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Original Research Article

Clinical profile and outcome of the children with diabetic ketoacidosis (DKA) in hilly Himalayan state of north India

Parveen Bhardwaj[1], Vijay Yadav[1], Minoo Sharma[2]*

[1]Department of Pediatrics, [2]Department of Physiology, Indira Gandhi medical College, Shimla, Himachal Pradesh, India

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***Correspondence:**
Dr. Minoo Sharma,
E-mail: minoo01sharma@gmail.com

ABSTRACT

Background: Context: DKA is an important complication of undiagnosed or poorly controlled diabetes mellitus. Proper management of DKA can prevent morbidity and mortality attributable to diabetes mellitus. The aim of the research was to study the clinical profile and outcome of the children admitted with Diabetic Ketoacidosis (DKA).
Methods: A descriptive retrospective study was conducted in pediatric ICU of tertiary level care hospital over three years between January 2013 and December 2015. 29 patients were diagnosed with DKA during the three-year period, the data was collected by reviewing the medical record of the patient and information with respect to personal details, clinical features, laboratory parameters, management and outcome was recorded.
Results: 29 patients were diagnosed with DKA, of these 17 were males and 12 females. M:F was 1.4:1 and mean age at presentation was 11.4±4.4 yrs. DKA was the presenting manifestation of Diabetes in 48.2% patients and 51.8% were already known cases of Diabetes. Abdominal pain (62%), polyuria (58.6%), fast breathing (58.6%), vomiting (55.1%), and altered sensorium (44.8%) were common presenting symptoms of DKA. Severe ketoacidosis was noted in 48.2% and severe dehydration in 31%. Shock was observed in 27.5% patients and 20.9% had cerebral edema. Metabolic abnormalities like hponatremia, hypernatremia, hyperkalemia, hypokalemia were seen in 44.8%, 13.7%, 24.1%, 17.2% respectively. We had 1 (3.4%) mortality.
Conclusions: Diabetic Ketoacidosis (DKA) is an important cause of hospital admissions and 48.2% of newly diagnosed cases presented with DKA. Infections and omission of the insulin were the most common precipitating factors. For the long- term management strategy it is important to educate of the patients and their parents regarding regular blood sugar monitoring and insulin dosing.

Keywords: Cerebral edema, DKA, T1DM

INTRODUCTION

DKA is the end result of the metabolic abnormalities resulting from a severe deficiency of insulin or insulin effectiveness. The latter occurs during stress as counter-regulatory hormones block insulin action.[1] Type 1DM mostly presents as diabetic ketoacidosis (DKA) which is a common pediatric endocrine emergency and is a state of absolute or relative insulin deficiency. About 25-40% of the newly diagnosed T1DM children present with DKA whereas the risk of developing DKA in established T1DM children is 1-8% per patient per year.[2] DKA is a major complication of the childhood type 1DM and is associated with increased risk of morbidity and mortality.[3,4] Frequent causes include stress and infection in a new diabetic or a missed insulin dose in an established diabetic.[5] The mortality rate of DKA in children in the developed countries has declined to

0.15%-0.31%.[6,7] Cerebral edema is the most common cause of death in DKA and is associated with a high rate of permanent neurologic morbidity.[8-11] Other causes of morbidity and mortality in DKA include hypoglycemia, infections, pulmonary edema, central nervous system hemorrhage or thrombosis, other large vessel thromboses, cardiac arrhythmias caused by electrolyte disturbances, pancreatitis, renal failure and intestinal necrosis.[8-11] However, in places with less developed medical facilities, the risk of death from DKA is greater, and children may die before receiving appropriate treatment. This report describes our three-year experience of DKA management and outcome in a pediatric population at a tertiary care hospital in hilly Himalayan state of north India.

METHODS

This was a retrospective study done in pediatric intensive care unit (PICU) in a tertiary care level hospital from January 2011 to December 2013 in children admitted with DKA. The data was collected by reviewing the medical records of the patients. We recorded the age, sex presenting symptoms and signs at admission, precipitating factors like intercurrent illnesses and investigations like complete blood counts, serum electrolytes, serum urea, serum creatinine, arterial blood gases, blood and urine culture and urinary ketone bodies. DKA was defined as presence of hyperglycemia (blood glucose >200mg/dL) with a venous pH <7.3 and/or bicarbonate <15mmol/L with associated glycosuria, ketonuria, and ketonemia.[4] DKA was categorized as mild (venous pH <7.30; bicarbonate <15mmol/L), moderate (pH <7.2; bicarbonate <10mmol/L), and severe (pH <7.1; bicarbonate <5mmol/L).[8]

