BICARBONATE DOSING AT DIFFERENT BLOOD PH LEVELS IN METABOLIC ACIDOSIS, ITS RELEVANCE AND OUTCOME- A SINGLE-CENTERED PROSPECTIVE STUDY.

Dr. Mrinal Kanti Taye[a], Dr. Kamal Chandra Deori[a], Dr. Dipak Kumar Sarma[b], Dr. Ali Asgor Ahmed[b]

[a] *Anesthesiology and Critical Care, FAAMCH, Faculty Quarter, Faamch, Barpeta, Assam, Pin-781301.*
[b] *Department of Emergency Medicine, Gauhati Medical College and Hospital Guwahati, Assam, India, Pin 781032.*

Abstract

Background:

Acute acidemia is a frequently observed condition in intensive care units. Intravenous sodium bicarbonate is one possible modality of treatment along with IV fluid and treating the primary cause. Most authors advocate bicarbonate therapy at an arterial pH of ≤ 7.1 but some advocate as early as when the blood pH level starts falling below 7.2. This cut-off level remains a controversy. This study aimed to administer sodium bicarbonate in metabolic acidosis at different blood Ph levels and observe its effects on patients' ABG and outcomes.

Methods:

The target was to maintain arterial pH of 7·20 and above. The comparison was made based on biochemical and clinical outcomes in two different groups where group I with a blood pH. of < 7.2 and Group II with a blood pH of < 7.1.

Results:

In the present study on day 2 pH increased from 7·16 $\pm$ 0.0453 to 7.27 $\pm$ 0.056 in group I and from 6.82 $\pm$ 0.0944 to 6.85 $\pm$ 0.0448 in group II. Hco3 increased from 8.9 $\pm$ 2.82 to 14.8 $\pm$ 3.46 in group I and from 3.9 $\pm$ 3.48 to 4.9 $\pm$ 3.8 in group II. Group I showed better correction of acidosis and bicarbonate level on day 2. Higher bolus dosages are required in group II and still an insignificant rise in bicarbonate or change in pH.

Conclusion:

Preemptive sodium bicarbonate administration at a blood pH of 7.2 and bicarbonate level <15 rather than waiting for pH to fall below 7.1 has shown better primary outcomes in patients with severe metabolic acidemia. Its effects on mortality have not been evaluated. Whether sodium bicarbonate infusion could influence the outcome must be evaluated more in future trials. Additional investigations evaluating therapeutic interventions could be of significance within the cohort of individuals experiencing metabolic acidosis in the ICU.

Keywords: sodium bicarbonate, metabolic acidosis, pH, Submitted: 2023-06-21 Accepted: 2023-06-25

1. Introduction:

Acute acidemia is a frequently observed condition in intensive care units. The administration of intravenous Sodium bicarbonate in metabolic acidosis to improve clinical outcomes in critically ill patients is still controversial. The use of sodium

bicarbonate brings a good outcome in patients with kidney injury and diarrhea. There is a disagreement about administering sodium bicarbonate based on blood pH level. Most authors advocate bicarbonate therapy at an arterial pH of 7.1 and below. But some advocate that bicarbonate administration should be started as early as when the blood pH level starts falling below 7.2[1,2]. This cut-off level remains a controversy. However, treating the primary cause, correction of fluid and electrolytes, and pco2 level along with bicarbonate therapy is the mainstay of treatment. The present study aims to administer sodium bicarbonate in metabolic acidosis at different blood Ph levels and observe its effects on patients' ABG and other outcomes.

2. Methodology:

This single-center, prospective study was conducted in the ICU of the Emergency Medicine Department in a tertiary hospital between April 2019, and March 2020. We enrolled ICU patients with acidemia (pH $\leq$7·2, PaCO2 $\leq$35 mm Hg, and sodium bicarbonate concentration $\leq$15 mmol/L), adult patients between the age of 18-75yrs admitted to ICU within 48hrs of crisis. Sodium bicarbonate was administered based on the pH, PCO2, and HCO3 status of the patients. The target was to maintain arterial pH of 7·20 and above. The comparison was made based on biochemical and clinical outcomes in two different groups where group I is with blood pH of $<$ 7.2 and group II with blood pH of $<$ 7.1. The amount of bicarbonate requirement was calculated by the formula,

HCO3 deficit (mEq) = 0.5 x lean body wt.(kg) x (desired HCO3 - measured HCO3)

Lean body weight is defined as IBW (ideal body weight).

