Short Report

Clinical profile and outcome of diabetic ketoacidosis in children

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ABSTRACT

Background. There is little information on the clinical profile and outcome of children with diabetic ketoacidosis in India. We analysed the data of children managed by us at a tertiary care hospital.

Methods. We retrospectively analysed the case records of 21 children (13 boys and 8 girls) with diabetic ketoacidosis admitted to our hospital from January 2004 to August 2008. They were managed using a standard protocol including intravenous fluids and insulin infusion. Blood glucose, serum electrolytes, blood urea, arterial blood gases and urinary ketones were monitored at regular intervals. The outcomes were assessed.

Results. The median age at presentation was 8 years and 17 children (80%) were detected to have diabetes mellitus at the time of presentation. Twelve children (57%) presented with severe diabetic ketoacidosis. Polyuria with polydipsia was the commonest clinical presentation (17). All of them had elevated HbA_1C levels. The average length of stay in the paediatric intensive care unit was 2.9 days. The median time for the arterial blood gases to become normal was 19 hours and for urinary ketones to become non-detectable was 28 hours. None of the children received bicarbonate and there were no complications or mortality. All the children were doing well on follow up at 3 months.

Conclusion. The outcome of active management of diabetic ketoacidosis in children is rewarding. The use of a standard protocol for management was associated with no complications or mortality in our series.

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INTRODUCTION

Diabetic ketoacidosis (DKA) is a common and serious complication of diabetes mellitus (DM), accounting for 8%–28%[1] of all hospital admissions for DM with mortality rates of 0.15%–0.3%.[2,3] Though guidelines are available for managing DKA in children, routine practices in India vary widely. A high index of clinical suspicion with timely administration of appropriate intravenous fluids, rational use of sodium bicarbonate, continuous rather than bolus insulin infusion and monitoring in an intensive care unit (ICU) are associated with better outcomes.[4] There is a paucity of data from India on the clinical profile and outcome of DKA in children. We retrospectively analysed our data of 4 years at a tertiary care children's hospital.

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METHODS

The Kanchi Kamakoti CHILDS Trust Hospital, Chennai, Tamil Nadu is a 220-bed private multispecialty teaching and referral hospital for children. The paediatric ICU is a 10-bed unit equipped for mechanical and non-invasive ventilation and has a high frequency oscillator. The case records of children hospitalized with DKA from January 2004 to August 2008 were reviewed. All patients were monitored every hour for clinical features (heart rate, respiratory rate, blood pressure, urine output, oxygen saturation, sensorium, headache, vomiting) and every 4 hours for blood sugar, arterial/venous blood gas analysis, serum electrolytes, urine for ketones, haematocrit and blood urea till 24 hours after admission.

Diabetic ketoacidosis was diagnosed when the blood sugar at admission was >200 mg/dl with acidosis (pH<7.3 or plasma bicarbonate <15 mmol/L) and positive urine ketones. Patients were considered to have newly diagnosed diabetes if they were previously undiagnosed and had presented with DKA. The severity of DKA was graded as mild, moderate and severe (Table I). The treatment protocol followed has been reported previously.[5] Briefly, the children were started on intravenous fluid therapy irrespective of the severity of DKA. Children with moderate and severe DKA were started on insulin infusion after starting rehydration and potassium replacement. The infusion was stopped when the children were alert, able to tolerate oral feeds, metabolically stable (blood pH >7.3, plasma bicarbonate >15 mmol/L and blood sugar around 250 mg/dl), and were shifted out of the ICU. Regular insulin (Human actrapid) at 0.2–0.4 U/kg was administered before stopping the insulin infusion. Children with mild DKA were started on subcutaneous regular insulin. Regular insulin was replaced with mixed insulin (30/70) once the blood sugar had stabilized. Potassium replacement/restriction was given as required. Bicarbonate was given if the patients had a pH <6.9, potentially life-threatening hyperkalaemia and cardiopulmonary compromise. All children were given prophylactic ceftriaxone (75 mg/kg/day) till the blood/urine cultures were reported to be sterile.

All children with moderate and severe DKA were managed in the paediatric ICU and children with mild DKA were managed in the high dependency unit that has similar monitoring facilities as the paediatric ICU (as children are more prone to hypoglycaemia).

