



PROFILE OF CHILDHOOD DIABETIC KETOACIDOSIS - A SINGLE CENTRE EXPERIENCE

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ABSTRACT

Context: Diabetic ketoacidosis (DKA) is a common paediatric emergency. **Aims:** To present the patient profile of childhood DKA **Methods:** This is a retrospective study of 26 children presenting with DKA to pediatric emergency. All the patients had random plasma glucose, serum electrolytes, renal function tests, arterial blood gas analysis, hemogram, complete urine examination, chest radiograph done. **Results:** Of the 26 patients studied 20 were girls. 42% of the children were new onset type 1 diabetes presenting with DKA. 70% were comatose at the time of presentation. Mean age of patients was 8.9 ± 4.52 years. Severe, moderate and mild DKA was present in 13, 8 and 5 patients respectively. The precipitating factor for DKA are skipping the dose in 9, acute febrile illness due to respiratory tract infection in 9, both skipping the dose and respiratory tract infection in 2, diarrhea in 1 and 5 didn't have a precipitating factor. 10 out of 26 patients had hyperosmolar state at the time of presentation. The mean plasma glucose, PH, bicarbonate, anion gap and osmolality were 372 ± 108 mg/dl, 7.11 ± 0.27 , 13.8 ± 5.9 meq/l, and 307 ± 15.8 mosm/l respectively. Mean sodium and potassium were 140 ± 10.8 meq/l and 3.8 ± 0.88 meq/l respectively. Hypokalemia is the most common complication seen in 21 out of 26 patients. The mean hospital stay was 9.8 ± 5.65 days. One patient died of pneumonia and sepsis with shock. **Conclusions:** Type 1 diabetes can present with DKA as first manifestation, 1/3 of patients had hyperosmolar state and hypokalemia is the most common complication.

KEYWORDS: Diabetic ketoacidosis, type 1 diabetes mellitus, hypokalemia, cerebral edema.

INTRODUCTION

Diabetic ketoacidosis (DKA) is a potentially life-threatening acute complication of type 1 diabetes mellitus (T1DM), characterized by a biochemical triad of hyperglycemia, ketonemia (ketonuria) and acidosis.^[1] DKA results from absolute or relative deficiency of circulating insulin and the combined effects of increased levels of the counter regulatory hormones: catecholamines, glucagon, cortisol and growth hormone.^[2,3]

DKA is the leading cause of mortality in childhood diabetes.^[4] Absolute insulin deficiency occurs in previously undiagnosed T1DM and when patients on treatment deliberately or inadvertently do not take insulin, especially the long-acting component of a basal-bolus regimen. DKA occurs in 20%-40% of children with known DM who omit insulin doses or who are not managed appropriately during an intercurrent illness.^[5] DKA is the most frequent cause of diabetes-related death

in children with mortality rate ranging between 6% and 24% in developing countries.^[6,7] Recent studies have documented a 21% to 24% mortality rate in patients who develop cerebral edema with DKA, hence accounting for most of the DKA deaths and a high rate of permanent neurologic morbidity.^[5,8] Other causes of morbidity and mortality in DKA include hypoglycemia, infections, pulmonary edema, central nervous system hemorrhage or thrombosis, other large vessel thrombosis, cardiac arrhythmias caused by electrolyte disturbances, pancreatitis, renal failure and intestinal necrosis.^[8]

The severity of DKA is defined by the degree of acidosis: mild is a venous pH between 7.2 and 7.3; moderate is a pH between 7.1 and 7.2; and severe is a pH less than 7.1.^[9] Management requires careful replacement of fluid and electrolyte deficits, intravenous administration of insulin and close monitoring of clinical and biochemical parameters directed towards timely detection of complications, including hypokalemia,

hypoglycemia and cerebral edema. Very few studies have been conducted assessing the clinical and biochemical profile of childhood DKA in India. Data was collected retrospectively over 1 year at a tertiary centre in South India.

METHODS

This is a retrospective study of 26 children presenting with DKA in the emergency department of Narayana medical college and hospital. DKA was diagnosed by significant hyperglycemia ($>250\text{mg/dl}$), ketonuria or ketonaemia, serum Bicarbonate $<18\text{mmol/l}$ and DKA-associated chemical signs (e.g. dehydration, Kussmaul respiration, etc.).

