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The Generation and Maintenance of Metabolic Alkalosis in Intensive Care Unit

Kreethi Suresh kumar[*1], Sivaprakash Vijayarajan , Aasini Maria Georgina[3], Varshini Baskaran[4]

[1]Kreethi Suresh kumar, Lecturer, Department of Critical care, SRM Medical College Hospital and Research Centre, Kattankulathur.

[2]Sivaprakash Vijayarajan, Assistant Professor & RSO, Department of Radio-Diagnosis, Panimalar College of Allied Health Sciences, PMCH&RI, Chennai.

[3]Dr. Aasini Maria Georgina, Lecturer, Panimalar College of Allied Health Sciences, Chennai

[4]Varshini Baskaran, Lecturer, Department of Medical Laboratory Technology, Panimalar College of Allied Health Sciences, Chennai

Corresponding Author:

Kreethi Suresh kumar,

Lecturer, Department of Critical care, SRM Medical College Hospital and Research Centre, Kattankulathur,

Email ID: Kreethukreethi77@gmail.com

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ABSTRACT

Background: Metabolic alkalosis is a common acid-base disorder in critically ill patients and can contribute to adverse outcomes if not managed appropriately. Understanding its etiology and optimal treatment strategies in the intensive care unit (ICU) is crucial for improving patient care and reducing complications.

Objective: This study aimed to identify the underlying causes of metabolic alkalosis in ICU patients and evaluate the effectiveness of different management approaches in improving patient outcomes.

Materials and Methods: A prospective analysis was conducted in the Critical Care Medicine Department to assess metabolic alkalosis in ICU patients. Data were collected on patient demographics, clinical symptoms, and arterial blood gas (ABG) parameters, including pH, bicarbonate levels, and electrolyte concentrations. The study explored contributing factors such as diuretic use and persistent vomiting. Laboratory investigations, including ABG analysis, serum electrolytes, and renal function tests, were performed to establish correlations between these factors and the development of metabolic alkalosis.

Results: The study included 80 patients, with 61.25% being male and 38.75% female. The most common causes of metabolic alkalosis were diuretic therapy (48.75%) and hemodynamic instability (51.25%). Management primarily involved normal saline administration, with potassium chloride supplementation provided to 50% of hypokalemic patients. Adjustments to diuretic doses were made to prevent further organ dysfunction. ABG monitoring over three days revealed significant improvements in pH ($p < 0.001$) and bicarbonate levels ($p < 0.001$).

Conclusion: Effective management of metabolic alkalosis in ICU patients requires a structured approach, including the regulation of diuretic therapy and fluid resuscitation with normal saline. Addressing electrolyte imbalances, particularly hypokalemia and hypochloremia, is essential for patient stabilization and recovery. The successful transfer of 75% of patients to the general ward suggests that appropriate interventions can reduce ICU stays and associated healthcare costs. Implementing these strategies in clinical practice may enhance patient outcomes and optimize resource utilization in ICU settings.

Keywords: *Metabolic Alkalosis, pH Imbalance, Bicarbonate Excess, Electrolyte Disturbance, Hypokalemia*

1. INTRODUCTION

Metabolic alkalosis is an acid-base disorder marked by plasma bicarbonate (HCO_3^-) >26 mmol/L and arterial pH >7.43. It often coexists with hypokalemia and results from hydrogen ion loss via the GI tract (e.g., vomiting), renal excretion (e.g., diuretics), or excess alkali intake. Persistence is due to impaired renal bicarbonate excretion, influenced by hypovolemia,

chloride depletion, hypokalemia, hyperaldosteronism, or renal failure. Assessment of volume status, urinary chloride, and plasma renin and aldosterone levels aids diagnosis and treatment planning. (1) Metabolic alkalosis is the most common acid-base disorder in critically ill patients. Though acidosis is well-studied, alkalosis has gained recognition for its clinical impact. Severe cases are associated with higher morbidity and mortality, with about 50% of general surgical patients developing postoperative metabolic alkalosis. (2) It arises from alkali accumulation or acid loss, often leading to dehydration due to extracellular fluid (ECF) contraction from chloride loss. (3)

