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Bicarbonate Therapy in Severe Diabetic Ketoacidosis

Ritu Baloda[1*], Tripat Kaur Bindra[2]

[1]Assistant Professor, PGIMS, Rohtak. Email: ritubaloda@gmail.com
*Corresponding author

[2]Professor, Government Medical College, Patiala

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Abstract

Background: Diabetic Ketoacidosis (DKA) is a known catastrophic complication of Diabetes Mellitus, which requires aggressive management including intravenous fluids, intravenous insulin to treat hyperglycemia, avoidance of hypoglycemia and correction of acid-base balance. Sodium bicarbonate is infused if decompensated acidosis starts to threaten patient's life, especially when associated with either sepsis or lactic acidosis. However, rapid and early correction of acidosis with sodium bicarbonate may worsen hypokalemia and cause paradoxical cellular acidosis, and has been correlated with cerebral oedema in children. **Material and Methods:** Two cases of severe DKA in ICU with pH<6.9. Initial treatment was given in form of IV fluids and insulin administration, but there was persistence of kussmaul's respiration, metabolic acidosis and hemodynamic instability. Then patients were given iv bicarbonate therapy and serial ABG analysis were done to monitor response. **Results:** After giving the bicarbonate therapy, clinical improvement was seen and acidosis improved in both the patients. No complication was noted. **Conclusion:** However it can be safely concluded that in severe DKA, it is worthwhile to give intravenous sodium bicarbonate .Our case report and similar case reports by other authors provide evidence in favour of it. However, large scale prospective trials need to be done for establishing routine use of bicarbonate therapy in all cases of DKA.

Keywords: Diabetic Ketoacidosis, Sodium Bicarbonate.

INTRODUCTION

Diabetic Ketoacidosis(DKA) is a known catastrophic complication of Diabetes Mellitus, which requires aggressive management including intravenous fluids, intravenous insulin to treat hyperglycemia, avoidance of hypoglycemia and correction of acid-base balance. Sodium bicarbonate is infused if decompensated acidosis starts to threaten the patient's life, especially when associated with either sepsis or lactic acidosis. Yet, there are no randomized controlled trials to recommend routine use of bicarbonate therapy for DKA. However, rapid and early correction of acidosis with sodium bicarbonate may worsen hypokalemia and cause paradoxical cellular acidosis, and has been correlated with cerebral oedema in children.

We discuss the management of two cases of severe DKA in ICU with

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pH<6.9.The intractable acidosis improved with bicarbonate therapy and both patients recovered without any complication.

CASE REPORT

Case 1

A 10 years old male patient was admitted to our ICU with respiratory distress at the time of admission, he was in altered sensorium and his GCS was E1V1M3.

Since 2 weeks. Past medical history revealed no prior illness. Patient's vitals were BP=100/50 mm of Hg, HR=120/min, RR=36/min with characteristic Kussmaul's respiration, bode temperature was 37.20 Celsius (axillary). Physical examination revealed decreased skin turgor.

Patient was intubated and put on AC mode of ventilator with FiO2=100%, TV=250ml, RR=12/min. laboratory findings were TLC=9000/cu.mm., Blood Glucose=488mg/dl, S.Na+=151mmoL/dl, S.K+=3.15mmoL/dl, S.Cl-=114mmoL/dl, S.Creatinine=1.0mg/dl, urine-analysis showed Glucosuria+++. ABG (Arterial Blood Gas) showed pH=6.76, pCO2=15mm Hg, pO2=367mm Hg, HCO3=2.6, BE=33, sPO2=99. ECG showed sinus Tachycardia.

Primary diagnosis of Diabetic Keto-Acidosis was made, triggered by Respiratory tract infection. Infusion of Normal Saline was started at rate of 20mL/kg/hr for first hour (500mL for first 2 hours rapidly followed by 250mL/hr for the next 4 hours), along with intravenous Insulin (Loading Dose of 3IU followed by 0.1IU/kg/hr) using infusion pump. Insulin was administered after K+ and HCO3replacement therapy, K+ replacement was administered at 20mEq/L for first 4 hours, followed by 5mEq/Hr. HCO3- replacement was started at 100mmol diluted in 500mL of Distilled Water, infused at 50 mmoL/hr and then measured every 6 hours with serial ABG till pH=7 was obtained. Amoxycillin-Clavulunate, Metronidazole and Hydrocostisone were administered for respiratory infection.

