

CLINICO-DEMOGRAPHIC, BIOCHEMICAL PROFILE AND OUTCOME IN CHILDREN WITH DIABETIC KETOACIDOSIS (DKA) IN A TERTIARY CARE MEDICAL COLLEGE, RURAL TELANGANA

K. Sailaja¹, Zion Eluzai Gaddam¹, Mounica Reddy Banda², N. R. Sahasa³, Sanjay Earna³, Anisha Yalamanchili³, Puja Devi Kalla³, Navya Gogula³

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Corresponding Author:

Dr. N. R. Sahasa,

Email:

mahabubnagar.sahasareddy72@gmail.com

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¹Professor, Department of Pediatrics, SVS Medical College, Yenugonda, Mahbubnagar, Telangana, India

²Senior Resident, Department of Pediatrics, SVS Medical College, Yenugonda, Mahbubnagar, Telangana, India

³Junior Resident, Department of Pediatrics, SVS Medical College, Yenugonda, Mahbubnagar, Telangana, India

Abstract

Background: Type 1 diabetes mellitus is one of the most common chronic endocrinology disorders in children and adolescents. Delayed diagnosis or poorly managed T1DM may present as diabetic ketoacidosis and it is a common Pediatric endocrine emergency. Timely diagnosis and proper management of DKA can prevent serious morbidity and mortality. The objective of the study was to assess the demographic profile, clinical presentation upon admission, management, and complications in patients diagnosed with diabetic ketoacidosis (DKA). **Materials and Methods:** A prospective observational study was conducted among children admitted to the Pediatric Intensive Care Unit of SVS Medical College and Hospital, Mahbubnagar, Telangana, from April 2021 to March 2024. Children aged between 1 year to 16 years presented with diabetic ketoacidosis were included in the study. The clinical, demographical, biochemical profile, management and outcome were collected using a pre-designed pro forma. **Result:** In this study of 32 children with diabetic ketoacidosis (DKA), 62.5% were newly diagnosed with type 1 diabetes mellitus (T1DM) at presentation. The mean age was 10.94 ± 3.94 years, with 71.9% of participants from rural areas. Common symptoms included vomiting (81.3%), polyuria (68.8%), polydipsia (65.6%), and abdominal pain (68.8%). Key laboratory findings revealed a mean pH of 7.11 ± 0.15 , bicarbonate levels of 10.78 ± 2.25 mEq/L, and blood glucose at 487.40 ± 80.79 mg/dL. Most children exhibited moderate to severe dehydration (81.3%) with moderate acidosis in 46.9%, severe acidosis in 34.4%, and mild acidosis in 18.7% of cases. The mean duration for acidosis correction was 31.74 ± 4.87 hours, with new-onset cases taking longer (34.2 ± 4.06 hours) compared to known T1DM cases (27.58 ± 2.94 hours) ($p=0.000015$). Transition to subcutaneous insulin occurred at an average of 29.78 ± 5.03 hours, with new-onset cases requiring more time (31.95 ± 4.68 hours) than known cases (26.17 ± 3.24 hours) ($p=0.0003$). The average hospital stay was significantly longer for new-onset cases (6.8 ± 0.77 days) compared to known cases (5.17 ± 0.58 days) ($p=0.00001$). All patients fully recovered, achieving a 100% survival rate. **Conclusion:** This observational prospective study of 32 children with diabetic ketoacidosis (DKA) highlights the importance of timely management, achieving a 100% recovery rate. Early recognition and intervention, particularly in newly diagnosed type 1 diabetes mellitus cases, are crucial due to their more severe metabolic disturbances. The findings emphasize the need for individualized treatment approaches and affirm the efficacy of current therapeutic strategies in managing DKA.

