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CASE REPORT

A Case Report on Diabetic Ketoacidosis in Children

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Abstract

Introduction: Diabetic Ketoacidosis (DKA) is a complex metabolic state characterized by hyperglycaemia, ketonemia and acidosis. DKA is a condition which is caused due to the complication of diabetes that results in accumulation of ketone bodies and acidosis in large quantities in the blood. It occurs as a result of a complication of Type 1 Diabetes Mellitus (T_1DM) and rarely in Type 2 Diabetes Mellitus (T_2DM) in children and adult. Presentation of the case: A 10 year old girl had the complaint of polyuria and polydipsiaand within 2 weeks her weight has reduced to 10 kg; she also had itching, burning sensation during micturition and history of vomiting 2 episodes. She was diagnosed as newly diagnosed T_1DM with DKA. She was given with Intravenous Fluids, cefotaxime, ranitidine and insulin. She was admitted back with complaint of fever, cough, headache and vomiting. She was managed for the condition and discharged. Conclusion: DKA is found to be common; hence early diagnosis and proper education and public awareness should be initiated.

Keywords: *Diabetic Ketoacidosis, Children, Polydipsia, Polyuria, Type 1 Diabetes Mellitus.*

Introduction

Diabetic Ketoacidosis (DKA) is a complex metabolic state characterized by hyperglycaemia, ketonemia and acidosis. This is caused due to the complication of diabetes resulting in accumulation of ketone bodies and acidosis in large quantities in the blood. It occurs as a result of complication of Type 1 Diabetes Mellitus (T_1DM) and rarely in Type 2 Diabetes Mellitus (T_2DM) in children and adult [1, 3, 4].The biochemical criteria for diagnosis of DKA is hyperglycaemia [blood glucose (BG) higher than 11 mmol/L or ≥200 mg/dl) with a venous P^H of <7.3 and/or a bicarbonate (HCO_3) level of <15 mmol/L; ketonemia, ketonuria and blood ß-hydroxybutyrate concentration should also be measured whenever possible, and a level of ≥3 mmol/L is the indication for DKA, but this test is not available universally [3,5,7]. Biochemical Criterion for diagnosing DKA is shown in Table 1.

Table 1: Biochemical criterion for diagnosing DKA

	Mild DKA	Moderate DKA	Severe DKA	HHS
Blood glucose	More than 200 mg/ dl	More than 200 mg/ dl	More than 200 mg/ dl	More than 600 mg/ dl
Venous ph	Less than 7.3	Less than 7.2	Less than 7.1	More than 7.3
Serum bicarbonate	Less than 15 mEq/l	Less than 10 mEq/l	Less than 5 mEq/l	More than 15 mEq/l
Urine Ketones	Positive	Positive	Positive	Small or None
Blood Ketone	Positive	Positive	Positive	Small or None
ß-hydroxybutyrate	High	High	High	Normal or Elevated
Serum Osmolality	Variable	Variable	Variable	Variable
Alteration in mental status	Alert	Alert/ drowsy	Stupor/ coma	Stupor/ coma

In Pathogenesis of DKA, generally insulin metabolises glucose into glycogen which is used as a source of energy. But in diabetic patients the severe lack of insulin causes lipolysis in the adipose tissues, resulting in the release of free fatty acids into the plasma. These free fatty acids are taken up by the liver where they are oxidized by acetyl-

coenzyme- A to ketone bodies, mainlyacetoacetic and Beta (ß) hydroxyl butyric acid. Such free fatty acid-oxidation to ketones is accelerated in the presence of elevated glucagon levels. Once the level of ketogenesis exceeds the rate at which the ketone bodies can be utilized, ketonemia &ketonuria occurs. Hyperketonaemia induces metabolic acidosis, respiratory compensation and marked increasein urinary excretion of acetoacetic acid and ß- hydroxyl butyric acid.

Acetone derived from the spontaneous decarboxylation of acetoacetic acid accumulates in the plasma and is slowly disposed off by respiration. At the cellular levels, the abnormal ketogenesis in DKA results from loss of insulin's normal modulating effects on free fatty acid released from adipose tissue and on the hepatic free fatty acid oxidation and ketogenesis. As a result, plasma free fatty acid and free fatty acid uptake by the liver are greatly increased. In the liver, insulin normally regulates free fatty acid oxidation and ketogenesis by indirectly inhibiting the transport of coenzyme- A derivatives of free fatty acid across the inner mitochondrial membrane and into the mitochondrial matrix. Glucagon stimulates hepatic long-chain fatty acid coenzyme- A transport for ketogenesis and oxidation in mitochondria. In DKA, the normal opposing effects are lost [8, 10, 11].The clinical manifestations of DKA are dehydration, tachycardia, tachypnoea, nausea, vomiting, polyuria, polydipsia, weight loss, fruity breath odour, and abdominal pain [7, 9, 12, 13].