Sodium bicarbonate administration: one ample of 25 ml 8.4% sodium bicarbonate has a concentration of 1mEq/ml. Each ml of solution contains 84.0 mg of sodium bicarbonate which gives 23.0 mg of sodium and 61.0 mg of bicarbonate (1mmol =1 mEq).

Concentration prepared as 50 mEq i.e., two ample of 25ml 8.4 % sodium bicarbonate in 100ml 1/2NS (range is 50 to 150 mEq). The same may be prepared in one liter D5W if hypernatremia persists. This preparation is given as a 100 ml bolus over 15 to 30 min followed by a 20 to 60 ml/hr infusion in a 50 ml syringe pump for the next 24 to 48 hours. Alternatively, two ample 25 ml of 8.4% sodium bicarbonate may be loaded directly into a 50 ml syringe pump and infused in a bigger vein via a central line. If repeat ABG after 1hr does not show the desired level of pH $\geq$7.2 and bicarbonate $\geq$ 15 mEq/L infusions will be continued. [3,5,7]

For example if suppose a patient's ABG shows pH 7.0, PaCO2 26mmHg, PaO2 80, HCO3 6 mmol/L, and Lactate 12mmol/L. we will give 100ml sodium bicarbonate bolus over 1 hr. If ABG done thereafter shows HCO3 8 then the correction factor is 100/ (8-6) =50 ml i.e. 50 ml of sodium bicarbonate will correct the HCO3 status of the patient by 1 mmol/L. Now to reach the desired HCO3 level of 15 from 8 the patient will need [15-8] $\times$50ml =350ml of sodium bicarbonate. So, we need 350 ml more sodium bicarbonate to correct the patient's acidosis i.e., bicarbonate level to 15. This amount can be given as 70 to 100m infusion/hr with hourly ABG monitoring. We used the SOFA score to assess the patient's status [Table 1]

3. Results:

Table **2 revealed** that more men than women developed acidosis among all age groups. People in the age group between 46 to 75 28 (70%) in group I and 32(77.5%) in group II, are more prone to develop acidosis compared to the younger age group of 18-45 which is 12(30%) in group I and 9 (22.5%) in group II. Among patients with acidemia, chronic liver disease is the most common disease developing acidosis in our ICU, a group I- 7 (17.5%) Group II -6(15%)] followed by chronic kidney disease group I- 6 (15%) Group II- 8 (20%) and Diabetes mellitus group I- 4 (10%) Group II- 7 (17.5%) **[Table3].** Physiological support, SOFA score, and CVP monitoring were compared between group I and group II [Table 4]. More patients in group II deteriorated

needing invasive mechanical ventilation-32 (80%) compared to 16(40%) in group I and Vasopressor support 26(65%) in group II compared to 12(30%) in group I. SOFA score recorded on **day 1 and day 2** shows some amount of improvement on day 2 following sodium bicarbonate infusion in group I. SOFA score in group I improves from 6.5±3.3 on day1 to 6.2±3.8 on day 2 whereas in group II patients SOFA score further deteriorated from 9.8±3.5 on day one to 11.8±4.1 on day 2. More patients needed CVP monitoring in group I, 6±4 on the day1, 8±2 on the day2, and in group II 8±4 on the day1 to 12±3 on the day2. The need for CVP monitoring increases despite treatment in both groups. ABG analysis on day 1 and day 2 before and after sodium bicarbonate infusion in group I and group II were compared [Table]. Data were collected and compared between two groups with pH of 7.1≤ (Group II) and pH of 7.2≤ (Group I) based on patient Abg, creatinine, and SOFA score. Parameters like pH and Hco3 in group I have shown significant correction with a p-value < 0.05 between day 1 and day 2. Whereas in group II change in pH, Hco3 and serum creatinine shows insignificant correction between day 1 and day 2 with a p-value >0.05. On day 2 pH increased from 7·16±0.0453 to 7.27±0.056 in group I and from 6.82±0.0944 to 6.85±0.0448 in group II. Hco3 increased from 8.9±2.82 to 14.8±3.46 in group I and from 3.9±3.84 to 4.9±3.8 in group II. Group I showed better performance here.

