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Study Of Lactate Levels In Stable COPD Indian Patients.

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ABSTRACT

Chronic Obstructive Pulmonary Disease (COPD) is associated with impaired exercise tolerance and altered skeletal muscle metabolism. Lactate, a marker of anaerobic metabolism, may provide insight into functional limitation in stable COPD patients. This observational cross-sectional study included 50 stable COPD patients aged 40–65 years diagnosed by spirometry. After obtaining ethical approval and informed consent, baseline clinical evaluation was performed. Blood lactate levels were measured at rest and immediately after the 6MWT. Data were analyzed using appropriate statistical methods, including paired t-test and Pearson correlation coefficient. The mean resting lactate level was 1.82 ± 0.45 mmol/L, which increased significantly to 3.10 ± 0.68 mmol/L post-6MWT ($p < 0.001$). A weak but statistically significant positive correlation was observed between COPD stage and change in lactate levels ($r = 0.292$, $p < 0.05$). Stable COPD patients demonstrate significant lactate elevation following submaximal exercise, indicating early anaerobic metabolism. Lactate levels may serve as a useful biomarker for assessing functional capacity and disease severity.

Keywords: COPD, Lactate Levels, Six-Minute Walk Test

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation, chronic inflammation, and systemic manifestations. It represents a significant public health burden in India due to high prevalence of risk factors such as tobacco smoking, biomass fuel exposure, and environmental pollution [1]. Beyond pulmonary impairment, COPD is increasingly recognized as a systemic disease affecting skeletal muscle metabolism, exercise tolerance, and overall functional capacity [2, 3]. Lactate, a byproduct of anaerobic metabolism, serves as an important biomarker reflecting tissue hypoxia and metabolic stress. In patients with stable COPD, altered oxygen delivery, impaired oxidative capacity of skeletal muscles, and early onset of anaerobic metabolism during exertion may lead to elevated lactate levels even in the absence of acute exacerbation. This metabolic shift contributes to exercise intolerance and dyspnea, which are hallmark features of COPD [4-6].

The assessment of lactate levels, particularly in relation to functional tests such as the six-minute walk test (6MWT), provides valuable insights into the physiological limitations and disease severity in stable COPD patients. In the Indian population, where demographic, environmental, and socioeconomic factors differ significantly, studying lactate dynamics may help in better understanding disease behavior and tailoring rehabilitation strategies [7]. This study aims to evaluate lactate levels in stable COPD patients and explore their correlation with functional capacity and disease severity.

STUDY METHODOLOGY

This observational cross-sectional study was conducted on 50 patients diagnosed with stable Chronic Obstructive Pulmonary Disease (COPD) based on clinical evaluation and spirometry findings. The study was carried out after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants. Patients aged between 40 and 65 years were included, while those with acute exacerbations, significant cardiovascular instability, or contraindications to exercise testing were excluded.

A detailed medical history was obtained, and thorough clinical examination was performed for all participants. Baseline parameters including age, gender, smoking status, and spirometric indices were recorded. Resting blood lactate levels were measured using standard laboratory methods. All patients then underwent the six-minute walk test (6MWT) according to standardized guidelines, and post-exercise lactate levels were recorded immediately after completion of the test.

The collected data were compiled and analyzed using appropriate statistical methods. Quantitative variables were expressed as mean and standard deviation, while qualitative variables were expressed as percentages. Correlation between lactate levels and COPD severity, as well as functional capacity, was assessed using Pearson correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic And Clinical Profile Of Study Participants (N = 50)

Variable	Category	Frequency (n)	Percentage (%)
Age Group (years)	40–50	18	36%
	51–60	22	44%
	61–65	10	20%
Gender	Male	34	68%
	Female	16	32%
Smoking Status	Smoker	30	60%
	Non-smoker	20	40%
COPD Severity (GOLD)	Mild (Stage I)	8	16%
	Moderate (Stage II)	20	40%
	Severe (Stage III)	15	30%
	Very Severe (IV)	7	14%

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Table 2: Comparison Of Lactate Levels Before And After 6mwt (N = 50)

Parameter	Mean ± SD (mmol/L)	Test Used	p-value
Resting Lactate	1.82 ± 0.45		
Post-6MWT Lactate	3.10 ± 0.68	Paired t-test	< 0.001*
Mean Difference	1.28 ± 0.52		

*Statistically significant

Table 3: Correlation Between COPD Stage And Change In Lactate Levels

Variable Compared	Pearson r	r^2	p-value	Interpretation
COPD Stage vs Δ Lactate (Post–Pre)	0.292	0.0854	< 0.05*	Weak positive correlation

*Statistically significant

DISCUSSION

The present study evaluated lactate dynamics in stable COPD patients and its correlation with disease severity and functional capacity, particularly following the six-minute walk test (6MWT). The findings demonstrated a statistically significant rise in lactate levels after exertion, along with a weak but significant positive correlation between COPD stage and change in lactate levels. These observations provide important insights into the metabolic and functional limitations associated with COPD in a stable clinical state [8, 9].

In this study, the majority of patients belonged to the 51–60 years age group with a male predominance, which is consistent with the epidemiological profile of COPD in India. The higher proportion of smokers further supports the well-established role of tobacco exposure as a primary risk factor. The distribution of disease severity showed that most patients were in moderate to severe stages, reflecting the tendency of COPD to remain underdiagnosed until symptomatic progression occurs. This demographic and clinical pattern aligns with previous Indian and global studies on COPD [10].