INTRODUCTION

Diabetic ketoacidosis (DKA), is the most common yet preventable complication of type I diabetes

mellitus (DM). It is defined as Hyperglycemia (blood glucose >11 mmol/L [≈ 200 mg/dl]), Venous pH <7.3 or serum bicarbonate <18 mmol/L(C), Ketonemia

(blood β -hydroxybutyrate ≥ 3 mmol/L) (C) or moderate or large ketonuria.^[1]

Insulin is synthesised by beta cells of the pancreas. A multifactorial spectrum ranging from genetic, environmental, drugs and infectious agents results in progressive destruction of beta cells. An absolute or relative lack of insulin and the consequent unrestricted flux of carbohydrate, amino acid and lipid nutrients to the blood. Due to a lack of insulin, cells are deprived of glucose. The counter-regulatory hormones (glucagon, glucocorticoids, catecholamines, and growth hormones) break down the triglycerides for energy, leading to increased glycerol and fatty acids to fulfil the body's requirements resulting in ketosis. Hyperglycaemia causes osmotic diuresis, with a loss of free water and electrolytes resulting in dehydration.^[2,3]

The prevalence of type 1 diabetes mellitus (T1D) is increasing worldwide. As per the latest update from the International Diabetes Federation 2020, among all diabetic patients, 10% constitute type 1 diabetes mellitus.^[4] The current prevalence of T1DM in India among 5–16-year-old children is 22.2/1,00,000 populations. Mortality from DKA is high at 13.2% in India while it is 0.15–0.31% in developed countries.^[5]

The risk of developing DKA in established T1DM children is 1 to 8% per patient per year,^[6] whereas, the annual incidence of DKA among children with type I DM is 1%-5% in the western series and accounts for 8%-28% of all primary admissions for DM to a hospital.^[7]

The mortality from DKA in developed countries has come down to 0.15-0.31%, but it still ranges between 3.4% and 13.4% in developing countries.^[8]

The classic clinical manifestations of new-onset diabetes in children reflect hyperglycemia and a catabolic physiological state. These include polyuria, polydipsia, polyphagia, and, in later stages, weight loss, fatigue, or recurrent infections. Additionally, abdominal tenderness, abdominal pain, nausea, vomiting, and dehydration are common presentations of Diabetic Ketoacidosis (DKA).^[9] Risk factors for DKA in patients with known diabetes include insulin omission, poor metabolic control, previous episodes of DKA, acute gastroenteritis with persistent vomiting, and inability to maintain hydration.^[10]

Treatment options for severe diabetic ketoacidosis is a multidisciplinary approach that emphasises correcting fluid and electrolyte imbalances, restoring insulin sensitivity, and preventing complications.^[11] Insulin therapy inhibits the production of keto acids and facilitates their metabolism, thereby helping correct acidosis.^[8]

Very few clinical data on the clinical, demographic, biochemical profile, and outcome of DKA in children are available from the rural parts of Telangana. We analysed our data from 2 years at a tertiary care hospital.

MATERIALS AND METHODS

This was a hospital-based prospective observational study conducted in the Pediatric Intensive Care Unit of SVS Medical College and Hospital, Mahbubnagar, Telangana, from April 2021 to March 2024.

All the children aged between 1 to 16 years who met the diagnostic criteria of Diabetic Ketoacidosis were included in the study. Diagnostic criteria included hyperglycemia (blood glucose >11 mmol/L [≈ 200 mg/dl]), venous pH <7.3 , or serum bicarbonate <18 mmol/L, ketonemia (blood β -hydroxybutyrate ≥ 3 mmol/L), or moderate to large ketonuria. The severity of DKA was categorized based on the degree of acidosis: Mild DKA was characterized by a venous pH of <7.3 and/or an HCO_3 level of <15 mmol/L, moderate DKA was characterized by a venous pH of <7.2 and/or an HCO_3 level of <10 mmol/L and severe DKA was characterized by a venous pH of <7.1 with or without an HCO_3 level <5 mmol/L (10). Diabetic ketoacidosis was managed according to the International Society for Pediatric and Adolescent Diabetes (ISPAD) guidelines. Fluid therapy for children with shock was initiated with a 10 ml/kg bolus of normal saline, followed by continuous fluid therapy to correct underlying severe dehydration in moderate to severe DKA cases (a deficit of 80-100 ml/kg). This deficit and maintenance fluid for 48 hours were distributed evenly over 48 hours. Insulin infusion commenced at a rate of 0.1 units/kg/hour. Improvement in acidosis on ABG (pH >7.3), blood sugar level normalisation and clinical conditions stabilisation were documented.

