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Case Report

A CASE OF EUGLYCEMIC DIABETIC KETOACIDOSIS IN A YOUNG GIRL WITH TYPE 1 DIABETES MELLITUS: A RARE PRESENTATION.

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Abstract: Euglycemic diabetic ketoacidosis (DKA) is a rare condition and has been reported in a few patients only. We report the case of a 17 year old girl with Diabetic ketoacidosis without abnormal blood glucose level who recovered with proper medical management.

Key words: Euglycemic, ketoacidosis, hyperglycemia.

Introduction: Diabetic ketoacidosis is defined as the presence of hyperglycemia (blood glucose > 250mg/dl) with ketosis and acidemia (pH <7.3).Despite elevated blood glucose levels accepted as necessary in the definition of DKA, unusual cases with only modestly increased blood glucose concentrations have been reported, and the term euglycemic diabetic ketoacidosis is used to define these cases. DKA can present with a normal or even low blood glucose level due to the complex interplay between substrate availability , action of insulin and counterregulatory hormones, and hydration status [1].However patients with diabetes may be at risk of developing ketoacidosis from conditions seen in non diabetics.Therefore making a distinction between the two is essential. About 10% of DKA population presents with so called Euglycemic DKA [2] .Our case is a 17 year old type 1 diabetic girl who presented with euglycemic diabetic ketoacidosis. Patient had severe ketoacidosis and was managed successfully in the ICU with intravenous insulin infusion and discharged with controlled blood sugars on subcutaneous insulin.

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Case Report: A 17 year old female, student of class XI, known case of Diabetes Mellitus for past 4 years on insulin therapy, was admitted with recurrent vomiting for 2 days. She was vomiting irrespective of eating anything, with vomitus containing yellowish, sour, non foul smelling fluid and food particles. There was no polyuria or polydipsia. She did not have any abdominal pain or alteration in bowels, fever, dysuria or jaundice. There was no associated headache or eye complaints, any alteration in

sensorium, head trauma or abnormal body movements. At home, she was on 12 units of mixact (30/70) insulin bd. She did not have any prior hospital admissions.

On examination, patient was conscious, well oriented with time, place and person, with a pulse of 110/min, regular; BP of 110/70 mmHg with no postural drop; temp of 98.2^0F; she was tachypneic with a respiratory rate of of 24/min and an acidotic breath. She was pale and mildly dehydrated. Weight was 45 kg, height 154 cm, BMI 18.98. There was no abdominal tenderness, neck was free and fundus was normal. Rest of the systemic examination was also unremarkable.

Investigations revealed a total leucocyte count of 15,300, blood sugar random of 150mg/dl ,repeat of 128 mg/dl ,arterial pH of 7.29 and a normal oxygen saturation (98%) on room air. .Renal function test ,liver function test, complete blood count and chest X ray were normal. Serum sodium was 136mmol/L and potassium of 4.6 mmol/L. Hb A1 c level was 7%, suggesting good glycemic control. Urinalysis revealed ketonuria without glycosuria.

Based on the above findings, a diagnosis of euglycemic diabetic ketoacidosis was made. In the casualty she was managed with oxygen, i/v fluids, Insulin , ondansetron and pantoprazole. Later the patient was shifted to ward where subsequent investigations revealed a blood sugar random of 480 mg/dl. Arterial pH was 7.03 and bicarbonate was 3.0.Patient was given Regular Insulin at the rate of 8 u/h and i/v fluids as Normal saline infusion at a rate of 50ml/h, potassium replacement and antibiotics, Piperacillin with Tazobactam were empirically added. Further investigations showed a blood sugar 340 mg/dl , urine was positive for ketones and sugar .Patient was shifted to medical ICU where instructions for hourly glucose monitoring , bicarbonate replacement and Insulin infusion were given till blood sugar reached 150mg/dl and bicarbonate 10 mEq/L. The various lab parameters of the patient during ICU care are displayed in Table 1. Intravenous Insulin infusion was given along with intravenous bicarbonate and dextrose. During further course of treatment in medical ICU, patient's arterial pH rose to 7.43 and bicarbonate 16.3 mEq/L while her blood sugars were also controlled. Overall condition of patient improved within 22 hours and she became completely stable. She was discharged with a blood sugar of 90mg/dl fasting and 239mg/dl post prandial on subcutaneous Insulin.