All the patients were managed with Milwaukee protocol. Monitoring of heart rate, respiratory rate, Blood pressure, oxygen saturation (spo2), Glasgow coma scale (GCS), capillary blood sugar, fluid intake and output were done every hourly.[1] Serum Electrolytes, urea, creatinine, hematocrit, blood sugar, urinary ketones and arterial blood gases (ABG) were done every 4 hourly. Hypernatremia and hyponatremia was defined as value >145mmol/L and <135mmol/L respectively.[12] Similarly hyperkalemia and hypokalemia were defined as value >5.5mmol/L and <3.5mmol/L respectively.[12]

The resolution of DKA was based on resolution of acidosis on ABG (ph>7.3), normalization of blood sugar level and stabilization of clinical conditions. All patients during treatment were monitored for cerebral edema. The diagnosis of cerebral edema was suspected in the following two scenarios a) declining neurological status after initial improvement; b) persistently poor neurological status without any obvious cause. The early signs that were suspicious for this condition included headache, vomiting, lethargy and decreased arousal, relative bradycardia and hypertension while the late signs included seizures, incontinence, pupillary changes, papilledema, upgoing plantars and respiratory arrest.[13]

The diagnosis of cerebral edema was verified on the basis of computed tomography (CT) scan in all the cases. Cerebral oedema was managed with elevation of head end of the bed to 30-degree, intravenous mannitol, hypertonic saline and restriction of fluids. Acute kidney injury was diagnosed on the basis of changes in serum creatinine and urine output and was classified according to Pediatric PRIFLE criteria.[14] Dehydration was assessed as per WHO criteria. The data was analysed by epi info.

RESULTS

A total of 29 children were admitted over a period of 3 years with DKA. Of these 17 were males and 12 were females. Male to female ratio was 1.4:1. 13.7% were between 0-5 years, 27.6% in 5-10 years, 37.9% between 11-15years and 20.7% were between 15-18%. Mean age and weight at admission were 11.4±4.4 years and 30.1±16kg. 48.2% were newly diagnosed and 51.8% were previously diagnosed cases of diabetes. Of the known diabetics 85.2% presented with second DKA episode and 14.8% had more than 2 episodes of DKA. Abdominal pain (62%) was the most common symptom noted followed by polyuria (58.6%), fast breathing (58.6%), vomiting (55.1%), fever (48.2%), altered sensorium (44.8%), polydipsia (41.3%) and diarrhea (24.1%) as shown in Table 1.

Table 1: Showing clinical features of children with DKA.

Symptoms	%(n=29)	Signs	%(n=29)
Fever	14 (48.2)	1. Dehydration	29 (100)
Polyuria	17 (58.6)	A) Mild	7 (24.1)
Polydipsia	12 (41.3)	B) Moderate	13 (44.8)
Vomiting	16 (55.1)	C) Severe	9 (31.0)
Abdominal pain	18 (62.0)	2. Shock	8 (27.5)
Fast breathing	17 (58.6)	A) Septic	5 (17.2)
Diarrhea	07 (24.1)	B) Hypovolemic	3 (10.3)
Altered sensorium	13 (44.8)	3. Pneumonia	6 (20.9)
ketoacidosis	29 (100)	4. Cerebral edema	6 (20.9)
Severe	14 (48.2)	5. Acute kidney injury	5 (17.2)
Moderate	9 (31.0)	6. UTI	5 (17.2)
Mild	6 (20.9)	7. Need for mechanical ventilation	3 (10.3)
		8. Sodium bicarbonate administration	6 (20.6)

Dehydration was seen in all patients, of these 31% had severe dehydration, 44.8% had moderate dehydration and 24.1% had mild dehydration. In ketoacidosis severe ketoacidosis was noted in 48.2%, moderate in 31.0% and mild 20.9% of the patients. Shock was present in 27.5% patients at admission of which 17.2% had septic shock and 10.3% had hypovolemic shock. Features of

pneumonia (clinically and radiologically) were seen in 20.9%. Cerebral edema was found in 20.9% of the children and acute kidney injury was noted in 17.2% due to dehydration and septicemia. 17.2% had culture proven urinary tract infection of these 2 had *E. coli* and Proteus species each and 1 had klebsiella and their further work up for urinary tract infection was normal. Sodium bicarbonate was administered in 20.6% of the patients who had severe ketoacidosis as their blood Ph remained <6.9 despite adequate fluid resuscitation in the first hour. 3 patients required mechanical ventilator support, of these two were successfully extubated, but one patient died of cerebral edema, septicemia, acute respiratory distress syndrome and septic shock.

The laboratory parameters at presentation are shown in Table 2. Leucocytosis was seen in 55.1% of the patients and septicemia was found in 7 patients of these 4 had culture proven septicemia. Blood cultures was positive in 4 patients out of which two had grown staphylococcus aureus and one had *E. coli* and Klebsiella each. The average duration of resolution of ketoacidosis was 16.8±8.3hours.

