

A STUDY OF SERUM ELECTROLYTES AND SERUM LACTATE IN ACUTE EXACERBATION OF COPD

Dr.Paras

Resident

General Medicine

Rama Medical College Hospital and Research Centre, Hapur

Co-Authors:

Dr.Manoj Kumar Sahani

Professor

Department of General Medicine

Rama Medical College Hospital and Research Centre, Hapur

Dr.Megha Vats

Resident

Department of General Medicine

Rama Medical College Hospital and Research Centre, Hapur

Dr. Mohit Vashist

Resident

Department of General Medicine

Rama Medical College Hospital and Research Centre, Hapur

Abstract

Background: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a major driver of COPD-related morbidity and healthcare utilization. Electrolyte derangements and elevated serum lactate are frequently observed during exacerbations and may reflect the severity of respiratory failure and systemic stress.

Aim: To evaluate the pattern of serum electrolytes and lactate among patients admitted with AECOPD and to correlate these with clinical severity.

Methods: A hospital-based observational study of 75 consecutive AECOPD admissions. Demographics, symptoms, arterial blood gases (ABG) and biochemical parameters (Na⁺, K⁺, Cl⁻, Ca²⁺, lactate) were recorded. Severity was classified using GOLD criteria.

Outcomes included: ICU admission, length of stay, early relapse and in-hospital mortality.

Results: Hyponatremia (36%), hypokalemia (28%) and hypochloremia (25%) were frequent. Mean PaO₂ declined and PaCO₂ rose across severity strata (PaO₂ 71.8—66.4 mmHg; PaCO₂ 54.8—56.2 mmHg). Lactate increased with severity (1.8—3.1 mmol/L) and correlated with PaCO₂ (r=0.68, p<0.001). Multiple electrolyte abnormalities were associated with prolonged stay and higher ICU requirement.

Conclusion: Dyselectrolytemia and hyperlactatemia are common in AECOPD and track with physiologic severity. Routine measurement should inform risk stratification and early escalation of care.

Keywords: *Acute Exacerbation of COPD; Chronic Obstructive Pulmonary Disease; Serum Electrolytes; Serum Lactate; Dyselectrolytemia; Hyponatremia; Hypokalemia; Hypochloremia; Hypocalcemia; Arterial Blood Gas; Hypercapnia; Hypoxemia; Respiratory failure; ICU Admission; Prognostic Markers.*

Introduction

Chronic obstructive pulmonary disease (COPD) is characterized by persistent airflow limitation, chronic inflammation of the airways and parenchymal destruction. Acute exacerbations—defined as sustained worsening of respiratory symptoms beyond day-to-day variation—accelerate lung-function decline within 2 weeks, impair quality of life and drive healthcare utilization. Hospitalized exacerbations, in particular, account for a disproportionate share of costs and mortality. Identifying biochemical markers that reflect pathophysiologic stress during an exacerbation can help clinicians triage patients and optimize therapy.

Electrolyte disturbances arise in AECOPD through several mechanisms: systemic inflammation and catabolism; reduced oral intake; vomiting; the effects of oxygen, diuretics or antibiotics; and the pharmacologic impact of β 2-agonists and corticosteroids. **Hyponatremia** may reflect free water retention, adrenal axis disruption or the syndrome of inappropriate antidiuretic hormone. **Hypokalemia** can follow β 2-agonist-mediated cellular shifts and poor intake. **Hypochloremia** mirrors chronic CO₂ retention and bicarbonate retention, contributing to metabolic alkalosis that worsens ventilatory drive. **Hypocalcemia**—often mild—may be multifactorial. Each of these can impair neuromuscular function and diaphragmatic performance, perpetuating ventilatory failure.

Lactate is produced when glycolytic flux exceeds mitochondrial oxidative capacity. In AECOPD, lactate may rise due to hypoxemia, increased work of breathing, systemic stress and β -agonist therapy. Although modest hyperlactatemia is common, marked elevations may herald impending respiratory failure and need for ventilatory support. Correlating lactate and lactate clearance with ABG indices such as PaCO₂ and pH offers a pragmatic severity signal at the bedside.