Risk factors are young children who are less than 2 years, delay in diagnosis of DM, poor diabetes control, previous episodes of DKA, missed insulin injections etc [7, 9, 14].Complications of DKA include inadequate rehydration, hypoglycaemic and hypokalemia which is commonly found and cerebral oedema, pulmonary oedema. The serious and most frequent seen complication in the paediatric population is cerebral oedema [5].The severity of DKA is categorized by the degree of acidosis which is given below Table 2 [4].

Table 2: Severity of the acidosis

Severity	Venous pH	And /or Bicarbonate Concentration
MILD	Venous p^h less than 7.3	Bicarbonate concentration of less than 15 mmol/L
MODERATE	Venous p^h less than 7.2	Bicarbonate concentration of less than 10 mmol/L
SEVERE	Venous p^h less than 7.1	Bicarbonate concentration less than 5 mmol/L

Diagnosis of DKA includes initial assessment of the severity of dehydration, level of consciousness through Glasgow Coma Scale, body weight and height if the person is mobile. Baseline investigations involve the measurement of Blood Glucose (BG) levels, beta-hydroxybutyric acid, serum electrolytes and renal functions. During physical examination, the physician may look for signs of dehydration, acidosis, and electrolyte imbalance, including shock, hypotension, acidotic breathing, central nervous system (CNS) status, etc [5].

Case Report

A 10 years old female patient was admitted with the complaint of polyuria, polydipsia, weight loss within 2 weeks was around 10 kg, burning sensation during micturition& itching; had 2 episodes of vomiting on the 12th of October 2018. She was diagnosed asT_1DM with the findings of blood report as given below in Table 3. Family history shows that her grandmother has T_2DM since the past 3 years and she is on regular medication. On examination, the patient's weight was found to be 19 kg, pulse rate was 120 beats/mins and respiratory rate was 30/minutes. She was diagnosed as newly diagnosed T_1DM with DKA. On the day of admission she was prescribed with intravenous fluids (IVF) normal saline at the rate of 60 micro drops per minutes. Along with this, injection cefotaxime(1g) IV twice daily and injection ranitidine 20 mg IV twice daily was given. On 13th October 2018 at 2.00 PM (NS) Potassium chloride (KCl) was added at the rate of 40 millilitres (ml)/ hour (hr) with 10 ml KCl in 500 ml NS along with normal saline. Also regular insulin 2 units/hr was added and the input-output chart wasmonitored as shown in Table 4.

She was referred to Paediatric intensive care unit (ICU) in tertiary care hospital for further management. She was readmitted on 26th of October 2018 at the Government Hospital (GH) with a complaint of fever, cough since one day and she had a history of headache and vomiting. Her vitals were normal. The laboratory investigations reports

are given below in Table 5. She was on regular insulin injection 40 IU/ 10 ml (5 -12 - 5 units) and isophane insulin injection (NPH) 40 IC/ 10 ml (8 -0-5 units). She was prescribed with normal saline 200 ml over 1 hour, cefotaxime 1 gram IV BD, amikacin 300 mg IV once daily, ondansetron 2 mg IV BD, ranitidine 20 mg IV BD and Tab.paracetamol 500 mg 3/4th four times a day which was given on admission and along with this medication insulin injection was also continued.

On day 2, she had a complaint of cough, fever and throat pain and she was given Inj.cefotaxime 1 gram IV BD, Inj.amikacin 300 mg IV once daily, Inj. ranitidine 20 mg IV BD, Tab. ibuprofen 200 mg twice daily, Tab.ranitidine 150 mg twice daily and cough syrup ½ teaspoons thrice daily and Tab.paracetamol 500 mg ½ four times a day. On day 3, she had complained of hypoglycaemia at night and the fasting blood sugar (FBS) was 67 mg/ dl and the same medication was continued expect Tab.ibuprofen. On day 4, she had a complaint of cough and here FBS was 103 mg/dl. Insulin was titrated to 5- 5- 5 units of regular insulin and no change in dose for NPH was done. Other medications were continued.

On day 5, she had a complaint of cough and she was asked to continue the same medications. On day 6, her cough had reduced and Capillary Blood Glucose (CBG) was 149 mg/dl and again her regular insulin was titrated to 5 units-10 units- 5 units and she continued other medications. On day 7, she was discharged with regular insulin 5-10-5 units, NPH 8-0-5 units and also the last dose of cefotaxime and amikacin were given and was to continue on cough syrup 2.5 ml at bedtime * 3 days. She was advised to take insulin as per the schedule.