Studies emphasize bicarbonate excess, hydrogen ion loss, and renal dysfunction. Emmett et al. (2020) stressed correcting electrolyte imbalances, (4) while Tinawi et al. (2021) classified alkalosis into chloride-sensitive and chloride-resistant types. (5) Vasquez & Soleimani (2022) detailed renal mechanisms, (6) and Berg et al. (2021) linked CFTR deficiency to poor bicarbonate excretion. (7) Park & Sidebotham (2023) highlighted its frequent coexistence with mixed acid-base disorders. (8)

Pathophysiology includes intracellular hydrogen shifts, renal and GI hydrogen loss, bicarbonate retention, and contraction alkalosis. (3) Normally, excess bicarbonate is excreted in urine, but this is impaired by hypovolemia, low GFR, chloride depletion, or hyperaldosteronism. (9) Causes include vomiting, nasogastric suction, hypokalemia, distal tubule dysfunction, and genetic disorders like Bartter and Gitelman syndromes, as well as diuretics. (10)

Classification is based on ECF status. A urinary chloride (UCl^-) <20 mmol/L suggests ECF contraction, commonly from vomiting, NG suction, villous adenoma, congenital chloridorrhea, ileostomy output, post-hypercapnia, diuretics, CF with sweating, or tubulopathies (Bartter, Gitelman). ECF expansion alkalosis is linked to conditions like primary aldosteronism, renal artery stenosis, renin-secreting tumors, (11) Cushing's, CAH, licorice ingestion, or Liddle syndrome.

Physiological effects include hypovolemia, hypokalemia, and chloride loss, resulting in arrhythmias, reduced myocardial contractility, confusion, neuromuscular symptoms, and impaired oxygen delivery. Respiratory compensation via hypoventilation raises arterial CO_2, which may cause hypoxia.

Treatment targets the cause. Chloride-responsive alkalosis (UCl^- <20 mmol/L) is managed with saline to restore volume, improve GFR, and aid bicarbonate excretion. Potassium repletion corrects hypokalemia and restores renal function. Chloride-resistant alkalosis (UCl^- >20 mmol/L), due to mineralocorticoid excess, is treated by addressing the cause—e.g., surgery or mineralocorticoid receptor blockers like spironolactone, eplerenone, or amiloride. (12)

Prompt diagnosis and targeted management are vital to prevent complications, reduce ICU stay, and improve outcomes. Understanding metabolic alkalosis helps optimize therapy and lower healthcare costs.

2. METHODS

This prospective observational study was conducted in the Department of Critical Care Medicine at Saveetha Medical College and Hospital to investigate the etiology and maintenance mechanisms of metabolic alkalosis in the intensive care unit (ICU). The study population included 80 patients admitted to the ICU with an arterial blood gas (ABG) diagnosis of metabolic alkalosis. Inclusion criteria comprised patients above 18 years who remained in the ICU for more than 24 hours. Patients below 18 years, those diagnosed with metabolic acidosis, or those with respiratory alkalosis were excluded.

Data collection focused on patient demographics, clinical presentations, vital parameters (heart rate, blood pressure, SpO_2, respiratory rate), and laboratory investigations, including ABG analysis and key electrolyte levels (Na^+, K^+, Ca^{2+}, HCO_3^-). Management strategies, including fluid administration, electrolyte replacement, and necessary respiratory support adjustments, were documented. Statistical analysis was performed using SPSS software to identify correlations between specific diagnoses, ABG values, and patient outcomes.

The primary objectives of this study were to determine the causes of metabolic alkalosis in ICU patients and to analyze its impact on patient outcomes.

3. RESULTS

A sample of 80 patients was investigated, to assess the incidence and causes of metabolic alkalosis in ICU.