RESULTS

Of the 36 689 hospital admissions from January 2004 to August 2008, a total of 21 children presented with DKA (a prevalence of 1 in 1747 hospital admissions). The median age at presentation was 8.2 years (range: 2–14 years) with a male:female ratio of 1.6:1; the mean duration of symptoms before hospitalization was 11.6 days (range: 1–30 days). Family history of type 2 DM was present in 13 (61%; paternal 9, maternal 4) and 17 (80.9%) were newly diagnosed DM. Fever was the precipitating factor

TABLE I. Clinical profile of children with diabetic ketoacidosis (DKA)

Age group (years)	Girls	Boys	Diabetes		Severity of DKA		
			Newly diagnosed	Known	Mild	Moderate	Severe
<5	4	3	7	-	-	1	6
5–10	2	5	5	2	3	1	3
>10	2	5	5	2	1	3	3

in 6 children (28.5%) and in 1 child with type 1 diabetes, the omission of insulin led to DKA. The most common presenting complaints were polyuria and polydipsia in 17, loss of weight in 9, polyphagia and fever in 7 each, and vomiting and abdominal pain in 5. A majority (12) presented with severe, 5 with moderate and 4 with mild DKA. Three children presented with shock requiring a fluid bolus (10 ml/kg of normal saline). The clinical profile of the children is shown in Table I. Chest X-ray was normal in all children, and their blood and urine cultures were sterile. Ultrasound abdomen (to rule out pancreatitis, fibrocalculous pancreatopathy) was normal in 18 and the rest (3) had mild hepatomegaly. The median time for the arterial blood gas to normalize and for urinary ketones to disappear were 19 hours and 28 hours, respectively, and the median duration for changing over to subcutaneous insulin was 1.4 days. Hypokalaemia was the common therapy-related complication observed in 7 children. None of the children had hypoglycaemia or cerebral oedema and there was no mortality. The average length of stay in the ICU was 2.9 days and for discharge from hospital (discharged when alert, able to take oral feeds well, the technique of insulin therapy taught/reinforced and warning signs explained) was 8.5 days. On follow up at a median of 3 months, all children were doing well.

DISCUSSION

Diabetic ketoacidosis is a life-threatening condition caused by a decrease in effective circulating insulin along with an increase in counter-regulatory hormones (glucagon, catecholamines, cortisol and growth hormone) leading to hyperglycaemia, hyperosmolarity, increased lipolysis, ketonaemia and metabolic acidosis.[4] The median age at presentation in our series was 8.2 years whereas two earlier studies have reported it to be 6.9 and 7.9 years.[6,7] The frequency of DKA is higher among boys than among girls as reported in earlier studies, except in the study by Neu *et al*.[8] who reported the frequency to be higher among girls. Most of our patients had new-onset DM. This could be because of lack of awareness among the parents of these symptoms being due to diabetes. Polyuria and polydipsia due to hyperglycaemia were the most common clinical symptoms in contrast to impaired level of consciousness as reported by Jayashree *et al*.[6] The major precipitating factors for DKA are infections (most commonly viral fever, pneumonia and urinary tract infections), omitting insulin, inadequate insulin administration during an intercurrent illness and intake of drugs such as high dose glucocorticoids, atypical antipsychotics and diazoxide. The administration of appropriate intravenous fluids, rational use of sodium bicarbonate, continuous rather than bolus insulin infusion is associated with better outcomes. Volume contraction is the hallmark of DKA.[9] Therefore, the objectives of fluid and electrolyte management in DKA are to restore the circulating volume, glomerular filtration rate to enhance the clearance of glucose and ketones from the blood, replace sodium and the extracellular and intracellular deficit of water, and prevent cerebral oedema. 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.[10] None of our patients received bicarbonate therapy. Hypokalaemia was the most common complication observed in our series as in other studies.[6] Cerebral oedema 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,[8] and is often precipitated by overzealous administration of fluids. In our study, none of the children developed cerebral oedema and there were no deaths.

Conclusion

The majority of children with DKA had new-onset DM and hypokalemia was a common occurrence. DKA, though a life-threatening event, can have a good outcome with no complications and mortality when diagnosed and managed using a standard protocol.

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