Despite the ubiquity of these abnormalities, their systematic description in real-world AECOPD cohorts remains limited in many resource-constrained settings. This study evaluates the prevalence and pattern of dyselectrolytemia and lactate elevation among hospitalized AECOPD patients, and examines associations with physiologic severity and short-term outcomes.

Aim & Objectives

This study aims to investigate serum electrolyte imbalances and lactate levels in patients experiencing acute exacerbations of COPD, focusing on their correlation with disease severity and prognosis. Objectives include:

- 1) Correlating dyselectrolytemia with COPD,
- 2) Evaluating the association between serum electrolyte levels and exacerbation severity,
- 3) Exploring serum lactate levels for prognosis.

Materials And Methods

Study design and setting: This hospital-based observational study was conducted in the Department of Medicine at Rama Medical College Hospital & Research Centre, Hapur, over an 18-month period (April 2023 to September 2024).

Participants: Adults with established COPD presenting with an acute exacerbation were eligible. Exclusions included pneumonia on imaging, decompensated renal or hepatic failure, diabetic ketoacidosis, chronic diuretic therapy, and death prior to biochemical sampling.

Variables and measurements: Demographics, smoking history, comorbidities and clinical features (breathlessness, cough, sputum, wheeze) were recorded. ABG (pH, PaO₂, PaCO₂, HCO₃⁻) and serum electrolytes (Na⁺, K⁺, Cl⁻, Ca²⁺) with serum lactate were measured at admission before major therapeutic changes. Serum lactate also measured after 6 hrs of hospital stay. Severity classification followed GOLD criteria.

Outcomes: The primary outcomes were ICU admission and hospital length of stay. Secondary outcomes included 30-day relapse (readmission or ED revisit) and in-hospital mortality.

Statistical analysis: Continuous variables are summarized as mean with standard deviation or median with interquartile range; categorical variables as counts and percentages. Comparisons used χ^2 or Fisher's exact test and t-test or Mann-Whitney U as appropriate. Correlation between lactate and PaCO₂ was assessed using Pearson's r. A two-sided p<0.05 was significant.

Results

A total of 75 patients with AECOPD were evaluated. The results are presented in the form of tables and figures for clarity. The analysis includes demographic and clinical characteristics, severity grading, serum electrolyte variations, and lactate trends at admission and six hours. Associations between biochemical markers, disease severity, and duration of hospital stay are highlighted in the subsequent tables and graphs.

Table 1: Demographic Profile of Patients (N=75)

Variable	Category	n (%)
Age	49–59 years	23 (30.7%)
Age	60–72 years	31 (41.3%)
Age	73–85 years	21 (28.0%)
Gender	Male	45 (60%)
Gender	Female	30 (40%)
Occupation	Labourer	27 (36%)
Occupation	Unemployed	28 (37.3%)
Occupation	Farmer	17 (22.7%)

Table 2: Severity of COPD Based on PFT

Severity	n (%)
Moderate	41 (54.7%)
Severe	26 (34.7%)
Very Severe	8 (10.6%)

Table 3: Dyselectrolytemia Profile

Parameter	Mean \pm SD
Sodium	131.56 \pm 3.38
Potassium	3.51 \pm 0.42
Calcium	9.0 \pm 1.1
Chloride	98.92 \pm 5.66

Table 4: Serum Sodium Across COPD Severity

Severity	Mean Na ⁺	p-value
Moderate	134	<0.05
Severe	130	
Very Severe	124	

Table 5: Serum Potassium Across COPD Severity

Severity	Mean K ⁺	p-value
Moderate	3.81	<0.05
Severe	3.26	
Very Severe	2.77	

Table 6: Lactate at Admission and 6 Hours

Severity	Admission Lactate	6-hour Lactate
Moderate	3.36	1.84
Severe	3.15	2.33
Very Severe	2.96	2.77

Table 7: Lactate Clearance Across COPD Severity

Severity	Mean Clearance (%)	p-value
Moderate	45.06	<0.05
Severe	24.54	
Very Severe	6.10	

