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DEVELOPMENT OF METABOLIC ALKALOSIS LEADING TO COMPENSATORY HYPERCAPNIA IN HOSPITALIZED COPD PATIENTS INITIALLY ADMITTED WITH RESPIRATORY ACIDOSIS: A CASE SERIES

Pulmonary Medicine

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KEYWORDS

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is frequently complicated by acute respiratory acidosis during exacerbations requiring hospitalization. Non-invasive ventilation (NIV), particularly BiPAP, is a cornerstone of management. However, during hospitalization, some patients develop metabolic alkalosis, which can impair respiratory compensation and reduce the efficacy of ventilatory support.

This case series presents 20 COPD patients admitted with respiratory acidosis who developed metabolic alkalosis during hospitalization leading to compensatory hypercapnia, primarily due to diuretic therapy, dehydration, or other factors. The subsequent compensatory hypercapnia reduced the efficacy of BiPAP. The cases illustrate that identification and management of metabolic alkalosis is essential for successful respiratory support and recovery.

Aims And Objectives

- **Aim:**

To evaluate the impact of metabolic alkalosis on the respiratory management of COPD patients initially admitted with respiratory acidosis.

- **Objectives:**

1. To identify the prevalence and causes of metabolic alkalosis in hospitalized COPD patients.
2. To assess changes in serial arterial blood gas (ABG) parameters, particularly pH, pCO_2, and HCO_3- levels.
3. To evaluate the effectiveness of BiPAP therapy alone in the context of metabolic alkalosis with compensatory hypercarbia.
4. To discuss the implications of acid-base disturbances on patient outcomes and management strategies.

MATERIALS AND METHODS

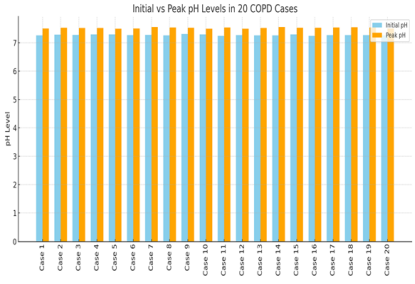
This case series included 20 hospitalized COPD patients who initially presented with respiratory acidosis. Patient data were collected prospectively, including initial and serial arterial blood gas parameters and peak levels during hospitalization. Variables examined included initial and peak pCO_2, pH, and HCO_3- values, along with the effectiveness of BiPAP therapy alone and identified causes of metabolic alkalosis (e.g., diuretics, dehydration, vomiting, electrolyte loss).

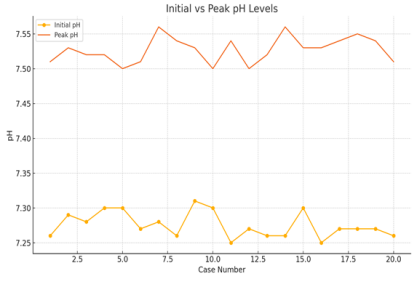
Data analysis included descriptive statistics (mean, median, range, standard deviation) and graphical representations to identify trends and correlations among variables.

Summary Table Of 20 Cases

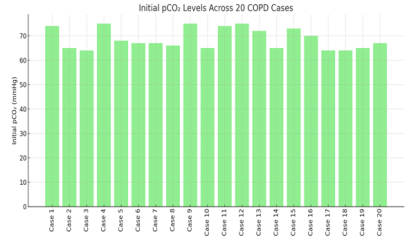
Case	Initial pCO_2 (mmHg)	Initial pH	Peak HCO_3-- (mEq/L)	Peak pH	BiPAP Effective	Cause of Alkalosis
Case 1	74	7.26	35	7.51	No	Diuretics
Case 2	65	7.29	42	7.53	No	Dehydration
Case 3	64	7.28	43	7.52	No	Dehydration
Case 4	75	7.3	36	7.52	No	Diuretics
Case 5	68	7.3	41	7.5	No	Vomiting
Case 6	67	7.27	36	7.51	No	Vomiting
Case 7	67	7.28	43	7.56	No	Dehydration
Case 8	66	7.26	39	7.54	No	Electrolyte Loss
Case 9	75	7.31	44	7.53	No	Dehydration
Case 10	65	7.3	40	7.5	Yes	Vomiting
Case 11	74	7.25	44	7.54	No	Dehydration
Case 12	75	7.27	38	7.5	Yes	Dehydration
Case 13	72	7.26	36	7.52	Yes	Diuretics
Case 14	65	7.26	35	7.56	No	Electrolyte Loss
Case 15	73	7.3	38	7.53	No	Dehydration
Case 16	70	7.25	39	7.53	No	Electrolyte Loss
Case 17	64	7.27	36	7.54	No	Vomiting
Case 18	64	7.27	38	7.55	No	Dehydration
Case 19	65	7.27	36	7.54	No	Diuretics
Case 20	67	7.26	41	7.51	No	Vomiting