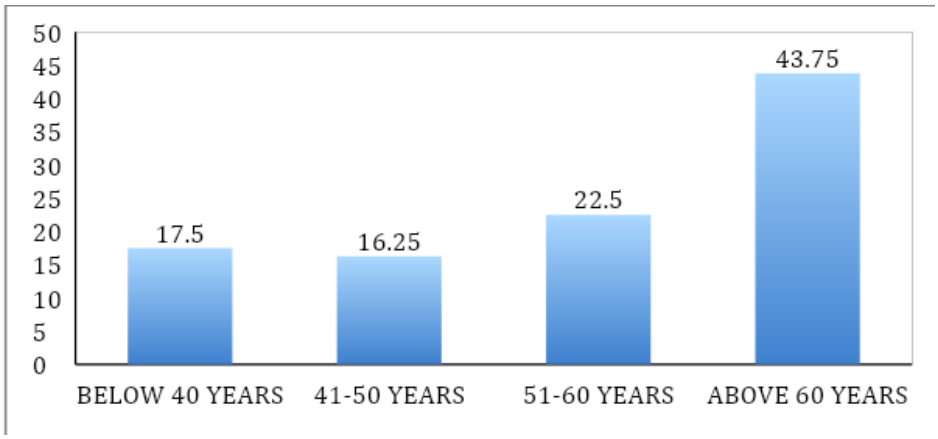

Figure:1 Age distribution of the patient

According to Figure 1, the age distribution of patients shows that 17.5% were below 40 years, 16.25% were between 41-50 years, 22.5% were aged 51-60 years, and the majority, 43.75%, were above 60 years. Among them, 61.25% were male, while 38.75% were female. In terms of comorbidities, 32.5% (26 individuals) had both Type 2 Diabetes Mellitus (T2DM) and Systemic Hypertension (SHTN), while 18.75% (15 individuals) had SHTN alone. Additionally, 23.75% (19 individuals) had T2DM alone, and 25% (20 individuals) did not exhibit either of these comorbidities.

Causes of metabolic alkalosis	**Number of patients**	**percentage**
Hypovolemic	12	15
RTA to Hypovolemic	4	5
Hypokalemic	18	22.5
Vomiting	4	5
Cystic fybrosis	3	3.75
Diuretic use	39	48.75
Total	**80**	**100**

Table 1: Causes of metabolic alkalosis

Table 1 shows the various etiologies contributing to metabolic alkalosis among ICU patients. The most common cause identified was diuretic use, affecting 48.75% of patients. Hypokalemia was observed in 22.5% of cases, while hypovolemic states accounted for 15%. Vomiting and road traffic accidents each contributed to 5% of cases, whereas cystic fibrosis was the least common cause, affecting 3.75% of patients. These findings highlight the diverse underlying factors responsible for metabolic alkalosis in critically ill patients.

Furthermore, potassium (K^+) correction was necessary for 50% (40 patients), while the other half did not require correction. Similarly, diuretic intervention with Inj. Lasix was administered to 48.75% (39 patients), whereas 51.25% (41 patients) did not require diuretics. These findings indicate a nearly balanced need for potassium correction and diuretic therapy within the study population, emphasizing the diverse etiological factors contributing to metabolic alkalosis in critically ill patients.

Values of Ph	**Before ABG**	**After ABG**			**Before ABG (in percentage %)**	**After ABG (in percentage%)**
		Day-1	**Day-2**	**Day-3**		

						Day-1	Day-2	Day-3
7.35-7.45	2	19	37	57	2.5	23.75	46.25	71.25
7.45-7.50	65	53	40	23	81.25	66.25	50	28.75
7.50-7.55	11	8	3	_	13.75	10	3.75	_
>7.55	2	_	_	_	2.5	_	_	_
Total	80	80			100	100		

Table 2: Comparison with pH values of before ABG and after ABG

Table 2 shows the changes in pH levels before and after arterial blood gas (ABG) analysis over three days. Initially, only 2.5% of patients had a pH within the normal range (7.35-7.45), but this increased significantly to 71.25% by Day 3. Conversely, the majority of patients (81.25%) initially had a pH between 7.45-7.50, which gradually decreased to 28.75% by Day 3. Similarly, higher pH ranges (7.50-7.55 and above 7.55) saw a steady decline over time. These findings indicate an overall improvement in pH balance with treatment.

