Review Article

Review on the Use of Sodium Bicarbonate

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ABSTRACT

Sodium bicarbonate is a commonly used electrolyte supplement, urinary alkalinizer and a serum alkalizing agent given orally or intravenous. The usage of sodium bicarbonate can be a daunting riddle in many clinical scenarios and its appropriate usage needs to be defined. The appropriateness of sodium bicarbonate in non-severe metabolic acidosis needs to be defined. The situation where its use leads to harmful effects in places where it is otherwise indicated needs to be acknowledged. The recent study showing its use in severe AKI patients throws a light on its expanding use.

Keywords: Sodium bicarbonate, acidosis, non – severe metabolic acidosis, sodium bicarbonate indications.

INTRODUCTION

Sodium bicarbonate can be categorized as an alkalising agent, antacid and an electrolyte supplement (both oral and parenteral). They are widely used in adults with cardiac arrest (in patients with prolonged cardiac arrest after adequate alveolar ventilation has been established and effective cardiac compression), metabolic acidosis in patients with chronic kidney disease (oral use, off label), metabolic acidosis, hyperkalemia, renal tubular acidosis, urine alkalisation, an antacid in the prevention of contrast induced nephropathy (off label use).[1]

Each sodium bicarbonate tablet of 650mg contain 7.7mEq each of sodium and bicarbonate (1 mEq $NaHCO_3$ is equivalent to 84mg of $NaHCO_3$). Sodium bicarbonate is used in both oral and parenteral forms. They are administered orally in patients with metabolic acidosis with CKD, renal tubular acidosis, as an antacid and as a urinary alkaniser (except in cases of specific overdoses where they are given I.V). Intravenous usage is usually reserved for patients with hyperkalemia, metabolic acidosis and in cardiac arrest.[2] They are given as an infusion for the prevention of contrast induced nephropathy and in metabolic acidosis. For infusion, they should be made into a solution having a concentration of 154mEq/L sodium bicarbonate in 5% dextrose in water for the prevention of CIN. The solution is given 1 hour prior to contrast administration at a dose of 3ml/kg and subsequently 1ml/kg/hr is infused during and 6 hours post procedure.[3] When given orally, 2-3 tablets of 650mg per day is usually given for patients with metabolic acidosis in CKD (which is titrated based on serum bicarbonate concentration). In patients where they are used as a urinary alkalinizer an initial 6 tablets is given followed by 1-3 tablets every 4 hours. Not more than 25 tablets per day should be given for patients less than 60 years of age and in patients above 60 years the total daily dose should be capped at 12 tablets.[1]

USE OF SODIUM BICARBONATE IN METABOLIC ACIDOSIS

Sodium bicarbonate use in metabolic acidosis has been a topic of varying opinion.[4] Most clinicians treat patients with severe metabolic acidosis (pH < 7.1) with sodium bicarbonate. This use has been supported by the fact that worsening acidosis leads invariably to detrimental effects such as arrhythmias, reduced ventricular contractility, arterial vasodilation and venoconstriction and blunted response to catecholamine vasopressors.[5] The beneficial effects of increasing the arterial pH to circumvent the aforementioned adverse events are especially useful in situations such as cardiac arrest and CKD.[6, 7]

Sodium bicarbonate is generally warranted only for severe acidemia with acute metabolic acidosis (arterial pH < 7.1 and serum bicarbonate less than 6mEq/L or less). If the bicarbonate levels are more than 6mEq/L, then PCO_2 should be assessed for respiratory acidosis and mechanical ventilation should be initiated otherwise bicarbonate use will worsen the respiratory acidosis.[8] The formulae used for calculating $NaHCO_3$ dose in metabolic acidosis is given by the equation:

$$HCO_3^-\ (mEq) = 0.5 * \text{weight (Kg)} * [24 - \text{serum } HCO_3^-\ (mEq/L)]$$

Potential harmful effects of Sodium bicarbonate use

The beneficial effects of sodium bicarbonate is offset by its potential harmful effects which leads to controversies in its usage. Rapid infusion of sodium bicarbonate to treat metabolic acidosis can cause increase in arterial and tissue capillary CO_2, which may impede the rise in pH and subsequently lead to overcorrection of acidaemia.[9] This is due to the fact that bicarbonate is converted into carbonic acid after combining with H^+ ions which is subsequently

degraded to CO_2 and H_2O.[10] But in order for this reaction to take place effectively, proper ventilation and tissue perfusion needs to be established otherwise carbonic acid and carbon dioxide will accumulate and this will eventually lead to a rise in pCO_2 and a drop in arterial pH. Administering sodium bicarbonate can also induce a "paradoxical" reduction in pH of the CSF. The mechanism for this is two factors; one is due to the aforementioned rise in pCO_2 and the second aspect is the reduced drive for hyperventilation due to systemic rise in pH which causes an increased pCO_2 (which will cause increased pCO_2 in the CSF) coupled with relatively reduced supply of bicarbonate to the brain. This imbalance of bicarbonate supply to the brain and pCO_2 increase results in rise in pCO_2 with reduced pH leading to neurological deterioration.[11] Reduction in ionised calcium which leads to adverse cardiac outcomes coupled with bicarbonate's extracellular volume expanding potential makes it a risky choice in cardiac failure / cardiac insufficiency, this being substantiated by the fact that a 50ml of a 1mEq/mL hypertonic solution of $NaHCO_3$ raises the sodium level by 1mEq/L and the extracellular volume by 250mL in a subject of 70kg.[12] Along with these factors the lactate enhancing effects of $NaHCO_3$ also needs to be acknowledged.[13]

New evidence for sodium bicarbonate use in patients with severe AKI

Traditionally sodium bicarbonate was not utilised for patients with arterial pH between 7.1-7.2 (less severe acidaemia). A recent randomized controlled trial conducted in France throws the light on the potential use of bicarbonate in patients with less severe metabolic acidosis. The patients with less severe acidemia and a sequential organ failure assessment score of greater than or equal to 4 or an arterial lactate concentration of 2 mmol/L or more were randomized to receive either 4.2% I.V infusion of sodium bicarbonate or no sodium bicarbonate in a total study population of 389 critically ill patients. The mortality at 28 days has no statistical significant difference between the control group and the bicarbonate group (46% vs 55%) or neither did organ failure at 7 days (62% vs. 69%). But a statistically significant benefit was observed in patients with severe AKI and moreover the bicarbonate arm showed an improved overall outcome. In patients with severe AKI, sodium bicarbonate therapy reduced the 28 day mortality (46 vs. 63 percent) and the need to do dialysis (51 vs. 73 percent).[14]

CONCLUSION

Although the indication for usage of sodium bicarbonate is clear cut in most situations, its utilization in patients with less severe acidosis is non-conclusive.[14] Although a positive trend in overall outcome was observed in patients receiving it, the evidence is not strong enough to warrant its use in their population. But sodium bicarbonate use may be recommended in patients with severe AKI who present with an arterial pH of 7.1-7.2 and a serum bicarbonate value less than or equal to 20mmol/L and a $PaCO_2$ less than or equal to 45mmHg.

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