The infusion ceased based on the resolution of DKA displayed by stable clinical parameters and normalization of biochemical values (blood pH >7.3 , plasma bicarbonate >15 mmol/L, and blood sugar around 250 mg/dl). Regular insulin (Human Actrapid) at 1 U/kg/day (in 4 divided doses) was administered 30 minutes before halting the insulin infusion. Subsequently, children transitioned to a split regimen (NPH + Regular insulin) in younger children and a basal-bolus regimen in older children (Glargine + Regular insulin). The adverse outcome was defined as death or refusal of treatment. All patients were monitored every hour for vitals and clinical signs (heart rate, respiratory rate, blood pressure, urine output, oxygen saturation, sensorium, headache, vomiting), every 2 hours for blood sugar and every 4 hours for venous blood gas analysis, serum electrolytes. The renal function test was assessed every 12 hours. A modified Kuppuswamy scale was used to assess socioeconomic status. Blood glucose was measured in a hospital laboratory from the venous sample. HbA1c was measured using an automated analyser by Biorad D10.

Data was entered in Excel and analyzed by using SPSS version 20. The quantitative variables were presented in the form of mean, and standard deviation. Categorical variables were expressed in frequency and percentage. The p-value was calculated using Fisher exact test and using T-test.

The study proposal was approved by the hospital's Institutional Research Ethical Committee.

Data collection: Children were examined, and information regarding clinical presentation, demographics, and socio-economic status using a Modified Kuppaswamy scale was recorded. Factors predisposing to diabetic ketoacidosis, biochemical data (including capillary blood sugar levels, venous blood gas, plasma or urine ketone levels, HbA1c levels, serum electrolytes, renal function tests, complete blood counts, and cultures of blood or other body fluids), as well as treatment details regarding fluid management, insulin therapy, antimicrobial therapy, and mechanical ventilation, were recorded using a pre-designed proforma. Confidentiality of the collected data was strictly maintained.

RESULTS

During a comprehensive 3-year study period, a total of 32 children were admitted to the Pediatric Intensive Care Unit (PICU) with a diagnosis of Diabetic Ketoacidosis (DKA), resulting in a prevalence rate of 1.12% among 2,850 total admissions. The cohort consisted of 17 boys and 15 girls, with ages ranging from under 5 to 16 years and a mean age of 10.94 years (± 3.94). Most participants resided in rural areas (23 children), while 9 were from urban settings. Socioeconomic backgrounds varied, with the largest proportion belonging to the upper lower class, followed by the lower middle class, lower upper class, and upper middle class. Additionally, six children had a family history of type 2 diabetes mellitus. Notably, 62.5% ($n=20$) of the children were newly diagnosed with Type 1 Diabetes Mellitus (T1DM) at the time of presentation, while the remaining 37.5% ($n=12$) had a prior diagnosis of T1DM [Table 1].

Common symptoms among the cohort included vomiting (81.3%), polyuria (68.8%), polydipsia (65.6%), and abdominal pain (68.8%) [Table 2]. Laboratory assessments indicated a mean heart rate of 113 ± 11.44 bpm, a respiratory rate of 38.31 ± 4.61 cycles per minute, and an oxygen saturation of $97.75 \pm 0.76\%$. Blood tests revealed a mean pH of 7.11 ± 0.15 , a mean bicarbonate (HCO_3^-) level of 10.78 ± 2.25 mEq/L, mean sodium levels of 140.03 ± 5.82 mEq/L, mean potassium levels of 4.185 ± 0.61 mEq/L, and mean chloride levels of 108.62 ± 5.62 mEq/L. Blood glucose levels were significantly elevated at 487.40 ± 80.79 mg/dL, with mean serum ketones measuring 4.07 ± 0.59 mmol/L and HbA1c at $12.21 \pm 2.6\%$ [Table 3].

Urinalysis indicated that 6 children (18.75%) had trace levels of ketones, 18 (56.25%) had moderate levels, and 8 (25%) had severe levels. Similarly, 7 children (21.85%) had trace glucose, 15 (46.85%) had moderate glucose, and 10 (31.2%) had severe glucose [Table 4]. Regarding dehydration, 12 children exhibited severe dehydration, 14 had moderate dehydration, and 6 had mild dehydration.

In terms of acidosis severity, 15 children presented with moderate acidosis (pH 7.00-7.24), 11 with severe acidosis (pH <7.00), and 6 with mild acidosis (pH 7.25-7.30) [Table 5].

