resolution of acidosis (p = 0.0002), duration of PICU stay (p = 0.0002) and duration of hospital stay (p = 0.0022).

Multivariable regression analysis showed significance only for metabolic acidosis (low pH). However, other parameters didn't show any significance.

Other complications observed in our cases were as follows: cerebral edema in 3 patients, foot drop in 2 patients, and deep vein thrombosis in 1 patient. Foot drop was observed in 2 children who had AKI too. Phosphorus was very low (<0.5 mg/dl) in these children with CPK being 14,441 U/L and 3725 U/L respectively.

DISCUSSION

Diabetic ketoacidosis is the most often initial presentation in children with new-onset type 1 diabetes. The incidence of type 1 diabetes in children is on the rise as reported by various countries.[1] We found a higher incidence of DKA

Table 1. Association between gender and past episodes of DKA with AKI

	Without AKI	%	With AKI	%	Total	%	Chi-square	*p*-value
Gender								
Male	9	60.00	6	40.00	15	46.88	0.0050	0.9460
Female	10	58.82	7	41.18	17	53.13		
Past episodes of DKA								
No	12	50.00	12	50.00	24	75.00	2.1161#	0.1457
Yes	7	87.50	1	12.50	8	25.00		
Total	19	59.38	13	40.63	32	100.00		

#applied Yates corrected chi-square

Table 2. Association between AKI with shock at admission

Shock at admission	Without AKI	%	With AKI	%	Total	%	Yates corrected Chi-square	*p*-value
No	17	94.44	1	5.56	18	56.25	32.0000	0.0001*
Yes	2	14.29	12	85.71	14	43.75		

*p <0.05

Table 3. Comparison of DKA without AKI and with AKI with different clinical parameters by independent group t test

Clinical parameters	Without AKI (n=19) Mean (SD)	With AKI (n=13) Mean (SD)	t-value	*p*-value
Age (years)	9.59 (3.15)	9.92 (3.17)	-0.2849	0.7777
Heart Rate (HR)	114.84 (22.05)	130.31 (16.14)	-2.1593	**0.0390**
Systolic Blood Pressure (SBP)	104.32 (10.78)	98.23 (14.56)	1.3602	0.1839
Diastolic Blood Pressure (DBP)	65.47 (7.24)	64.23 (15.97)	0.2989	0.7670
Respiratory Rate (RR)	34.42 (10.66)	42.08 (8.68)	-2.1451	**0.0402**
Haemoglobin (g/dl)	14.36 (1.33)	14.10 (1.48)	0.5322	0.5985
Packed Cell Volume (%) (PCV)	41.22 (3.87)	42.17 (4.60)	-0.6309	0.5329
Total Cell Count (TC) (cells/µL)	16975.2 (8676.7)	31863.0 (12805.5)	-3.9301	**0.0005**
Urea (mg/dl)	25.16 (7.79)	39.46 (18.21)	-3.0557	**0.0047**
Creatinine (mg/dl)	0.75 (0.13)	1.16 (0.67)	-2.6571	**0.0125**
Blood sugar at admission (mg/dl)	475.00 (172.19)	627.46 (174.27)	-2.4481	**0.0204**
Corrected sodium (mEq/L)	136.68 (3.44)	139.38 (6.78)	-1.4821	0.1487
Chloride (mEq/L)	91.37 (20.24)	96.62 (7.29)	-0.8917	0.3796
pH	7.17 (0.13)	6.87 (0.11)	6.5859	**0.0001**
Bicarbonate (mmol/L)	6.56 (5.19)	3.32 (2.20)	2.1165	**0.0427**
Glycated Hemoglobin (Hb%)	13.25 (2.85)	14.37 (2.08)	-1.2113	0.2352
Highest Chloride (mEq/L)	103.68 (5.19)	112.54 (9.25)	-3.4658	**0.0016**
Time for resolution of acidosis (hours)	19.63 (12.13)	73.77 (53.88)	-4.2550	**0.0002**
Duration Of PICU stay (days)	1.58 (0.69)	7.00 (5.58)	-4.2172	**0.0002**
Duration of hospital stay (days)	6.53 (3.08)	13.23 (7.96)	-3.3436	**0.0022**

*p <0.05

among newly diagnosed cases i.e., in 24 (75 %) cases compared to various studies reporting incidence ranging from 47.9 % to 71%.[6–9] Most episodes of DKA in known cases of diabetes was due to poor compliance and improper sick day management.