Table 3: Laboratory Investigation Reports

Investigations	Before admission	Day of admission
Blood Sugar	R- 589.3 mg/ dl	R- 284 mg/ dl @ 11.30 AM R- 247 mg/ dl @ 12.30 AM R- 570 mg/ dl @ 2.00 PM F- 380 mg / dl
Sugar	++++	+++ @ 2.00 PM
Acetone	-	Positive @ 11.30 PM Positive @ 2.00 PM
Bile salt	Negative	-
Pus Cells	2-3 hpf	1-2 hpf
Epithelial Cells	0-1 hpf	2-3 hpf
pH	5	7.35
Po_2	28 mmHg	-
Pco_2	27 mmHg	27 mmHg
Hco_3	14.8 mmHg	17 mmHg
Sr. Hco_3	17.1 mmHg	-
Sodium	126 mmol/ L	126 mmol/ L
Potassium	4.7 mmol/ L	4.7 mmol/ L
Calcium	4.2 mmol/ L	-
pH	7.35	7.35

Table 4: Input Output Chart

TIME (PM/AM)	INPUT	OUTPUT
2.15 PM	10 ml (water) & Normal Slaine 60 ml/ hr	300 ml
3.20 PM	40 ml (water) & Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	100 ml
5.30 PM	50 ml (water) &Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	100 ml
6.30 PM	20 ml (water) &Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	100 ml + urine PD
7.45 PM	20 ml (water) & 5% DextroseNormal Saline 40 ml/ hr with 10 ml Potassium Chloride	100 ml + urine PD
8.00 PM	20 ml (water) &5% Dextrose Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	100 ml + urine PD
8.30 PM	Stopped 5% Dextrose Normal Saline With Potassium Chloride	
9.30 PM	40 ml (water) &Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	250 ml + urine PD

10.30 PM	40 ml (water) &Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	250 ml + urine PD
11.30 PM	40 ml (water) &Normal Saline 40 ml/ hr with 10 ml Potassium Chloride	250 ml + urine PD

Table 5: Laboratory Investigations Report after readmission

Investigations	Values
Haemoglobin	13.6 g/dl
Total count	$11.2*10^3$ cells/mm^3
Red Blood Cells	$5.60*10^6$ cells/ l
Haematocrit	40.7%
Epithelial pus cells	3-4/ hpf

Discussion

DKA is a life-threatening condition in which early diagnosis should be made. It is commonly found in younger children (predominantly < 2 years) and may lead to severe DKA and coma, occasionally death. The frequency varies geographically; the overall frequency was estimatedapproximately 20 % to 67% [4, 15]. Newly diagnosed with type 1 DM accounts for approximately 15 % - 25 % of DKA [3, 15].

Misdiagnosis of diabetes is very commonly seen in younger children (34% of children ≤5 years of age compared to 8.5% in those greater than 10 years of age) [2] .The goal of the therapy is to manage dehydration, control blood glucose, correct ketoacidosis, hypovolemia and monitoring and management of the complication [3,7].

Complications of DKA are cerebral oedema, Hypokalaemia, Hyperchloremic acidosis, Hypoglycaemia and Inadequate rehydration. DKA management in children is not as same as that in adults. In children, the metabolic rate and surface area are different when compared to adults and special care is needed to manage the electrolytes [3]. Insulin infusion should not be discontinued until acidosis is corrected, because insulin will stop the production of ketones.

While managing the DKA, potassium should be given because insulin will drive glucose and potassium into the cells, thereby reduction in serum potassium level will be observed. Therefore potassium should be added even though the serum potassium level is normal [3]. The limitation was, we were unable to collect the management given in Coimbatore Medical College and Hospital. The patient was advised to take insulin regularly 25-30 mins prior to food. Prefer a small diet rather than having a heavy or large diet. The patient was advised not to skip or miss any dose of insulin and not to discontinue or withhold insulin. Patient was advised to take fibre rich food rather than food containing carbohydrate andto check glucose level and ketone bodies regularly. Directions were given to store insulin at/ below 4^0 C and to not rub the site of injection. Site for injecting insulin are 1) Abdomen: Stay 2 inches away from the belly button or scars. Insulin is absorbed best from the abdomen 2) Arms: Measure one hand width down from the shoulder and one hand width up from the elbow. Use the fleshy outer surface 3) Legs: Measure one hand width down from the groin and one hand width up from the knee. Use the top and outer part of the leg staying away from the inner part of the thigh 4) Buttocks: Use the upper outer area.

Conclusion

DKA is one of the common presentations in type 1 and type 2 DM in children and adolescents. We conclude that initial diagnosis is necessary for children and adolescents. When comparing both, children need more priority than adolescents. The purpose and key to prevent the DKA are through early recognition and managing DM. As DM is more likely to occur in younger children, public awareness is essential. Awareness programmes should be conducted by healthcare professionals regarding early detection of DM prior to the onset of DKA. Managing DKA without any complications in younger children can be done with the support of the American Diabetes Association.

The role of the pharmacist is to teach the patient about how and where to administer insulin. Advice the patient to monitor blood glucose frequently. Pharmacists need to be particularly attentive to patients who are at high risk for DKA such as patients who have poor diabetes control, patients with lower socioeconomic status or inadequate access to

outpatient care, adolescents who have or may have eating disorders and patients with recurrent ketoacidosis. Patients treated with insulin pumps also have a higher risk for DKA as undetected insulin delivery interruption can precipitate DKA in as little as 4 hours.

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