Pair	Mean Difference	Std. Deviation	Std. Error Mean	95% CI (Lower - Upper)	t-value	df	p-value (Sig. 2-tailed)
pH Before ABG - pH After ABG Day 1	0.01853	0.03543	0.00396	0.01064 - 0.02641	4.677	79	<0.001
pH Before ABG - pH After ABG Day 2	0.04143	0.03551	0.00397	0.03352 - 0.04933	10.434	79	<0.001
pH Before ABG - pH After ABG Day 3	0.06344	0.04481	0.00501	0.05347 - 0.07341	12.663	79	<0.001
pH After ABG Day 1 - pH After ABG Day 2	0.02290	0.03408	0.00381	0.01532 - 0.03048	6.011	79	<0.001
pH After ABG Day 1 - pH After ABG Day 3	0.04491	0.04107	0.00459	0.03577 - 0.05405	9.782	79	<0.001

pH After ABG Day 2 - pH After ABG Day 3	0.02201	0.03547	0.00397	0.01412 - 0.02991	5.550	79	<0.001

Table 3: Paired t-Test Analysis of pH Values Before and After ABG Correction

The results show statistically significant differences ($p < 0.001$) in pH values before and after ABG analysis across all time points, indicating a meaningful improvement in acid-base balance over time.

Values of HCO3	**Before ABG**	**After ABG**			**Before ABG (in percentage %)**	**After ABG (in percentage%)**		
		Day-1	**Day-2**	**Day-3**		**Day-1**	**Day-2**	**Day-3**
>35	3	1	_	_	3.75	1.25	_	_
35-30	26	8	1	_	32.5	10	1.25	_
30-25	51	71	64	35	63.75	88.75	80	43.75
<25	_	_	15	45	_	_	18.75	56.25
Total	80	80			100	100		

Table 4: Comparison with HCO3 values of before ABG and after ABG

Table 4, shows changes in bicarbonate (HCO_3^-) levels before and after arterial blood gas (ABG) analysis. HCO_3^- levels above 35 decreased from 3.75% to 1.25% after Day 1. Levels between 35-30 dropped from 32.5% to 10% on Day 1 and 1.25% on Day 2. In contrast, levels between 30-25 increased from 63.75% to 88.75% on Day 1, then declined to 80% on Day 2 and 43.75% on Day 3. No patients had HCO_3^- below 25 initially, but 18.75% reached this range by Day 2 and 56.25% by Day 3, indicating metabolic alkalosis correction.

Pair Comparison	**Mean Difference**	**Std. Deviation**	**Std. Error Mean**	**95% CI (Lower - Upper)**	**t-value**	**df**	**p-value**
HCO_3^- Before ABG – HCO_3^- After ABG Day 1	1.7413	1.3093	0.1464	1.4499 – 2.0326	11.895	79	<0.0001
HCO_3^- Before ABG – HCO_3^- After ABG Day 2	3.3426	1.6940	0.1894	2.9657 – 3.7196	17.649	79	<0.0001

HCO_3^- Before ABG – HCO_3^- After ABG Day 3	5.0751	1.9136	0.2139	4.6493 – 5.5010	23.722	79	<0.0001
HCO_3^- After ABG Day 1 – HCO_3^- After ABG Day 2	1.6014	1.1778	0.1317	1.3393 – 1.8635	12.161	79	<0.0001
HCO_3^- After ABG Day 1 – HCO_3^- After ABG Day 3	3.3339	1.6820	0.1880	2.9596 – 3.7082	17.729	79	<0.0001
HCO_3^- After ABG Day 2 – HCO_3^- After ABG Day 3	1.7325	1.3724	0.1534	1.4271 – 2.0379	11.291	79	<0.0001

Table 5: Paired T-Test for Bicarbonate (HCO_3^-) Levels Before and After ABG

This table presents the paired t-test results for bicarbonate (HCO_3^-) levels before and after ABG, indicating a significant decrease over time ($p < 0.0001$).