Four hours after initiating the therapy, improvement was noted in vitals (BP=110/70mm Hg, HR=90/min, Kussmaul's respiration disappeared) and patient was shifted to SIMV mode of the Ventilator. (FiO2=40%, PSV=12, PEEP=5, HR=90/min).
Six hours after initiating the therapy, ABG analysis started showing recovery. (pH=7.11, pCO2=12mm Hg, HCO3-=3.08mmoL, SBE=24mmoL/hr)
At eight hours of therapy, insulin infusion as reduced to 0.05IU/kg/hr.
On the second day, patients vitals improved further so the patient was gradually weaned off the ventilator and extubated.

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Case 2

A sixteen year old female patient was admitted to ICU with complaints of pain in abdomen, vomiting, loose stools and respiratory distress. The patient was received in Unconscious state, not responding to DPS and her GCS was 3 (E1V1M1). Patient was a known case of Diabetes Mellitus type-1, and was on regular insulin since one year. The patient had missed her insulin doses for 2 days. On receiving, the vitals were as follows: BP=86/40 mm of Hg, HR=164/min, RR=40/min (with characteristic Kussmaul's respiration), body temperature=104 degree F. Patient was intubated and put on AC mode of ventilation. Physical examination revealed signs of dehydration including decreased skin turgor. Laboratory findings revealed Leukocytosis (TLC=19,800/cu.mm.), blood glucose=310mg/dL, S.Na+=158mmol/L, S.K+=3.07mmol/L, S. Creatinine=1.7mg/dL, S.Cl-=1.25mmol/L, urine-analysis=Glucosuria++++. Arterial Blood Gas Analysis (ABG) showed pH=6.9, pCO2=26.5, pO2=262, HCO3-=4.9, Cl-=125, BE=25, O2=99. ECG rhythm was Sinus Tachycardia.

Primary diagnosis of Diabetic Keto-Acidosis was made triggered by missed doses of Insulin and infection. Infusion of Normal Saline was started at rate of 20mL/kg/hr for first hour (2 L for first 2 hours rapidly followed by 500mL/hr for the next 4 hours), but Blood pressure did not improve, so infusion of Noradrenaline at 0.1µg/kg/min was started. Low dose intravenous Insulin at 0.1IU/kg/hr (without loading Dose) using infusion pump. Insulin was administered after K+ and HCO3replacement therapy, K+ replacement was administered at 20mEq/L for first 4 hours, followed by 5mEq/Hr. HCO3- replacement was started at 100mmol diluted in 500mL of Distilled Water, infused at 50 mmoL/hr and then measured every 6 hours with serial ABG till pH=7 was obtained. Amoxycillin-Clavulunate, Metronidazole and Hydrocostisone were administered for respiratory infection.

Table 1: Serial Arterial Blood Gas Analysis of Case 1

	Day-1 08:00 AM	Day-1 02:00 PM	Day-2 08:00 AM	Day-2 02:00 PM	Day-3 08:00 AM	Day-3 02:00 PM
pH (7.35-7.45)	6.76	7.11	7.26	7.25	7.27	7.35
pCO2(32-48)mmHg	15	12.8	30.6	31.2	33.9	38
pO2(83-108)mmHg	147	117	110	108	99	110
S. Na+(136-146)Mmol/L	151	137	142	145	147	145
S. K+(3.4-4.5)Mmol/L	3.30	3.5	2.63	3.5	3.79	4.0
S. Ca2+(4.61-5.17)mg/dL	4.65	4.5	5.0	5.05	4.49	5.15
S. Cl-(98-106)Mmol/L	114	111	104	108	111	112
S. Lactate (0.3-20.0)	1.2	1.4	1.4	1.2	0.9	1.0

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Mmol/L						
HCO3-Mmol/L	2.1	12	13	16	15	18
SBCMmol/L	21.3	16	18.3	17.5	16.6	12.4
ABEMmol/L	28.8	14	11.5	10.4	10	7.6
SBEMmol/L	29.3	14.5	12	11	10.5	8
Anion GapMmol/L	34	21	24	20	21	15
SpO2mmHg	99%	99.4%	99.3%	99%	98%	99.5%

