



Case Report

Successful medical management of diabetic ketoacidosis at first presentation in a child with type 1 diabetes: A case report

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ABSTRACTS

Introduction and importance: Diabetic ketoacidosis (DKA) is considered to be a common presentation of type 1 diabetes mellitus in children. It occurs when absolute or relative insulin insufficiency prevents glucose from entering the cells for use as metabolic fuel, causing the liver to quickly break down fat into ketones for use as fuel source. As a result, ketones are overproduced, accumulating in the blood and urine making the blood acidic.

Case presentation: A 4 years and 8 months old child presented with the complaint of abdominal pain and vomiting along with polyuria, polydipsia and polyphagia. Routine examination of blood revealed that increased random blood glucose level. Once diagnosed, DKA was managed with fluid and insulin therapy with close monitoring and supervision.

Clinical discussion: DKA can be easily diagnosed. Proper management should be done on time to prevent complications like hypokalemia, hyponatremia leading to cerebral edema and shock.

Conclusions: Diabetic awareness programs and school educational tutorials are beneficial for community awareness of the signs and symptoms of diabetes.

1. Introduction

Type 1 diabetes is due to autoimmune destruction of pancreatic beta cells which leads to insufficient insulin production resulting in hyperglycemia [1]. The common symptoms presenting in type 1 diabetes are polyuria, polydipsia, and weight loss [2]. Severe fall in insulin levels leads to increase in lipolysis which will lead to increase in level of ketone bodies resulting metabolic acidosis and compensatory respiratory alkalosis [3]. Diabetic ketoacidosis (DKA) is the common presentation of type 1 diabetes mellitus in children. The prevalence of onset of diabetic ketoacidosis among type 1 diabetes mellitus was found to be 26.3% in one of the studies [4]. Common complications observed due to ketoacidosis are electrolyte abnormalities like hypokalemia, hyponatremia leading to cerebral edema and shock [5].

If Diabetic ketoacidosis is not treated on time, the compensatory mechanism will fail soon and lead to cerebral edema, mental confusion, unconsciousness, coma and death [3,6]. DKA is the most common cause of death in children and adolescents with type 1 diabetes and cause of half of all deaths in diabetic patients under the age of 24 years [7].

Immediate and aggressive intervention is required. Early medical management could prevent complications like cerebral edema, mental confusion, shock and death. Here, we present a successful medical management of a diabetic ketoacidosis as a first presentation in a child with type 1 Diabetes mellitus. This case has been reported accordingly in line with SCARE 2020 criteria [8].

2. Case presentation

A 4 years 8 months old school going male child presented to the Emergency department of Shree Birendra Hospital with complaints of abdominal pain in periumbilical region for 1 day. Pain was mild, acute on onset, non-radiating associated with irritability. He also had a history of multiple episodes of vomiting for the last 3 days. Vomitus was non-projectile consisting of partially digested food particles and water. It was non-bile stained, was not mixed with blood, and was non-foul smelling. He also had a history of polyuria, polydipsia, and polyphagia for 7 days. Day by day there was an increase in frequency of urine which was associated with increased water intake. There is a history of a 2 kgs

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weight loss within a week. He was born healthy at term following an uncomplicated pregnancy and was the second child of a non-consanguineous marriage. There was no any family history of type I DM and any chronic illness.

On physical examination, the child was irritable and dehydrated with a dry tongue and mucosa. Vitals recorded at the time of admission were as follows; blood pressure of 80/50 mmHg, pulse rate of 128 beats/min and were low volume, respiratory rate of 24 breaths/minute, oxygen saturation of 95% on room air, and body temperature were 98 °F. Systemic examinations were normal.

Routine examination of blood revealed an increase in random blood glucose level with value of 448 mg/dl (ref. 140 mg/dl) and hyponatremia with value of 130 mEq/L (ref. 136–145 mEq/L), however serum potassium, urea and creatinine levels were normal. Urine examination revealed that the urine was acidic, acetone positive and sugar was present in urine. Arterial blood gas analysis showed pH 7.23 (ref. 7.35–7.45), pCO₂ 31 (ref. 35–45 mmHg), HCO₃⁻ 13.21 (ref. 22–26mmol/L), and PO₂ 102 (80–105 mmHg). Hematological examination was unremarkable.

Based on hyperglycemia, metabolic acidosis, and ketonuria a diagnosis of DKA was made and management was initiated. The child was shifted to Pediatrics intensive care unit (PICU) where he was given intravenous fluid of 320 ml normal saline IV over 1 hour at the rate of 20 ml/kg. Similarly, regular insulin 0.5 ml hourly at the rate of 0.05 unit/kg hr (1ml insulin in 23ml NS) and injection ceftriaxone 500 mg IV 12 hourly at the rate of 63 mg/kg/day were administered for the next 23 hours. The child was kept in N/2 IV Fluid 100 ml/hourly with 1mEq KCl in each 100 ml IV fluid. If random blood sugar fell below 250 mg/dl, N/2 IV fluid and 5% dextrose at 100ml/hourly with 1mEq KCl in each ml of 100 ml IV fluid was indicated. We kept our patient on nil per oral. Similarly, if random blood sugar falls below 200 mg/dl, N/2 IV fluid and 10% dextrose at 100 ml/hourly was indicated.

