

Survey Report

Efficacy of Sodium Bicarbonate in Hyperkalemia-Induced Cardiac Arrest: A Retrospective Analysis

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Abstract:

Hyperkalemia is a critical, reversible cause of cardiac arrest requiring immediate intervention. Sodium bicarbonate is widely used in emergency settings to manage metabolic acidosis and promote intracellular potassium shifting. However, its efficacy in improving survival rates in hyperkalemia-induced cardiac arrest remains a subject of debate. This retrospective study analyzes data from 100 patients to assess the impact of sodium bicarbonate administration on resuscitation outcomes, electrolyte correction, and overall survival.

Keywords: Hyperkalemia, reversible cause, cardiac arrest, Sodium bicarbonate

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Introduction

Hyperkalemia is a medical emergency that can precipitate life-threatening arrhythmias and cardiac arrest.

- ECG Changes in Hyperkalemia and Associated Arrhythmias
- Hyperkalemia significantly affects cardiac conduction, leading to characteristic electrocardiogram (ECG) changes as potassium levels rise. These progressive changes include:
 - Mild Hyperkalemia (5.5–6.5 mmol/L): Peaked T waves with narrow bases, especially in precordial leads.
 - Moderate Hyperkalemia (6.5–7.5 mmol/L): Prolonged PR interval, decreased P wave amplitude, and widening of the QRS complex.
 - Severe Hyperkalemia (>7.5 mmol/L): Absent P waves, further QRS widening, sine-wave pattern, leading to ventricular fibrillation or asystole.
- Types of Arrhythmias Due to Hyperkalemia.
- Bradyarrhythmias: Sinus bradycardia, atrioventricular (AV) blocks.
- Tachyarrhythmias: Ventricular tachycardia (VT), ventricular fibrillation (VF).
- Conduction Abnormalities: Bundle branch blocks, sine-wave pattern leading to cardiac arrest.

Early recognition of these ECG changes is crucial in preventing fatal arrhythmias and guiding timely intervention. Common causes include chronic kidney disease, severe metabolic acidosis, and certain medications such as potassium-sparing diuretics. Traditional management strategies focus on immediate

stabilization using calcium gluconate, insulin-dextrose therapy, and sodium bicarbonate. However, recent studies have questioned the efficacy of sodium bicarbonate in improving patient survival.

Aims & Objectives

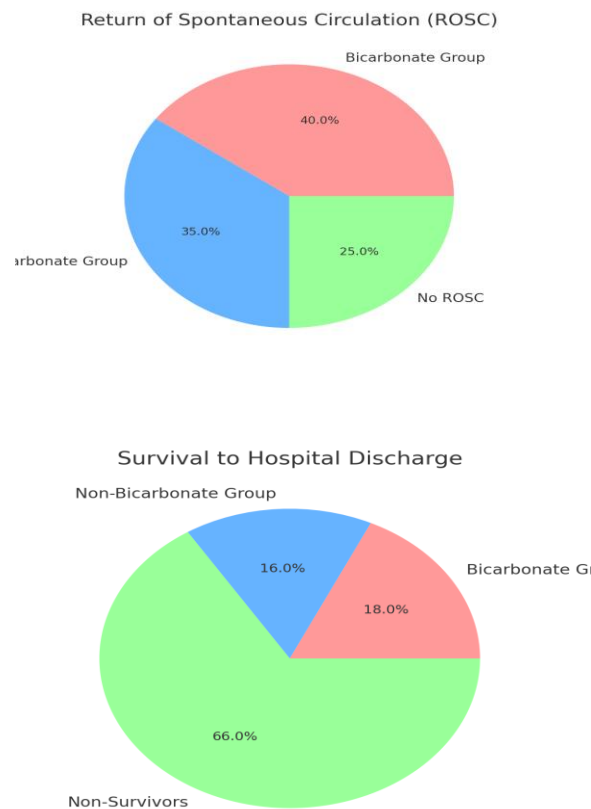
1. To evaluate the efficacy of sodium bicarbonate in improving resuscitation outcomes in hyperkalemia-induced cardiac arrest.
2. To assess the impact of sodium bicarbonate on serum potassium levels post-administration.
3. To determine the survival rates and return of spontaneous circulation (ROSC) in patients receiving sodium bicarbonate.
4. To compare sodium bicarbonate therapy with alternative standard treatments for hyperkalemia.