Graphical Analysis

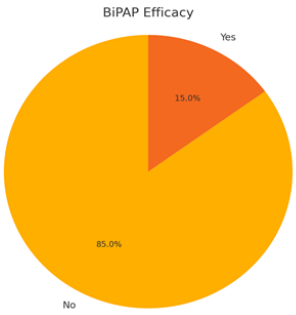




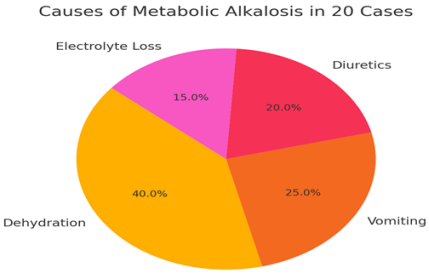
Initial Vs Peak pH Levels



Initial pCO_2 Levels Across Cases



BiPAP Efficacy



Distribution of Causes of Metabolic Alkalosis

Descriptive Statistics

Parameter	Mean	Median	Min	Max	SD
Initial pCO_2 (mmHg)	68.1	67	64	75	3.93
Initial pH	7.275	7.27	7.25	7.31	0.02
Peak pH	7.526	7.53	7.50	7.56	0.02
Peak HCO_3-- (mEq/L)	38.9	38.5	35	44	3.11

RESULTS

- **Initial ABG Findings:**
- **Mean initial pCO_2:** 68.1 mmHg
- **Mean initial pH:** 7.275

- **Peak ABG Findings:**
- **Mean peak pH:** 7.526
- **Mean peak HCO_3-:** 38.9 mEq/L

- **Common Causes of Metabolic Alkalosis:**
- Dehydration (8 patients)
- Vomiting (5 patients)
- Diuretic use (4 patients)
- Electrolyte loss (3 patients)

- **BiPAP Efficacy:**
- Ineffective in 17 cases due to ongoing alkalosis and compensatory hypercapnia
- Only 3 cases showed effectiveness of BiPAP

- **Graphical Analysis:**
- **Trends in rising pH and HCO_3-** associated with worsening hypercapnia
- High pH correlated with reduced BiPAP efficacy

DISCUSSION

This case series underscores a common but often overlooked progression in hospitalized COPD patients—namely, the shift from respiratory acidosis to metabolic alkalosis is multifactorial. Factors such as diuretic therapy, vomiting, and dehydration contribute to this metabolic disturbance. Elevated bicarbonate levels increase blood pH, which can suppress respiratory drive and lead to compensatory hypercapnia.

The findings highlight the limitation of BiPAP when metabolic alkalosis is not concurrently managed. Even with BiPAP support, patients continued to experience elevated pCO_2 levels until metabolic causes were addressed. This emphasizes the need for a holistic approach to acid-base management in COPD care, where respiratory support must be accompanied by correction of underlying metabolic imbalances.

CONCLUSION

In hospitalized COPD patients, vigilant monitoring of acid-base balance is essential. The emergence of metabolic alkalosis can blunt respiratory compensation, leading to persistent or worsening hypercapnia despite ventilatory support. Early detection and management of metabolic alkalosis are crucial for optimizing outcomes and ensuring the effectiveness of interventions such as BiPAP.