Further, the vitals were monitored hourly; random blood sugar and neurological assessment were done 2 hourly; renal function test, electrolyte were monitored 6 hourly. X-ray of chest to rule out pulmonary infections such as pneumonia, ultrasonography of abdomen to rule out any organ damage, ophthalmology consultations to rule out papilloedema were done. There were no significant findings on any one of them. After 5 days on PICU, our patient's symptoms gradually improved. He was started on an oral fluid and liquid diet. The random blood glucose levels monitored at different time intervals were in the normal range. We discharged the patient after 7 days of admission with advice of Insulin Glargine 6 units sc once a day and Insulin Lispro 4 units before breakfast, lunch and dinner. The patient was properly instructed to follow up after 1 week for insulin management as per glucose report.

3. Clinical discussion

DKA is life threatening complications of uncontrolled diabetes mellitus if proper intervention is not done on time [9]. Risk of developing DKA at manifestation of diabetes is high in young children (<2 years), girls, children of ethnic minority status, low socio-economic status [10–12]. Successful management of diabetic ketoacidosis depends upon swift diagnosis, regular monitoring of clinical and biochemical parameters with prompt intervention. The diagnosis of DKA can be made on the basis of biochemical criteria of random blood glucose level greater than 200mg/dl with a venous pH of level <7.3 and/or a bicarbonate (HCO₃) level of <15 mmol/L; ketonemia and ketonuria [11]. Early detection of diabetic ketoacidosis in our case led to proper medical management of a patient preventing him from complications like cerebral edema. However, diagnosis of DKA should not be confused with asthma, hypokalemia, metabolic acidosis, respiratory acidosis, pneumonia, salicylate poisoning, acute abdomen, gastroenteritis etc. [13].

Muktan et al. in their retrospective study found that polyurea, polydipsia, weight loss, abdominal pain, vomiting as the most common symptoms of DKA [14]. Our patient also presented with similar

symptoms from which we made a provisional diagnosis of DKA after physical examination which was later confirmed by biochemical examination. DKA can be managed in any hospital/private unit or in a pediatric inpatient ward in case of children by trained nursing and medical personnel.

Rosenbloom et al. in his study had described the management of Diabetic Ketoacidosis depends on the severity of DKA. The severity of DKA is categorized by acid-base status in which mild DKA has pH 7.2 to <7.3; bicarbonate 10 to <15 mEq/L, moderate DKA has pH 7.1 to <7.2; bicarbonate 5 to 9 mEq/L and severe DKA has pH <7.1; bicarbonate <5 mEq/L [15]. Our patient had pH level of 7.23 and bicarbonate level at 13.21. Thus, he had a mild DKA and was treated accordingly.

The child presented with DKA should be closely monitored in the unit. Blood glucose, electrolyte level, neurological assessment, vitals, and urine routine examination should be monitored on hourly basis [16]. We monitored our patient's vitals hourly; random blood glucose level and neurological assessment 2 hourly; electrolyte, input/output charting, and urine routine examination 6 hourly.

The patient with DKA is treated with intravenous fluids and intravenous insulin if the child is nauseated/vomiting, clinically dehydrated or is not alert [17]. We managed our patient as per his symptoms. We used normal saline for the first 24 hours to treat and manage dehydration and mild sodium depletion. We kept our patient on Insulin to control increased random blood glucose level. KCL along with intravenous fluids was given to manage the impending hypokalemia. In order to treat and prevent possible bacterial infections, ceftriaxone was given. Since we kept our patient nil per oral, dextrose in intravenous fluids was introduced the next day.

4. Conclusion

We present a case of diabetic ketoacidosis in a child with type 1 diabetic mellitus. It is a life threatening complications if timely intervention is not done. Timely management with fluid therapy along with insulin should be done. Regular monitoring and neurological observation are equally important to prevent complications like cerebral edema. Educational programs, diabetes awareness campaigns, and school educational tutorials can be beneficial for community awareness of the signs and symptoms of diabetes.

Author agreement statement

We the undersigned declare that this manuscript is original, has not been published before and is not currently being considered for publication elsewhere.

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

We understand that the Corresponding Author is the sole contact for the Editorial process. He/she is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs.

Ethical approval

This is a case report, therefore, it did not require ethical approval from the ethics committee.

Consent

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the editor-in-chief of this journal on request.