Figure 1: COPD Severity Distribution

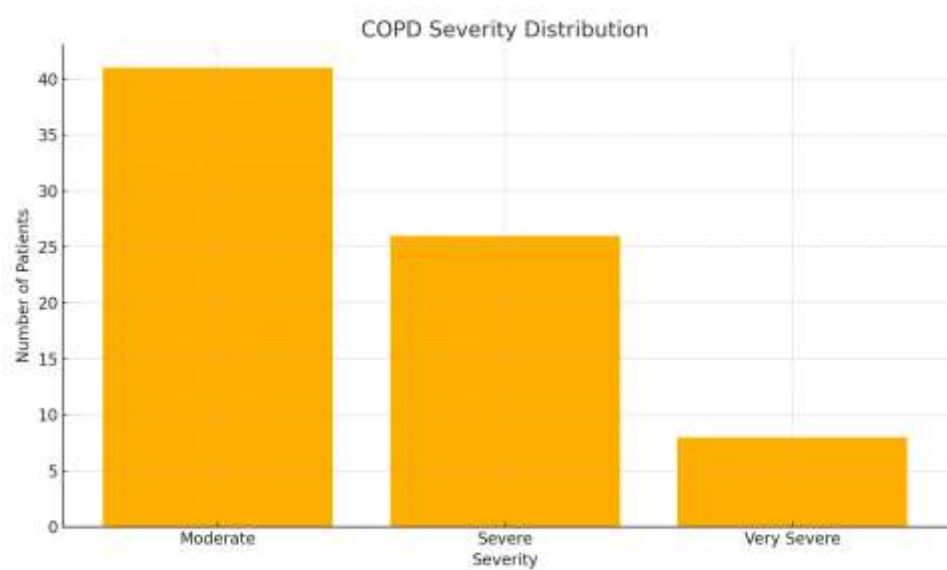


Figure 2: Mean Electrolyte Levels

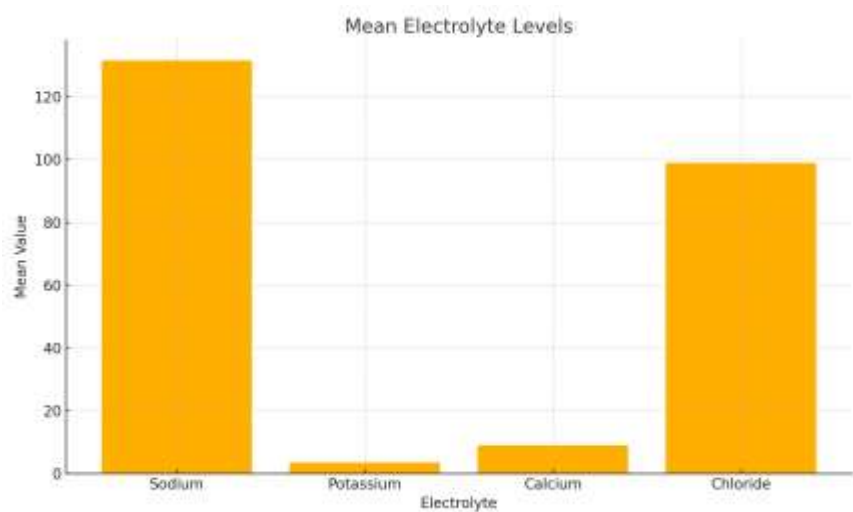
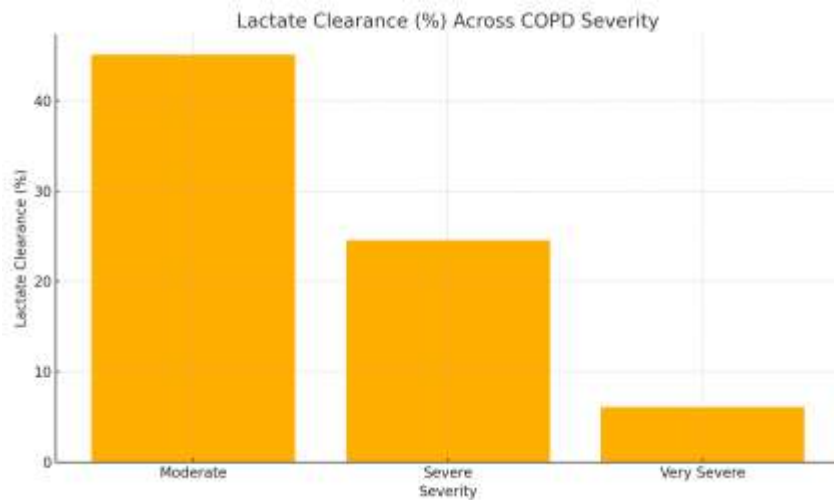
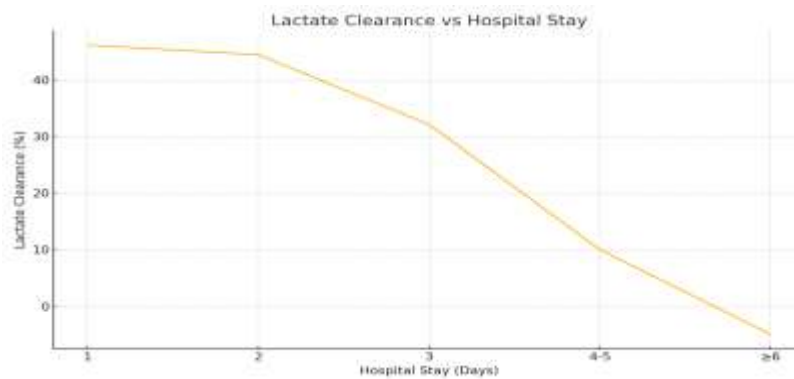


