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CLINICAL STUDY

Bicarbonate therapy in diabetic ketoacidosis

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Abstract: In the treatment of diabetic ketoacidosis (DKA), the aggressive management of hyperglycemia, avoidance of hypoglycemia, and anticipation of the diabetic systemic complications improve patient outcome. In past, the bicarbonate therapy in DKA has been advocated. Yet, no prospective randomized studies on the use of bicarbonate in DKA with arterial pH values <6.9 have been published. However, the bicarbonate therapy in DKA has several potentially deleterious effects including worsening of hypokalemia and intracellular acidosis. Here we present a patient with DKA pH<6.9, treated with bicarbonate, who demonstrated a clinical benefit *(Ref. 11)*. Full Text (Free, PDF) *www.bmj.sk*.
Key words: diabetic ketoacidosis, hyperglycemia, bicarbonate.

Diabetes mellitus (DM) is a common diagnosis in patients requiring a critical care. Patients may present with the diabetic ketoacidosis (DKA) as a primary diagnosis, a complication of another diagnosis, or may develop DKA during the course of concomitant treatment. The most common causes of DKA are infection, noncompliance, and new-onset of type 1 diabetes (1). A successful therapy requires the correction of dehydration, hyperglycemia, and electrolytes imbalances; and identification of co-morbid precipitating events (2). A routine use of supplemental bicarbonate is not recommended in the treatment of DKA (3). However it remains unclear whether specific DKA patients may benefit from bicarbonate therapy.

We present a case report on a diabetic ketoacidosis; the patient who had an intractable deep acidosis, had to undergo the bicarbonate therapy and had recovered without any complication.

Case report

A 42-year old male was admitted to our emergency department with shaking chills, fever, nausea and vomiting. At the admission, he was somnolent, with limited cooperation and orientation, and his Glascow Coma Scale (GCS) was 12 (E3 V4 M5). His relatives told that he had dysuria, frequency and urgency with an increased thirst for two weeks; but his weight loss was approximately 20 kg. No prior illnesses were present in past medical history. Blood pressure (BP) was 90/50 mmHg, heart rate (HR) 118 bpm, respiration rate (RR) 36/min with characteristic Kussmaul respirations and body temperature was 38.5 celsia grades axillary. The physical examination revealed an abdominal distention with decreased bowel sounds and prominently decreased skin turgor. Laboratory findings revealed leukocytosis of 16.200 mm3/dl, blood glucose 425 mg/dl, Na^+ 161 mEq/L, K^+ 1.9 mEq/L, Cr 1.9 mg/dl, Cl^- 116 mEq/L; urinalysis (++++) glucosuria, 300 WBC and urine ketostix reaction >(++++). Arterial blood gases (ABG) showed: pH: 6.82, PCO_2 "very low" (undetectable), PO_2: 86.4, HCO_3: 8.4, BE: -26.6, sPO_2: 99. ECG rhythm was a sinus tachycardia.

A diabetic ketoacidosis was the primary diagnose triggered by urinary tract infection. A normal saline infusion with the rate of 20 ml /kg/weight/hr for the first hour (2 L for the first 2 hours rapidly then 500 ml/h for the following 4 hours) and a low dose insulin administration (0.1 U/kg/h without loading dose) using an infusion pump started after the K^+ replacement therapy. K^+ replacement started at 40 mEq/L for the first 4 hours, followed by 10 mEq/h. The arterial blood gases at the second hour were the same, then HCO_3^- replacement started as 100 mmol diluted in 400 ml H_2O, infused at 200 ml /h as recommended in the last consensus statement (3) then checked with ABG levels every 2 h until pH>7.0. Levofloxacin started for the urinary tract infection and paracetamol was given for reducing the fever.

The patient was transferred to the intensive care unit for monitoring. Four hours after the initiation of therapy, vital signs started to improve (BP 100/50, HR 110 bpm, RR 28/min Kussmaull respirations disappeared and body temperature was 37.5 °C). Despite the replacement of HCO_3, the amount of CO_2 remains undetectable, pH<6.90 and tachypnea continued. At the eighth hour of the therapy, ABG measurements started to recover: pH: 7.06, PCO_2: 5.9, HCO_3: 14, and BE: 23.2. In the second day, all the vital signs and laboratory findings became normal and he

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was transported to the clinic of internal medicine for further evaluation and treatment.

Discussion

In the treatment of DKA, the aggressive management of hyperglycemia, avoidance of hypoglycemia, and anticipation of the diabetic systemic complications improve patient, outcome (1). In our patient, clinical and laboratory findings supported the diagnosis of DKA, however no published data exist on such a low PCO_2 level as "undetectable". It is a general knowledge and also published in literature that PCO_2 level may be low due to the compensative mechanisms of DKA. Even a low PCO_2 level, high serum urea, and bicarbonate therapy are the risk factors predictive of cerebral edema (4). Low measurements of PCO_2, as in our case report, first suggested a false result. However, the machine was calibrated and the serum examined twice. An elevated acetone production results in high partial pressures of arterial carbon dioxide, which worsen metabolic acidosis (5). In early phases, is it compensated with accelerating the respiration rate; afterwards the Kussmaul respirations can be clinically detected. Probably most patients are diagnosed in early phases of metabolic acidosis thus the treatment can be initiated before blood pH levels fall under 6,90. Our patient might be lately diagnosed; dependently his metabolic acidosis might be worsened and resulted in low (or "undetectable") PCO_2 levels.

In past, bicarbonate therapy in DKA has been advocated. But current reviews do not recommend the routine use of alkali therapy in DKA (6). Bicarbonate therapy in DKA has several potentially deleterious effects including worsening of hypokalemia, worsening of intracellular acidosis, delaying ketoanion metabolism and producing a paradoxical acidosis of the central nervous system (1, 6–9). Okuda et al (10) demonstrated a paradoxical increase in acetoacetate levels during the alkali administration and a delay in the improvement of ketosis. On the contrary, in our patient, 4 hours after the administration of bicarbonate therapy, a clinical response has been detected and acidosis gradually improved. Lever and Jaspan have observed 27 patients with diabetic ketoacidotic coma and blood pH below 7.10; all recovered to full alertness using the administration of bicarbonate infusions simultaneously with the increase of the low blood pH to 7.29–7.40 (8). But another prospective randomized study in 21 patients failed to show either beneficial or deleterious changes in morbidity or mortality using the bicarbonate therapy in DKA patients with an arterial pH between 6.9–7.1 on admission (11). No prospective randomized studies on the use of bicarbonate in DKA with arterial pH values <6.9 have been reported (9). Our case is an example and is supporting the usage of sodium bicarbonate when pH is lower then 6.9. The progress of our patient may help to develop treatment protocols until a well-designed prospective study establishes one. The last consensus statement from the American Diabetes Association about hyperglycemic crises concluded that severe acidosis may lead to a mydriad of adverse vascular effects, and the adult patients with pH<6.9 should receive sodium bicarbonate until the venous pH is 7.0 (3).

Conclusion

In the diabetic ketoacidosis, bicarbonate therapy still remains controversial. No prospective randomized studies on the use of bicarbonate in DKA with arterial pH values <6.9 have been reported so far. A severe acidosis (pH<6.90) despite the aggressive therapy may be an indication for the administration of sodium bicarbonate.

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