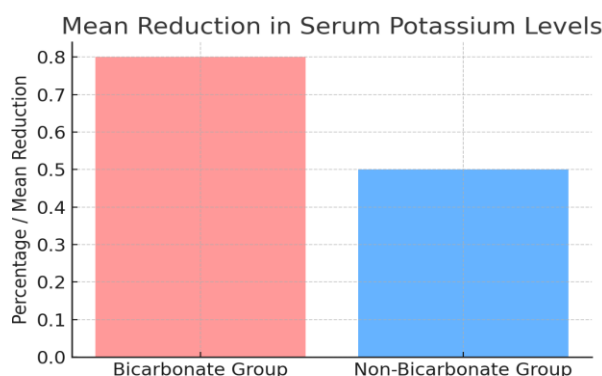
Methodology

This retrospective study was conducted at KIMS Amalapuram, analyzing medical records of 100 patients who experienced hyperkalemia-induced cardiac arrest between 2018 and 2023. Patients were divided into two groups: those who received sodium bicarbonate as part of resuscitation and those who did not. Key outcome measures included return of spontaneous circulation (ROSC), serum potassium reduction post-intervention, and survival to hospital discharge.

Results

Out of 100 patients, 55 received sodium bicarbonate during cardiac arrest management, while 45 did not. The return of spontaneous circulation (ROSC) was observed in 40% of patients in the bicarbonate group compared to 35% in the non-bicarbonate group. However, survival to hospital discharge remained similar in both groups (18% vs. 16%). Post-treatment serum potassium levels showed a mean reduction of 0.8 mmol/L in the bicarbonate group versus 0.5 mmol/L in the control group.





Discussion

The findings indicate that while sodium bicarbonate may facilitate temporary reductions in serum potassium levels, its impact on long-term survival outcomes remains minimal. The marginal improvement in ROSC rates suggests that sodium bicarbonate may play a role in early resuscitation, but it should not replace standard hyperkalemia treatments like insulin-dextrose and calcium gluconate. Additionally, excessive bicarbonate administration can lead to paradoxical intracellular acidosis, potentially worsening cardiac instability.

Conclusion

Sodium bicarbonate remains a widely used intervention in hyperkalemia-induced cardiac arrest, yet its efficacy in improving survival to hospital discharge remains questionable. While it may aid in transient potassium reduction, other first-line treatments should be prioritized. Future randomized controlled trials are necessary to determine its definitive role in cardiac arrest resuscitation.

References:

1. Weisberg LS. Management of severe hyperkalemia. *Crit Care Med.* 2008;36(12):3246-3251.
2. Kim H, Lee J, Kim HJ. The effects of sodium bicarbonate administration in hyperkalemic cardiac arrest: A systematic review. *Am J Emerg Med.* 2019;37(4):696-702.
3. Kraut JA, Madias NE. Metabolic acidosis and the role of sodium bicarbonate in chronic kidney disease. *Kidney Int.* 2018;93(4):787-796.
4. Weisberg LS. Management of severe hyperkalemia. *Crit Care Med.* 2008;36(12):3246-3251.
5. Kim H, Lee J, Kim HJ. The effects of sodium bicarbonate administration in hyperkalemic cardiac arrest: A systematic review. *Am J Emerg Med.* 2019;37(4):696-702.
5. Kraut JA, Madias NE. Metabolic acidosis and the role of sodium bicarbonate in chronic kidney disease. *Kidney Int.* 2018;93(4):787-796.
6. Montague BT, Ouellette JR, Buller GK. Retrospective review of the frequency of hyperkalemia in patients with congestive heart failure and its treatment. *J Clin Hypertens (Greenwich).* 2008;10(8):578-583.
7. Palmer BF. Managing hyperkalemia caused by inhibitors of the renin-angiotensin-aldosterone system. *N Engl J Med.* 2004;351(6):585-592.
8. Acker CG, Johnson JP, Palevsky PM, Greenberg A. Hyperkalemia in hospitalized patients: Causes, adequacy of treatment, and results of an attempt to improve physician compliance with published therapy guidelines. *Arch Intern Med.* 1998;158(8):917-924.
9. Mahoney BA, Smith WAD, Lo DS, Tsoi K, Tonelli M, Clase CM. Emergency interventions for hyperkalemia. *Cochrane Database Syst Rev.* 2005;(2):CD003235.
10. Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. *Am J Emerg Med.* 2000;18(6):721-729.