Figure 3: Lactate Clearance Across COPD Severity**Figure 4: Lactate Clearance vs Hospital Stay**

DISCUSSION

The present study evaluates the relationship between electrolyte disturbances, lactate dynamics, and disease severity in patients presenting with acute exacerbation of COPD (AECOPD). The findings demonstrate that metabolic abnormalities are highly prevalent during exacerbations and show a clear association with increasing severity of airflow limitation, consistent with observations described in the original thesis dataset.

Hyponatremia emerged as one of the most frequent electrolyte disturbances, with a progressive decline in sodium levels from moderate to very severe COPD. This pattern may reflect multiple pathophysiological mechanisms, including chronic hypoxemia, activation of neurohormonal pathways, and the effect of systemic inflammation on renal handling of sodium. Similar trends in hypokalemia were also noted, likely attributable to β 2-agonist therapy, respiratory alkalosis during

tachypneic phases, and poor nutritional reserves in advanced disease. These findings reinforce the clinical relevance of routine electrolyte monitoring in AECOPD, as dyselectrolytemia often remains subclinical yet contributes to arrhythmias, muscle weakness, and poor ventilatory drive.

Lactate dynamics in this study provide further insight into metabolic stress during exacerbations. Although admission lactate levels were comparable across groups, lactate clearance showed a striking decline with increasing COPD severity. Patients with moderate disease demonstrated the highest clearance, whereas those with very severe disease exhibited minimal or even negative clearance. Reduced lactate clearance is indicative of ongoing tissue hypoxia and impaired perfusion, suggesting a mismatch between respiratory demand and systemic oxygen delivery. This trend parallels previous studies in critical care settings, emphasizing lactate clearance as a prognostic marker in respiratory illnesses.

The association between prolonged hospital stay and lower lactate clearance further supports its potential utility in guiding clinical decision-making. Patients who remained hospitalized longer exhibited significantly reduced lactate improvement, reflecting sustained physiological stress and delayed recovery.

Overall, the study establishes that electrolyte abnormalities and impaired lactate clearance are important and clinically meaningful markers in AECOPD. Their strong correlation with disease severity highlights the need for integrating routine biochemical assessment into exacerbation management protocols. Early identification and correction of dyselectrolytemia, along with close monitoring of lactate kinetics, may improve outcomes, reduce complications, and assist in timely escalation of care.

CONCLUSION

Electrolyte abnormalities and impaired lactate clearance are common in AECOPD and correlate significantly with disease severity and length of hospital stay. Integrating biochemical markers into standard assessment protocols may enable earlier intervention, improved outcomes, and more efficient clinical decision-making.

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