A key finding of the study was the significant increase in lactate levels following the 6MWT. This indicates an early shift toward anaerobic metabolism even during submaximal exercise in stable COPD patients. Under normal physiological conditions, aerobic metabolism predominates during low to moderate intensity exercise; however, in COPD, impaired pulmonary function leads to reduced oxygen delivery to peripheral tissues. Additionally, skeletal muscle dysfunction, characterized by reduced oxidative enzyme activity and mitochondrial abnormalities, further contributes to inefficient energy utilization. As a result, lactate production increases even with minimal exertion, leading to early fatigue and exercise intolerance.

The observed mean rise in lactate levels after the 6MWT highlights the role of lactate as a marker of metabolic stress and reduced exercise capacity. This finding is in agreement with earlier studies which have reported elevated lactate levels in COPD patients during exercise testing. The increase in lactate is also associated with increased ventilatory demand due to lactic acidosis, which further exacerbates dyspnea—a hallmark symptom of COPD. Thus, lactate accumulation not only reflects metabolic impairment but also contributes to the clinical manifestations of the disease.

Another important observation was the weak but statistically significant positive correlation between COPD severity and change in lactate levels. Although the correlation coefficient was modest ($r = 0.292$), it suggests that as the disease progresses, the tendency for lactate accumulation during exercise increases. This may be attributed to worsening airflow limitation, increased ventilation-perfusion mismatch, and progressive skeletal muscle deconditioning in advanced stages of COPD. However, the relatively weak strength of correlation indicates that factors other than disease severity, such as individual physical conditioning, nutritional status, and comorbidities, also influence lactate production.

The coefficient of determination ($r^2 = 0.0854$) suggests that approximately 8.5% of the variation in lactate change can be explained by COPD severity alone. This reinforces the multifactorial nature of

exercise limitation in COPD. It also emphasizes the need for comprehensive assessment beyond spirometry, including evaluation of peripheral muscle function and metabolic responses.

The clinical implications of these findings are significant. Measurement of lactate levels before and after exercise can serve as a simple and objective tool to assess exercise tolerance and metabolic reserve in COPD patients. It may also help in identifying patients who would benefit from pulmonary rehabilitation programs aimed at improving muscle efficiency and aerobic capacity. Furthermore, monitoring lactate responses could be useful in tailoring individualized exercise prescriptions and tracking therapeutic outcomes [11].

However, certain limitations of the study must be acknowledged. The sample size was relatively small, and the study was conducted in a single center, which may limit generalizability. Additionally, factors such as physical activity levels, nutritional status, and muscle mass were not assessed, which could have influenced lactate dynamics. Future studies with larger sample sizes and inclusion of these parameters are recommended.

CONCLUSION

In conclusion, the present study demonstrates that stable COPD patients exhibit significant lactate elevation following submaximal exercise, reflecting impaired aerobic metabolism and reduced exercise capacity. The weak but significant correlation with disease severity suggests that lactate levels can provide additional insight into functional impairment in COPD. These findings support the role of lactate as a useful biomarker in the assessment and management of COPD.

REFERENCES

[1] Murray CJL, Lopez AD. Alternative projection of mortality and disability by cause 1990-2020: Global Burden of Disease Study. Lancet 1997; 349:1498-504.

[2] Barnes PJ, Celli BR. Systemic manifestations and comorbidities of COPD. EurRespir J 2009; 33:1165-85.

[3] Burrows B, Fletcher CM, Heard BE, Jones NL, Wootliff JS. The emphysematous and bronchial types of chronic airways obstruction: a clinicopathological study of patients in London and Chicago. Lancet 1966; 1:830–835.

[4] Decramer M, Janssens W, Miravitlles M (April 2012). "Chronic obstructive pulmonary disease". Lancet 379 (9823): 1341–51.

[5] Barnes PJ, Shapiro SD, Pauwels RA. Chronic obstructive pulmonary disease: molecular and cellular mechanisms. Eur Respir J 2003; 22:672–688.

[6] Agusti AG, Noguera A, Sauleda J, Sala E, Pons J, Busquets X. Systemic effects of chronic obstructive pulmonary disease. Eur Respir J 2003; 21:347–360.

[7] Agusti AG. COPD, a multicomponent disease: implications for management. Respir Med 2005; 99:670–682.

[8] MacDonald MI, Polkinghorne KR, MacDonald CJ, Leong P, Hamza K, Kathriachchige G, Osadnik CR, King PT, Bardin PG. Elevated blood lactate in COPD exacerbations associates with adverse clinical outcomes and signals excessive treatment with β_2 -agonists. Respirology. 2023 Sep;28(9):860-868.

[9] Amey Diwakar Paranjape, Varun Vijay Giri. Study of correlation of blood lactate levels with symptom score (BODE index) in COPD patients. MedPulse International Journal of Medicine. March 2021; 17(3); 50-54.

[10] Forey BA, Thornton AJ, Lee PN. Systematic review with meta-analysis of the epidemiological evidence relating smoking to COPD, chronic bronchitis and emphysema. BMC Pulm Med 2011; 11: 36. 12.

[11] Eisner MD, Anthonisen N, Coultas D, Kuenzli N, PerezPadilla R, Postma D, et al. An official American Thoracic Society public policy statement: novel risk factors and the global burden of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2010; 182: 693-718.