

NORMAL BLOOD GLUCOSE LEVEL AT PRESENTATION DOES NOT RULE OUT DIABETIC KETOACIDOSIS IN A SICK CHILD

To the editor: A 3-year-old male child was brought to the emergency department with fever, vomiting, and rapid breathing for 2 days. He was asymptomatic and thriving prior to his present illness. His pulse rate was 126 beats/min, respiratory rate was 68 breaths/min, and blood pressure was normal for age, and there were signs of some (5%) dehydration. Systemic examination was unremarkable except for lethargy. Preliminary investigations revealed blood glucose of 5.3 mmol/L, serum sodium of 144 mmol/L, and potassium of 4.6 mmol/L. Venous blood gas analysis showed metabolic acidosis with a pH of 7.14 and bicarbonate of 10 mmol/L. A chest radiograph was normal. In view of the significant acidosis, his urine was tested by the dipstick method, which showed a large amount of ketonuria without glucosuria. With the strong suspicion of diabetic ketoacidosis (DKA), intravenous insulin infusion was started along with fluid (0.45% saline in 5% dextrose with potassium chloride 40 mmol/L) replacement. The glycosylated hemoglobin (HbA_{1c}) level was 7.5% (normal < 5.9%), supporting the diagnosis of DKA.

The child showed gradual improvement in clinical parameters and resolution of his metabolic acidosis. He had a few episodes of hyperglycemia (blood glucose > 10 mmol/L) while being titrated with subcutaneous insulin. At follow-up, the

child was growing well and maintaining acceptable blood glucose and HbA_{1c} levels on intermittent insulin injections.

In the emergency setting, random blood glucose estimation is part of the screening for DKA given the classic description that includes hyperglycemia with acidosis and ketonuria. However, due to the complex interplay between substrate availability, action of insulin and counterregulatory hormones, and hydration status, DKA can also present with a normal or low blood glucose level.¹ Jenkins and colleagues identified 0.8 to 1.1% of the 722 DKA episodes to be euglycemic.² Most of the reported pediatric cases had previously diagnosed type 1 diabetes mellitus and had received insulin just prior to presentation; previously undiagnosed diabetes presenting with euglycemic DKA was rare.³ It is hypothesized that poor oral intake and vomiting, as in our patient, may decrease the available carbohydrate reserve and result in a normal blood glucose level.

A low glucose level should not deter the use of insulin because insulin's primary role in DKA is to reverse ketosis. Failure to start insulin may allow glycogenolysis, lipolysis, ketogenesis, and metabolic acidosis to progress unchecked. Rapid rehydration (> 4 L/m²/24 h) during early management, which may be appropriate for uncomplicated dehydration, may increase the risk of cerebral edema in DKA. Life-threatening hyperkalemia and depleted intracellular potassium and phosphate concentration may be formidable to correct with rehydration

alone, increasing the risk of exposure for the patient.

The decision to initiate insulin therapy should best be based on certain markers suggesting DKA as the cause of ketoacidosis. Primary starvation ketoacidosis is not expected if the child is in satisfactory physical and social condition. A history of adequate or high urine output favours the possibility of DKA and does not support starvation and dehydration as the cause. Usually, children with primary sepsis have clinically evident symptoms and signs if presenting with such a degree (as in this case) of metabolic acidosis. Elevated free fatty acids suggest an insulin deficiency state. However, it may be difficult to rule out certain organic acidemias or a severely septic child with ketocidosis.⁴ The therapeutic response to insulin and dextrose infusion under strict clinical and biochemical monitoring seems the best approach.

Hyperglycemia was almost certainly present prior to the onset of the DKA, given the elevated HbA_{1c}.

DKA should be strongly considered in children presenting with acute illness and acidosis, irrespective of the blood glucose level. A delay in the diagnosis of DKA and an infusion of insulin can significantly alter morbidity and mortality in these patients.

Bhanu Kiran Bhakhri, MD

Assistant Professor, Department of Pediatrics, Lady Hardinge Medical College & Associated Hospital, Shahid Bhagat Singh Marg, New Delhi, India

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