Table 2: Serial Arterial Blood Gas Analysis of Case 2

	Day-1	**Day-1**	**Day-2**	**Day-2**	**Day-3**	**Day-3**
	05:30 PM	**11:50 PM**	**10:00 AM**	**06:00 PM**	**08:00 AM**	**06:00 PM**
pH (7.35-7.45)	6.9	7.13	7.18	7.21	7.35	7.36
pCO2(32-48)mmHg	26.5	11.1	20.1	24	27.3	28
pO2(83-108)mmHg	116	107	105	110	106	118
S. Na+(136-146)Mmol/L	158	125	149	156	159	155
S. K+(3.4-4.5)Mmol/L	3.07	2.6	2.8	3.01	3.14	3.25
S. Ca2+(4.61-5.17) mg/dL	5.25	5.16	5.06	5.08	5.06	5.07
S. Cl-(98-106)Mmol/L	125	123	122	124	122	123
S. Lactate (0.3-20.0) Mmol/L	0.9	1.0	1.0	4.1	4.0	3.0
HCO3-Mmol/L	5.9	3.5	8.0	11.9	15.7	20.0
SBCMmol/L	6.2	7.1	10.1	10.3	16.7	14.5
ABEMmol/L	-27.0	24.9	-19.9	18.0	9.5	8.5
SBEMmol/L	-25.1	24.6	-19.7	18.5	9.8	9.0
Anion GapMmol/L	29.9	33.5	28.7	20.4	16.1	13.6
SpO2mmHg	99.4%	99.4%	99.3%	99.6%	99.5%	99.6%

DISCUSSION

Diabetic Ketoacidosis (DKA) of is one of the life threatening complication of diabetes mellitus which mostly occurs due to noncompliance of insulin regimen or infection. Mortality rate ranges from 0.67% to 7.96%, demonstrating the need of early diagnosis and prompt treatment of DKA.

Pathophysiology:

DKA is a complex metabolic disorder consisting of triad of ketonemia, hyperglycemia and acidemia. Hyperglycemia is the consequence of absolute or relative insulin deficiency

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and increase in counter-regulatory hormones (i.e. glucagon ,catecholamines, growth hormones, epinephrine).Insulin deficiency also leads to lipolysis resulting in elevated fatty acid levels which are metabolized to ketone bodies (namely β-hydroxybutrate,acetone and acetoacetate).

Differential Diagnosis:

Nonketotic hyperosmolar coma was ruled out in this case as there was absence of severe hyperglycemia with ketosis, a blood pH below 7.3,a serum bicarbonate less than 15mEq/l and increased anion gap.
Hyperchloremic metabolic acidosis and acute pancreatitis was distinguished by elevated anion gap in our patient.

Treatment:

Intial fluid resuscitation was done by isotonic saline (20 ml/kg) to restore peripheral infusion. After this intial fluid, it was switched to 0.45% NS to avoid hypernatremia and when serum glucose level reached 250 to 300 mg/dl, it was replaced by 5% dextrose.

Insulin was administered as bolus of 0.1U/kg followed by 0.1U/kg/hr infusion. Blood sugar charting was done every hour in both the patients and infusion was titrated accordingly.
There is no data available regarding the benefits of bicarbonate therapy in DKA as it can lead to dreadful complications like worsening of hypokalemia, intracellular acidosis and cerebral oedema. In a study of 27 patients conducted by Lutterman JA et al comparing those who received iv bicarbonate and those did not, the rate of neurological recovery or mortality was not different.[1]However, the incidence of coma was reported higher in bicarbonate treated patients. Okuda et al illustrated that the alkali administration results in paradoxical increase in acetoacetate levels and delay in improvement of ketosis.[2]

In our cases, even after the replenishment of iv fluids and insulin administration, there was persistence of kussmaul's respiration, metabolic acidosis and hemodynamic instability. After giving the bicarbonate therapy, a clinical response was seen and acidosis also improved slowly in both the patients. Lever and Jaspan reported the benefits of bicarbonate therapy in diabetic ketoacidotic coma and pH below 7.10.[3]

CONCLUSION

Since no randomized controlled trials are available for role of sodium bicarbonate in life threatening diabetic ketoacidosis, its use in it cannot be recommended in all cases. However it can be safely concluded that in severe DKA, it is worthwhile to give intravenous sodium bicarbonate .Our case report and similar case reports by other authors provide a sufficient evidence for it. However, large scale prospective trials need to be done for establishing a recommendation for the same.

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