Discussion: DKA is a state of absolute or relative Insulin deficiency. DKA is frequently associated with decrease in calorie intake which may be due to vomiting or worsening ketoacidosis itself. Those patients who continue taking Insulin may be euglycemic but the ketone body formation continues. Thus such patients present with mildly elevated glucose levels or euglycemia. Prolonged fasting also depletes the glycogen reserves which further contributes to normoglycemia [3],[4],[5],[6],[7).Fasting also leads to accelerated lipolysis and free fatty acid production. Insulin is less effective in suppressing lipolysis and ketogenesis during a fast, further aggravating acidosis. Munro et al has defined Euglycemic diabetic ketoacidosis as DKA with blood glucose <300 mg/dl and plasma bicarbonate $\leq$ 10 meq/L. Predisposing factors are young age, alcohol ingestion, glycogen storage diseases, chronic liver disease [8,][9] , severe depression [10] and pregnancy [11].The average glucose of pregnant patients with DKA was 16 mmol/L compared with 27 mmol/L in non pregnant patients [12].Cases have also been reported in patients with Duchene [13] and other forms of muscle dystrophy , likely due to reduced muscle mass and deficiency of substrate for gluconeogenesis, subnormal muscle content of lactate [14] and subnormal plasma plasma alanine levels [15],[16].

Patients with diabetes may be at risk of developing acidosis from conditions that are also seen in patients without diabetes, and a distinction between non diabetic euglycemic ketoacidosis and euglycemic DKA is essential. These include differential diagnosis such as prolonged starvation, excess alcohol consumption, salicylate overdose, lactic

acidosis, tricyclic antidepressant overdose, and renal tubular acidosis [17],[18].Coccaine abuse is a trigger for euglycemic DKA in Type 1 diabetic patients due to its stimulatory effect on cortisol, epinephrine, and norepinephrine release from the adrenal gland [19],[20]. Euglycemic DKA also appears to be a worrisome adverse event associated with SGLT-2 inhibitor use in patients with type 1 and type 2 diabetes [21].

Munro FJ et al. found that euglycemia in DKA may vary from 39 mg/dl to 299 mg/dL(3).In 1973, Munro et al reported a series of 211 episodes of DKA, of which 37 episodes were described as euglycemic (defined as blood glucose of 300mg/dl(16.7mmol/l) or less with plasma bicarbonate of 10 mEq/l or less. Jenkins et al (1993) reported 23 episodes of euglycemic DKA (in a series of 722 episodes), based on the same diagnostic criteria. It has since been argued that glucose readings above 200mg/dl (11.1mmol/l) cannot be considered to represent euglycemia, and therefore a blood glucose level of 200mg/dl(11.1mmol/l) should be used as a cutoff for defining true euglycemic DKA. Based on this criterion, only 16 of the 37 episodes in the study by Jenkins et al could be described as true euglycemic DKA. Since ketoacidosis is generally regarded as the metabolic outcome of excessive gluconeogenesis coupled with increased fatty acid release , it seems difficult to postulate that the relative euglycemia is due to a lesser glucose formation in such cases. A greater urinary glucose loss seems more likely. Whether this is a consequence of increased growth hormone secretion affecting renal function , related to the enhanced clearance reported in early diabetic involvement [22],[23] or simply represents one end of the spectrum in terms of renal threshold to glucose, remains to be determined.

The triad of hyperglycemia, ketosis and acidosis can be diagnosed within a few minutes of the patient presenting, by measuring blood glucose and ketones using a meter , and venous blood pH on a blood gas analyser [24].The initial management should be , as in any case of ketoacidosis ,to correct the fluid/ electrolyte abnormalities and re establish carbohydrate metabolism. Increased glucose administration using higher percentage of dextrose are required to facilitate the concomitant administration of the relatively large amounts of Insulin that are needed to correct the severe acidosis in these patients. It is recommended that patient should be started on constant insulin infusion and managed afterwards with 5% or 10% dextrose [25],[26]. Acidosis should improve with normalization of serum bicarbonate without the need for intravenous bicarbonate administration.

Conclusion: It is emphasized that DKA should be strongly considered in diabetic patients presenting with acute illness and acidosis, irrespective of blood glucose level, and the normalcy of latter should not falsely reassure the clinician. Assessment of the acid base status, urinary ketone readings is therefore important in all such patients at admission who have normal glucose levels.

Table 1.Biochemical parameters of the patient sequentially in the ICU over first 5 hours:

Glucose	150	128	480	340	288	259	293	193	150
pH	7.29	7.20	7.03	6.98	7.34	7.37	7.43	7.44	7.43
Hco3	16.3	14.0	3	3	9.7	14.5	20.3	22.0	23.9
pO2	91	92	92	90	93	92	91	92	92
pCO2	21	19	9	7	18	35	31	34	36
Anion gap	23	24	20	22	18	16	15	11	10

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