The mean duration for the correction of acidosis was 31.74 ± 4.87 hours, while the average time required to transition to subcutaneous insulin was 29.78 ± 5.03 hours. The overall mean hospital stay was 6.18 ± 1.06 days.

As shown in [Table 6], children with new-onset DKA had lower pH, HCO_3^- and higher plasma blood glucose and HbA1c compared to the children who had a known case of type 1 diabetes. These differences among HbA1c were found to be statistically significant ($p < 0.05$).

The average length of stay in the hospital was 6.18 ± 1.06 days while the average duration was 6.8 ± 0.77 days for new-onset cases and 5.17 ± 0.58 days for known T1DM with a statistically significant p-value of 0.00001.

The mean duration of correction of acidosis in hours was 31.74 ± 4.87 . The average time taken for ketoacidosis correction in known T1DM was 27.58 ± 2.94 hours, while 34.2 ± 4.06 hours in new-onset cases with a statistically significant p-value of 0.000015.

The mean time to shift to subcutaneous (s/c) insulin was 29.78 ± 5.027 , whereas the average time taken to move over to subcutaneous insulin was 31.95 ± 4.68 hours in new-onset cases and known cases had 26.17 ± 3.24 hours which was statistically significant with a p-value of 0.0003 [Table 7].

Remarkably, all children achieved full recovery, resulting in a 100% survival rate. Follow-up care includes regular glucose monitoring, ongoing metabolic evaluations, and diabetes education for families to ensure long-term health and prevention.

DISCUSSION

Our observational prospective study involving 32 children with diabetic ketoacidosis (DKA) offers a comprehensive analysis of the prevalence, clinical presentation, management, and outcomes of this critical condition. The cohort comprised 17 boys and 15 girls, with a mean age of 10.94 years (± 3.94), primarily consisting of adolescents aged between 11 and 16 years. Similar age distributions were reported by Shenoy et al. and Muktan et al.^[8,12,13] Notably, 62.5% ($n=20$) of the children were newly diagnosed with type 1 diabetes mellitus (T1DM) and presented with DKA at onset, while the remaining 37.5% ($n=12$) had a prior diagnosis of T1DM. These findings align with those of Rochmah et al. and Muktan et al.^[13,14]

The study highlights the classic symptoms of DKA, with vomiting (81.3%), polyuria (68.8%), polydipsia (65.6%), and abdominal pain (68.8%) being the most prevalent. These symptoms reflect the underlying metabolic disturbances characteristic of DKA, including dehydration, electrolyte imbalances, and hyperglycemia. Vomiting and abdominal pain are

frequently linked to acidosis and dehydration as the body attempts to correct elevated blood glucose levels. The significant prevalence of polyuria and polydipsia indicates severe hyperglycemia, demonstrating the body's effort to excrete excess glucose and maintain fluid balance. Similar findings have been documented by Pasi et al., Muktan et al., and Chauhan et al.^[4,13,15]

Laboratory results revealed severe metabolic derangements, with a mean pH of 7.11 (± 0.15), indicating significant acidosis, and an average bicarbonate (HCO_3^-) level of 10.78 mEq/L (± 2.25), confirming a considerable bicarbonate deficit. Elevated blood glucose levels (487.40 mg/dL ± 80.79) and high serum ketones (4.07 mmol/L ± 0.59) underscore the severity of the DKA episodes, reflecting both hyperglycemia and ketonemia. The HbA1c level of 12.21% (± 2.6) suggests chronic poor glycemic control, a well-established risk factor for DKA, indicating long-term hyperglycemia and inadequate diabetes management that can precipitate DKA. These findings are consistent with those reported by Bhardwaj et al. and Prasad et al.^[16,17]

Urine analysis further corroborated the diagnosis of DKA, with 18.75% of children exhibiting trace ketones, 56.25% showing moderate ketones, and 25% displaying severe ketones. Glucose levels in the urine ranged from trace (21.85%) to severe (31.2%). These results align with blood glucose and ketone levels, reflecting the extent of metabolic disturbance and the body's attempts to manage excess glucose and ketones. A study by Panakkal et al.^[12] reported a higher incidence of 3+ ketones in urine analysis.