3.1. ***Statistical analysis:***

36 samples were required in each group with a power of 80% at the significance level of 0.05. Considering an attrition rate of 15%, 40 patients were allocated to each group. The sequence was generated by a statistician. Data were reported as mean±SD, median, and n(%) wherever appropriate. The demographic data were analyzed using the c2 -test and the Wilcoxon-Mann-Whitney test was used to analyze non-normally distributed and heterogeneous data. The count data were analyzed by using Pearson's $\chi 2$ test and Fisher's exact test. A P-value<0.05 was considered statistically significant. The other statistical analyses were conducted in the SPSS 17.0 software package.

4. Discussion:

The benefit of administering sodium bicarbonate at an early stage of falling blood pH just below 7.2 rather than waiting to fall as low as 7.1 was tried to observe in this study. Most authorities would give bicarbonate to a patient with an arterial pH <7.1, but this is not a hard and fast rule. Even though administration of sodium bicarbonate is said to aggravate intracellular acidosis it can be well prevented by proper fluid balance and aggressive monitoring [3].

We are trying to compare the effect of sodium bicarbonate infusion on critically ill patients with metabolic acidemia between two groups where patients with blood pH ≤7·20 in ABG were made in group I and patients with blood pH ≤7·10 were made in group II. The target was to reach a Ph of $\geq$ 7.20 along with a bicarbonate level of ≥15 in both groups. Parameters used to compare between two groups were SOFA score, ABG, creatinine, and need for physiological support in terms of invasive mechanical ventilation and Vasopressor support. [6,7]

There are various methods of sodium bicarbonate infusion. We prepared sodium bicarbonate concentration as 50 mEq sodium bicarbonate per 100ml 1/2NS and gave 100ml bolus over 15 to 30 min followed by 20 to 60 ml/hr infusion in 50 ml syringe pump for next 24 to 48 hours. Alternatively, two 25 ml ample of 8.4% sodium bicarbonate(1meq/ml) may be loaded directly in a 50 ml syringe pump infused via a central line or in a bigger vein. Koda Kimble et al replaced 50% over 3 to 4 hours and the remainder over 24 hours. [4,7,8] Once the pH is 7.2 - 7.25, the serum [HCO3-] should not be increased by more than 4 to 8 mEq/L over 6 to 12 hours to avoid the risks of over-alkalinization. Serpa et al say the continuous infusion of bicarbonate is the most frequently selected method of administration and pH > 7.30 was the preferred target while monitoring with arterial blood gas analysis every 2 hours until the target is reached and then every 4 hours. [5,9,10]

The present study was to assess whether early and bolus sodium bicarbonate infusion to treat acidosis has any advantage. Here early means pH at 7.2 rather than waiting for pH to fall below 7.1 while monitoring the pH of a critically ill patient and bolus means 100 ml of sodium bicarbonate (1: 2 ratio) over 15 to 30 min in either group. Arterial blood gases analysis was part of the routine investigations and sodium bicarbonate infusion was titrated to reach and maintain a bicarbonate level of 15/mEq/lit. [11]. The present study showed that after treating the primary cause there is an easier correction of Hco3, RFT derangement, and lesser requirement of vasopressors and mechanical ventilator support in group I compared to group II. In the present study on day2 pH increased from 7·16 ±0.0453 to 7.27±0.056 in group I and from 6.82±0.0944 to 6.85 ±0.0448 in group II. Hco3 increased from 8.9±2.82 to 14.8±3.46 in group I and from 3.9±3.48 to 4.9 ± 3.8 in group II along with this PCO2 also increases in both the groups because Hco3 converts to Co2 and decrease in alveolar ventilation. Group, I showed significant improvement in pH and bicarbonate levels on day 2. Group II showed insignificant correction in bicarbonate and pH despite administering higher bolus doses of bicarbonate. As a portion of the HCO3 which was initially distributed in the ECF space subsequently enters the intracellular space so blood HCO3- concentration doesn't rise effectively in the beginning. More metabolic acidosis will lead to greater increases in bicarbonate volume of distribution, so larger amounts of sodium bicarbonate should have to be administered initially. It is important to correct the underlying cause in case of DKA, lactic acidosis, septic shock, cardiac arrest, intraoperative metabolic acidosis, etc. along with IV administration of bicarbonate to maintain a blood pH >7.2 to 7.25. [12,13,14.15]. We did not stratify patients according to causes of acidemia as the trial was designed to observe the effects of iv sodium bicarbonate irrespective of causes of acidemia. The causes of acidemia were heterogeneous and in our study, sepsis was the most common cause. It could be speculated in our study that delayed therapy waiting till pH fell below 7.10 or not administrating sodium bicarbonate in patients with pH 7.2 and Hco3 $\leq$ 15mEq/lit was associated with more refractory cardiovascular instability, kidney injury, or need for mechanical ventilation in critically patient. [16,17,18.19]