Data retrieved from the case notes included age, sex, presenting symptoms, duration of symptoms prior to presentation, presence of complications, and outcome of hospitalization. All the patients had random plasma glucose every hourly, serum electrolytes & arterial blood gas analysis every 4th hourly, renal function tests every 12th hourly & hemogram, complete urine examination, chest radiograph.

Clinical features (heart rate, respiratory rate, blood pressure, urine output, oxygen saturation, sensorium, headache, pupil size and reaction to light assessment) were monitored hourly.

RESULTS

Of the 26 patients included out of which 20 were girls. 11 patients (42%) were having new onset T1DM presenting with DKA. Out of 26 patients, 18 (70%) were comatose at the time of presentation. Mean age of patients was 8.9 ± 4.52 years. The mean plasma glucose, pH, bicarbonate, anion gap and osmolality is given in table 1 along with general characteristics of the patients. Skipping of insulin dose was the major precipitating factor for DKA followed by acute febrile illness due to respiratory tract infection (Table 2). Severity of DKA based on pH values are given in table 3. 10 out of 26 patients had hyperosmolar state at the time of presentation with two patients having severe hyponatremia (S. sodium $>150\text{ meq/dl}$). Mean sodium and potassium were $140 \pm 10.8\text{ meq/l}$ and $3.8 \pm 0.88\text{ meq/l}$ respectively and hypokalemia was the most common complication seen. The mean hospital stay was 9.8 ± 5.65 days. One patient died of pneumonia and sepsis with shock.

Table 1: Showing general characteristics of patients

	n = 26
Mean age (years)	8.9 ± 4.52
Sex ratio (male: female)	1:4
Mean plasma glucose (mg/dl)	372 ± 108
Mean pH (meq/l)	7.11 ± 0.27
Mean osmolality (mosm/l)	307 ± 15.8
Mean anion gap (meq/l)	13.8 ± 5.9
Mean hospital stay (days)	9.8 ± 5.65

Table 2: Showing precipitating factors implicated in DKA

Precipitating factors	N(%)
Skipping of dose	9 (34.1%)
Acute febrile illness	9 (34.1%)
Both	2 (10.5%)
Diarrhea	1 (5.2%)
No factor	5 (19.2%)

Table 3: Showing prevalence of DKA in terms of severity

Severity	N(%)
Severe	13 (50%)
Moderate	8 (30.8%)
Mild	5 (19.2%)

DISCUSSION

DKA is the major cause for mortality in children with Diabetes mellitus (DM). There is a shift towards younger age group and it is more of concern especially in developing countries where mortality in DKA is alarmingly high. The median age at presentation in our series was 8.9 years whereas Basavanthappa SP et al.¹⁰ had median age of 7.52 years while two earlier studies have reported it to be 6.9 and 7.9 years.^[11, 12]

The frequency of DKA is higher among girls than among boys in our study as reported by Neu et al.^[13] who reported the frequency to be higher among girls, while Basavanthappa SP et al.^[11] and other studies have shown DKA more common in boys than girls. Most of our patients had new-onset DM. The major precipitating factor for DKA were infections (most commonly viral fever, pneumonia and urinary tract infections) and omitting insulin dosage, inadequate insulin administration during an intercurrent illness. This shows lack of patient and care givers awareness and non compliance for the continues insulin treatment. Similar observations were found in earlier studies.^[10-13]

Like in other studies^[11] hypokalaemia was the most common complication observed in our series. There was no mortality with cerebral edema in our study while it accounts for 57%-87% of all deaths due to DKA and typically occurs 4-12 hours after the onset of treatment, though it can be present before treatment has commenced or at any time during treatment.^[13]

Severe DKA occurred in majority of patients in our study (50%) which is in correspondence to other study^[10] (53.8%). Similarly most patients present with severe clinical manifestations like 70% children presented in comatose position in our study.

Two patients in our study had severe hyponatremia. Both were initially treated in a private settings by a general practitioner and overzealous use of saline was documented which again shows the need of a pediatric endocrinologist expert in management of DKA in treating these patients.

The administration of appropriate intravenous fluids, insulin infusion is the gold standard for treatment. Continuous insulin infusion rather than subcutaneous insulin is recommended as severe acidosis leads to cutaneous vasoconstriction thereby reducing the absorption of insulin.^[14]

CONCLUSIONS

Diabetic ketoacidosis is a life threatening complication of diabetes mellitus in children. T1DM can present with DKA as first manifestation, precipitating by infections or missing of dose, which can be severe in substantial patients. Proper fluid management can avoid development of cerebral edema which is the leading cause of mortality.

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