From Table 6, it is observed that 75% of patients were transferred to the ward,20% of patients left against medical advice (AMA) and 5% of patients were declared.

HOSPITAL DISCHARGE	**NO. OF RESPONDANTS**	**PERCENTAGE**
WARD	60	75
AGAINTS MEDIACL ADVICE	16	20
DEATH	4	5
TOTAL	**80**	**100%**

Table 6: Outcome of the participants

4. DISCUSSION

Metabolic alkalosis in critically ill patients presents with diverse etiologies and significant clinical challenges, as demonstrated by our study and the case studies by Swagata Tripathy et al. and McCauley et al. While our study focused on a larger cohort of ICU patients, the case reports provided in-depth insights into severe individual presentations. A common finding across all studies was the prevalence of underlying conditions contributing to metabolic alkalosis. In our cohort, systemic hypertension and type 2 diabetes mellitus were frequently associated comorbidities, while Tripathy et al. and McCauley et al. highlighted conditions such as chronic kidney disease, diuretic abuse, and gastric acid loss from pyloric obstruction.

The etiology also varied, with diuretic use being the most common cause in our study (48.75%), aligning with Tripathy et al.'s findings, (13) where long-term diuretic abuse played a key role. However, McCauley et al. identified pyloric obstruction as a primary cause, emphasizing the role of prolonged gastric acid loss. (14) Electrolyte imbalances, particularly hypokalemia and hypochloremia, were consistently reported, with all studies documenting compensatory hypoventilation, metabolic alkalemia, and risks of severe complications such as seizures, arrhythmias, and neurological deterioration.

Management strategies across studies involved fluid resuscitation and electrolyte correction. Our study observed significant improvements in arterial blood gas parameters over three days with saline administration and diuretic dose adjustments. Similarly, Tripathy et al. reported resolution of alkalemia with fluid and electrolyte replacement in some cases, though severe cases required additional interventions like acetazolamide or dialysis. In contrast, McCauley et al.'s patient required ICU admission and ventilation due to hypercarbic respiratory failure.

Outcomes varied, with our study reporting a favorable response in 75% of patients but a mortality rate of 5%, reflecting the condition's severity. Tripathy et al. documented a fatal outcome due to intracranial hemorrhage, while McCauley et al.'s case required prolonged intensive care. These findings underscore the need for early diagnosis, individualized treatment, and continuous monitoring to improve prognosis in metabolic alkalosis. Further research is essential to refine treatment protocols and enhance patient outcomes.

5. CONCLUSION

Metabolic alkalosis remains a commonly encountered and clinically significant challenge in critical care settings. In this study, diuretic use and electrolyte disturbances such as hypokalemia were leading contributors. Effective treatment, including volume repletion with isotonic saline and careful correction of electrolyte imbalances, led to substantial improvement in patient outcomes. Adjusting or temporarily discontinuing diuretics proved beneficial in many cases, helping to prevent adverse effects on vital organs including the kidneys, heart, and brain. Notably, about 75% of the patients showed enough clinical improvement to be shifted from the ICU to general wards, indicating successful management. This not only reflects better recovery rates but also suggests a potential reduction in ICU duration and overall healthcare expenditure. These findings highlight the importance of early detection, personalized treatment strategies, and ongoing monitoring to improve patient stability and outcomes in metabolic alkalosis.

Conflict of Interest: According to the authors, there are no conflicts of interest.

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