Sodium bicarbonate might also be counterproductive in terms of myocardial contractility, CCF, and metabolic side effects such as hypernatremia, hypocalcemia, hypokalemia, metabolic alkalosis, systemic vasodilatation, tissue perfusion, cellular function, pulmonary oedema, intracellular acidosis, etc. although none of them seen to be life-threatening if related to sodium bicarbonate infusion. [20,21,22]

5. Limitations:

The limitation of our study was that no specific intravenous fluid and its amount were recommended, or the role of mechanical ventilation was not studied in either group. There is a significant role of intravenous fluid and mechanical ventilation in the acid-base balance. Another limitation was we used 8.4% sodium bicarbonate 100ml bolus infusion over 15 to 30 min in either group. Here we did not use the conventional formula for calculating the base deficit and rate of sodium bicarbonate infusion. We did not evaluate which mode of administration would have a better outcome. Despite these limitations, our trial represents strengths to encourage a new and generalized design of treatment in critically ill patients presenting with severe metabolic acidemia with organ failure. Here early sodium bicarbonate treatment at pH 7.2 with a bicarbonate level $<$ 15 rather than at pH 7.1 have been observed to have a better effect on the primary outcome. However, its effects on mortality have not been evaluated.

6. Conclusion:

Sodium bicarbonate can be administered preempt at a blood pH of 7.2 and at a bicarbonate level of $\leq$15 with the same justification that stands for a blood pH of 7.1. Waiting for blood pH to fall below 7.1 is found to be detrimental to

patients in many instances. It has shown better primary outcomes with early administration even though its effects on mortality have not been evaluated. Over and above sodium bicarbonate administration based on blood pH and bicarbonate level, treating the primary cause is the gold standard. Whether sodium bicarbonate infusion with different amounts and speeds could influence the outcome remains to be determined and should be evaluated more in future trials.

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Table 1: The sofa score: adrenergic agent (in mcg/kg/min). FiO2=fractional inspired oxygen concentration. PCV= packed cell volume, SOFA=Sequential Organ Failure Assessment. SOFA includes sub scores ranging from 0 to 4 for each of six components. Aggregated scores range from 0 to 24, with higher scores indicating more severe organ failure.

Organ System, Measurement	SOFA Score				
	0	1	2	3	4
Respiration PaO2/FiO2 mmHg	Normal	<400	<300	<200(with respiratory support)	<100(with respiratory support)
Coagulation Platelets x103/mm3	Normal	<150	<100	<50	<20
Liver Bilirubin, mg/dL (µmol/l)	Normal	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12.0 (<204)
Cardiovascular Hypotension	Normal	MAP<70 mmHg	Dopamine <5 or dobutamine (any dose)	Dopamine >5 or epinephrine <0.1 or norepinephrine <0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
Central Nervous System Glasgow Coma Score	Normal	13-14	10-12	6-9	<6
Renal Creatinine, mg/dL (µmol/l)or Urine output	Normal	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440) Or<500mL/day	>5.0 (>440) or<200mL/day