Organ dysfunction, commonly involving the brain and kidney in DKA has been reported by various authors.[1] Several theories proposed for cerebral injury include osmotic shifts, rapid rehydration, hypoxia/ischemia, severe acidosis, and inflammatory mechanisms.[7,10] Normal saline is the most preferred solution in the initial 4 -6 hours of management as it is isotonic to plasma and use of lower osmolar solutions can precipitate cerebral injury.[1] However, it has a high concentration of chloride (154 mEq/L), which is 40-50% more than plasma.[11] This high chloride causes renal vasoconstriction predisposing to AKI. Proposed mechanisms for renal injury by hyperchloremia consist of renal vasoconstriction which causes cortical hypoperfusion, interstitial edema, intracapsular hypertension, and reduction of GFR.[10,12,13] But, there is still no documented evidence to support the use of balanced fluid (chloride restrictive) in the initial management of DKA. An adequate balance of fluids is mandatory to avoid complications.

In our study, AKI was present at admission in 9 (69.2%) cases before treatment of DKA. The remaining 4 (30.8%) of them developed during hospital stay. The proportion of AKI in our study was 40.63%, which is almost similar to previous reports by others ranging from 35% to 64.2%.[6,8,14] Most of the AKI was seen with new onset and severe DKA except for one who had past episodes of DKA. This finding was similar to other reports by Baalaaji M et al., and Tiwari LK et al.[6,15]

Acute kidney injury has been reported to occur within 12-24 hours after DKA treatment similar to cerebral injury.[16] The development of AKI during treatment even after improving hydration status is an indication to search for causes other than prerenal factors like hypovolemia.[8] Our study found that AKI was associated with shock at admission, hyperchloremia during treatment, severe hyperglycemia, and severe acidosis.

Shock at admission showed a significant association with AKI (Table 2). Metabolic acidosis also showed a significant association with AKI (Table 4). None of the parameters showed any statistically significant association with AKI in multivariable analysis. The reason may be due to a smaller sample size. Hence, severe acidosis and shock at admission are important risk factors associated with AKI.

In our study, creatinine estimation was performed using the alkaline picrate-kinetic, IFCC-IDMS standardized method; however, it is not commonly done. Standardization of creatinine assay by Isotope Dilution Mass Spectrometry (IDMS) will improve the analysis and reduce the interference by interfering substances. Hence, creatinine estimation has to be standardized to appropriately compare the prevalence of AKI between various studies.[17] Urine output may not be the sole criteria for diagnosis of AKI in DKA as these children have initial polyuria. Thus, diagnosis of AKI based on only urine output is difficult. Hence, studies of AKI in patients with DKA have to consider these factors for accurate diagnosis.

Table 4. Multivariable logistic regression analysis of the incidence of AKI by clinical parameters

Factors	Adjusted OR (95% CI)	*p*-value*
Heart Rate (HR)	1.16 (0.68 – 2.00)	0.5780
Respiratory Rate (RR)	1.21 (0.87 – 1.67)	0.2530
Highest chloride	1.17 (0.80 – 1.71)	0.4260
pH	0.00 (0.00 – 0.04)	0.010*
RBS	1.01 (0.99 – 1.03)	0.3910
Corrected sodium	1.00 (0.99 – 1.00)	0.3170

*p <0.05

We observed a higher number of cases in severe DKA than in other categories. The study setting was in a tertiary referral hospital where in only sick children might have been referred. The higher number of AKI observed in our study could be due to late referral and severe DKA. Hence, early identification and management of shock and severe acidosis can prevent AKI. Early referral to the tertiary center may be considered in patients who have features that are harbingers of AKI progression like severe acidosis (pH <6.9) and shock at admission.

The variation in the incidence of AKI between various studies may be due to the use of different diagnostic criteria for AKI, the population studied, time of referral to the study center, regional referral guidelines, and sample size.

Identifying the root cause of AKI in DKA in resource-limited settings is highly relevant because of the increasing incidence of diabetes in children. Prospective, multi-center studies can identify the risk factors for AKI; management of DKA, and red flag signs in those children meriting early referral to a tertiary center. Other causes of AKI like the type of fluids used in the management and presence of sepsis were not included in our study. Smaller sample sizes from a single center are limitations of the study. Hence, the results regarding the risk factors need to be verified by other studies with larger populations of patients with DKA. Use of only KDIGO criteria can under or over-diagnose the AKI, hence employment of other criteria and their comparison with each other could have been done.

CONCLUSION

The management of DKA is very complex. Adequate fluid management is important to avoid complications. Severe acidosis predisposes to organ dysfunction. In our study, we found a high proportion of children with DKA had AKI, which causes prolonged acidosis and hospital stay. Hence, early identification of risk factors and prompt treatment prevent severe complications like AKI and limit the duration of hospital stay.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

CRediT Author Statement
SP: Conceptualization, Methodology, Formal analysis, Writing – original draft preparation, Writing – review and editing; **JK:** Conceptualization, Methodology, Writing – original draft preparation, Writing – review and editing; **MR:** Validation, Formal analysis, Writing – review and editing; **KK:** Writing – review and editing, Supervision, Project administration.

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Datasets generated and analyzed are included in the published article.

Author Disclosure
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