The study found that 12 children experienced severe dehydration, 14 had moderate dehydration, and 6 exhibited mild dehydration. Dehydration is a critical concern in DKA management, as it can exacerbate metabolic disturbances and prolong recovery. The severity of acidosis was classified into three groups: 15 children had moderate acidosis (pH 7.00-7.24), 11 had severe acidosis (pH <7.00), and 6 had mild acidosis (pH 7.25-7.30). This classification illustrates the variability in acidosis severity and emphasizes the need for individualized treatment approaches to correct metabolic imbalances. Similar observations have been made by Kanwal et al. and Rochmah et al.^[6,14]

The mean duration for acidosis correction was 31.74 hours (± 4.87), consistent with standard treatment protocols for DKA, which require careful monitoring and adjustment of fluids and electrolytes. The mean time to transition to subcutaneous insulin was 29.78 hours (± 5.03), indicating the necessary stabilization period before shifting to a more manageable form of insulin therapy. The average hospital stay was 6.18 days (± 1.06), reflecting the time required for comprehensive treatment and stabilization of the child's condition.

Our data reveal significant differences between newly diagnosed cases of DKA and those with a known history of T1DM in terms of clinical outcomes. These findings are corroborated by

existing literature, where newly diagnosed cases often present with more severe metabolic disturbances due to delayed recognition and management of hyperglycemia, resulting in longer recovery times. The duration of hospital stay was notably longer in new cases (6.8 \pm 0.77 days) compared to patients with known T1DM (5.17 \pm 0.58 days), with a highly significant p-value of 0.00001. This extended stay likely reflects the increased complexity in stabilizing newly diagnosed DKA patients, who may not yet be accustomed to managing their condition, unlike established patients who may receive earlier intervention.

Additionally, the time required for acidosis correction was longer in new cases (34.2 \pm 4.06 hours) compared to known cases (27.58 \pm 2.94 hours), with a p-value of 0.000015. The delay in correcting acidosis in newly diagnosed patients may be attributed to a more profound ketoacidosis state, often exacerbated by prolonged periods of hyperglycemia before clinical intervention. In contrast, patients with known T1DM are more likely to recognize the symptoms of impending DKA earlier and seek treatment sooner, facilitating a quicker correction of acidosis.

Moreover, the transition to subcutaneous insulin took significantly longer in new cases (31.95 \pm 4.68 hours) compared to known cases (26.17 \pm 3.24 hours), with a p-value of 0.0003. This finding underscores the challenges associated with achieving metabolic stability in newly diagnosed patients, where insulin resistance and severe hyperglycemia may necessitate prolonged insulin infusion before transitioning to subcutaneous administration. These findings differ from those reported by Panakkal et al.^[12]

Remarkably, all children in this study achieved full recovery, demonstrating a 100% survival rate. This outcome underscores the effectiveness of the treatment protocols employed and the importance of timely and appropriate management of DKA. The high survival rate attests to the quality of care provided and reinforces the efficacy of current therapeutic strategies in managing severe DKA in children. These findings were inconcordance with Panakkal et al.^[12]

Limitations

While the study provides valuable insights, it is important to acknowledge its limitations. The sample size of 32 children may limit the generalizability of the findings, and a larger cohort could provide more robust data. Variability in clinical protocols, unaddressed confounding factors, and reliance on caregiver-reported symptoms further complicate the findings. Future studies should focus on larger, multicenter trials to validate these findings and explore additional factors influencing DKA management and outcomes.

CONCLUSION

This observational prospective study involving 32 children with diabetic ketoacidosis (DKA)

demonstrates that timely and effective management significantly contributes to positive outcomes, as evidenced by a 100% recovery rate. The findings highlight the critical need for early recognition and intervention, particularly in newly diagnosed cases of type 1 diabetes mellitus, which present with more severe metabolic disturbances and longer recovery times. Our results emphasize the importance of individualized treatment approaches and underscore the efficacy of current therapeutic strategies in managing DKA, ultimately ensuring the well-being of affected children.