Table2. patients' demographic profile

Age (years)	Group I (n=40)	group II (n=40)
18-45	12(30%)	9 (22.5%)
46-75	28 (70%)	32(77.5%)
Sex		
Men	29 (72.5%)	31(77.5%)
Women	11(27.5%)	9(22.5%)

Table 3: Types of patients with causes of acidaemia

Types of patients	No of patient	
	GroupI(n=40)	GroupII (n=40)
Chronic liver disease	7(17.5%)	6(15%)
Pancreatitis	3(7.5%)	5(12.5)
Diabetes mellitus	4 (10%)	7(17.5%)
Cerebrovascular accident	4(10%)	3(7.5%)
Chronic heart disease	3(7.5%)	4(10%)
Chronic kidney disease	6(15%)	8 (20%)
COPD	6(15%)	4 (10%)
post operative	4(10%)	2(5%)
Road traffic accident	3(7.5%)	1(2.5%)
Source of admission		
Medical	33 (82.5%)	37(92.5%)
Surgical	7(17.5%)	3 (7.5%)
Main cause of metabolic acidosis		
Sepsis	32 (80%)	29 (72.5%)
Others	8(20%)	11(27.5%)

Table 4. Physiological support, SOFA score and cvp monitoring in group I and group II.

Physiological support				
Invasive mechanical ventilation	16 (40%)	32 (80%)		
Vasopressor support	12 (30%)	26(65%)		
SOFA score on admission (Average ±SD)				
	Group I		Group II	
	Day1	Day2	Day1	Day2
Respiratory	1.3± 1.2	1.2 ±1.3	2.2 ±1.5	2.7 ±1.3
Haematological	0.4± 0.8	0.5 ±0.9	0.5± 1.0	0.8 ±1.1
Hepatic	0.4 ±0.7	0.4 ±0.7	0.6 ±1.0	0.7± 1.0
Cardiovascular	2.2 ±1.3	2.1 ±1.4	3.0± 1.3	3.3 ±1.2
Neurologic	0.7± 1.1	0.9 ±1.2	1.5 ±1.6	2.5 ±1.5
Renal	1.4 ±1.2	1.1 ±1.2	2.0 ±1.5	1.9± 1.5
Total	6.5 ±3.3	6.2± 3.8	9.8 ±3.5	11.8 ±4.1
CVP monitoring	6 ±4	8±2	8±4	12±3

Table 5: ABG analysis on day1 and day2 before and after sodibicarb infusion in group I and group II

ABG	Group I Average with SD		P value	Group II Average with SD		P value
	Day1	Day2		Day1	Day2	
pH.	7·16 ±0.0453	7.27 ±0.056	.0001	6.82 ±0.0944	6.85 ±0.0448	0.229 7
Po2	75±14.16	110±11.0		58±7.62	80±6.78	
Pco2	25±6.67	37±7.61		18±9.61	30±6.89	
Hco3	8.9±2.82	14.8±3.46	.0001	3.9±3.84	4.9±3.8	0.245
Base deficit	-11.6±4.6	-9.4±3.8		-30.6±3.21	-21.4±6.82	
Serum lactate	3·4 ±0.34	2.1 ±0.18		6.5±0.26	4.4 ±0.14	
PaO2-to-FiO2 ratio (mm Hg)	235±28	278±41		138±33	112±34	
Serum creatinine (mg/dL)	2·46± 0,02	1.8±0.26	.0001	5.64±0.15	6.2±0.2	.0927
PCV	45±7	30±6		46±12	36±18	

Author biography

Dr. Mrinal Kanti Taye Associate Professor, Anesthesiology and Critical Care, FAAMCH, Faculty Quarter, Faamch, Barpeta, Assam, Pin-781301

Dr. Kamal Chandra Deori Assistant Professor, Anesthesiology and Critical Care, FAAMCH, Faculty Quarter no 8, Faamch, Barpeta, Assam, Pin-781301.

Dr. Dipak Kumar Sarma Professor of Surgery & HOD, Department of Emergency Medicine, Gauhati Medical College and Hospital Guwahati, Assam, India, Pin 781032.

Dr. Ali Asgor Ahmed PGT. Emergency Medicine, Department of Emergency Medicine, Guwahati Medical College and Hospital, Guwahati, Assam, India, Pin 781032.