Table 2: Showing lab parameters.

Laboratory parameter	Mean ±SD (n=29)
Blood sugar(mg/dl)	475.8±83.0
Ph	7.1±0.1
Pco2 (mmhg)	19.8±5.8
Bicarbonate level (meq/dl)	8.0±3.4
Base excess	-20.9±5.7
Lactate (mmol/l)	2.1±0.7
Sodium (meq/l)	139.5±9.1
Potassium (meq/l)	3.9±1.2
Total leucocyte count	11.6±5.4
Hba1c (%)	11.6±2.4
Condition normalized in hrs	16.8±8.3

DISCUSSION

In this retrospective case series, we evaluated the clinical profile and outcome of 29 children of DKA admitted in a tertiary care teaching hospital in India. DKA accounts for nearly 0.6% of total intensive care admission in PICU.[15] DKA, represents a decompensated phase of diabetes mellitus, which may require PICU admission, especially in the presence of cardiovascular instability, inability to protect the airway, altered state of consciousness, the presence of acute abdominal signs or symptoms suggestive of acute gastric dilatation.[16] DKA may be difficult to diagnose because of its non-specific symptoms and signs like pain abdomen, vomiting and fast breathing and may be misdiagnosed as pneumonia, gastroenteritis or acute abdomen. It is even more challenging to diagnose DKA in newly diagnosed DM due to lack of awareness regarding signs and symptoms of T1DM among general population and primary care physician and this contributes to T1DM complicating as DKA. In the present study, abdominal pain 62%, fast breathing 58%, polyuria 58% vomiting 55%, fever 45% and altered sensorium 44.8% were the predominant symptoms at admission these findings were similar to the study done by Kanwal et al where they noticed polyuria in 54.3%, vomiting 52.7%, abdominal pain 47.3%, fever in 40% and altered sensorium in 50% of the patients.[17]

In this case series we have seen shock was present in 27.5% of the patients, of these septic shock was present in 17.2% and hypovolemic shock in 10.3% of all the cases. All these were managed according to PALS guidelines. Cerebral oedema is the most threatening complication of DKA in children, occurring in 0.3-1% of DKA episodes.[8,10] It has a high mortality rate of 21-24% in different studies, whereas 21-26% of the survivors suffer from neurologic sequelae.[8-11]

Hence, it is very important to prevent and recognize the early warning signs of cerebral oedema. In the present study the cerebral edema was noted in 20.9% of the patients which was similar to Kanwal et al they reported cerebral edema in 14.5%.[17] Acute kidney injury was reported in 5 (17.2%) patients based on pediatric RIFLE criteria and these were managed conservatively and none of them required dialysis. Kanwal et al reported Acute renal failure in 7.2%, the reason for high incidence of acute kidney injury in the present study may be that dehydration was seen in all our patients, of these severe dehydrations was seen in 31%, moderate in 44.8% and mild in 24.1% whereas they reported dehydration in 92.6%, mild in 7.3% moderate in 69.9% and severe in 23.56% of all the patients. 3 patients required mechanical ventilatory support as they had poor glascow coma scale, ARDS and refractory shock. It has been seen that there is an increased risk of the development of cerebral oedema with mechanical ventilation in patients with severe diabetic ketoacidosis.[18]

All three of our mechanically ventilated patients developed cerebral edema and we had one mortality. In electrolytes disturbances Hyponatremia and Hypernatremia can both be seen in DKA. We noted hyponatremia in 44.8% and hypernatremia 13.7%. Hyperkalemia was noted in 24.1% of the patients, similar to Kanwal et al.[17]

We managed all our patients with Milwaukee protocol and during treatment 17.2% had hypokalemia. The mortality rate among DKA in Developed Countries is from 0.15-0.31% and in developing countries it is around 13% where infections remain as one of the most important precipitating factor of DKA.[8,19] In the present study pneumonia, urinary tract infections, diarrhea and omission of insulin were the common precipitating factors for DKA. One death (3.4%) occurred in our admitted children and died of cerebral edema with septic shock. Kannwal et al reported 7 (12.7%) deaths and they noticed cerebral edema with or without renal failure and sepsis were the common cause of deaths in Indian

children.[17] Madiha Syed et al in their study reported 3.4% deaths in patients with DKA.[20]

CONCLUSION

Diabetic Ketoacidosis(DKA) is an important cause of hospital admissions and 48.2% of newly diagnosed cases presented with DKA. Infections and omission of the insulin were the most common precipitating factors. For the long- term management strategy it is important to educate of the patients and their parents regarding regular blood sugar monitoring and insulin dosing.

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