REFERENCES

1. Glaser N, Fritsch M, Priyambada L, et al. ISPAD clinical practice consensus guidelines 2022: diabetic ketoacidosis and hyperglycemic hyperosmolar state. *Pediatr Diabetes*. 2022;23(7):835-856. doi:10.1111/pedi.13406.
2. Sood, Kanuja & Soni, Ankita & Kumar, Ashok & Yadav, Bibek. Diabetic Ketoacidosis -Review Article. *Journal of Cardiovascular Disease Research* ISSN: 0975-3583,0976-2833 VOL14, ISSUE4, 2023.
3. Praveen PA, Hockett CW, Ong TC, Amutha A, Isom SP, et al. Diabetic ketoacidosis at diagnosis among youth with type 1 and type 2 diabetes: Results from SEARCH (United States) and YDR (India) registries. *Pediatr Diabetes*. 2021 Feb;22(1):40-46. doi: 10.1111/pedi.12979. Epub 2020 Feb 17. PMID: 31943641; PMCID: PMC7748377
4. Pasi R, Ravi KS. Type 1 diabetes mellitus in pediatric age group: A rising endemic. *J Family Med Prim Care* 2022;11:27-31
5. Ayyavoo A, Ravikulan A, Palany R. Treatment of diabetic ketoacidosis with subcutaneous regular insulin in a non-ICU setting is effective and economical: A single-centre experience. *J Pediatr Endocrinol Diabetes* 2022;2:50-5
6. Kanwal SK, Bando A, Kumar V. Clinical profile of diabetic ketoacidosis in Indian children. *Indian J Pediatr*. 2012 Jul;79(7):901-4. doi: 10.1007/s12098-011-0634-3. Epub 2011 Dec 30. PMID: 22207489.
7. Basavanthappa SP, Pejaver R, Raghavendra K, Srinivasa V, Suresh Babu MT. Clinical profile and outcome of diabetic ketoacidosis in a tertiary care hospital in South India. *Int J Contemp Pediatr* 2015;2:29-31
8. Shenoy S, Upadhy N. The factors affecting the resolution of acidosis in children with diabetic ketoacidosis - A retrospective study from a tertiary care centre in India. *Indian J Child Health*. 2017;4(3):294-297
9. EL-Mohandes N, Yee G, Bhutta BS, et al. Pediatric Diabetic Ketoacidosis. [Updated 2023 Aug 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK470282/>
10. Raghupathy P. Diabetic ketoacidosis in children and adolescents. *Indian J Endocrinol Metab*. 2015 Apr;19(Suppl 1):S55-7. doi: 10.4103/2230-8210.155403. PMID: 25941653; PMCID: PMC4413392
11. Elendu C, David JA, Udoyen AO, Egbunu EO, Ogbuiyi-Chima IC, et al. A comprehensive review of diabetic ketoacidosis: an update. *Ann Med Surg (Lond)*. 2023 May 23;85(6):2802-2807. doi: 10.1097/MS9.0000000000000894. PMID: 37363479; PMCID: PMC10289692.
12. Panakkal SJ, Shirodkar D, Saldanha PRM. Clinical profile and outcome of children presenting with diabetic ketoacidosis at a tertiary care hospital in Dakshina Kannada. *Int J Contemp Pediatr* 2023;10:644-8.
13. Muktan D, Ghising LT, Singh RR. Clinical Profile of Diabetic Ketoacidosis among Children in Eastern Nepal. 2019; 15(4):226-9
14. Rochmah N, Faizi M, Harjantien N. Diabetic ketoacidosis in children: an 11-year retrospective in Surabaya, Indonesia. *Paediatrica Indonesiana*. 2015;55:40. doi:10.14238/PI55.1.2015.40-3.
15. Chauhan AV, Pathak G, Khare P. Study of clinical profile and treatment of type 1 diabetes mellitus in pediatric patients at a tertiary care centre. *Int J Contemp Pediatr* 2023;10:18-22.
16. Bhardwaj P, Yadav V, Sharma M. Clinical profile and outcome of the children with diabetic ketoacidosis (DKA) in hilly Himalayan state of north India. *Int J Res Med Sci* 2017;5:5402-5
17. Prasad D, et al., A retrospective case study of clinical profile of hospitalized children with type 1 diabetes mellitus at a tertiary health care center in northern India, *Clinical Epidemiology and Global Health* (2013), <http://dx.doi.org/10.1016/j.cegh.2013.02.002>.