

TO STUDY OF LDH LEVEL IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a progressive disease of the lungs characterized by structural changes such as emphysema, airflow limitation, dynamic hyperinflation, air trapping, and peribronchial fibrotic remodeling of the lungs with significant systemic inflammatory components, induced by chronic exposures to smoking and/or occupational or environmental sources.

Methods: It is a case control study of 100 subjects divided into two groups including 50 healthy controls and 50 cases of COPD. Patients with history of respiratory infection, pneumonia, coronary heart disease, heart failure, and neuromuscular disease, renal and hepatic dysfunction were excluded.

Results: The statistically significant increased value of serum lactate dehydrogenase in cases as compared to control group with p value < 0.05.

Conclusion: Rise in serum LDH levels, though known to occur in many respiratory conditions, has not been studied in depth. Based on our study results, we have proved that patients with COPD have raised serum LDH levels.

Keywords: COPD, Lactate dehydrogenase, Smoking.

Introduction

Chronic obstructive pulmonary disease (COPD) affects 210 million people worldwide and kills > 4 million people every year, accounting for around 9% of total deaths. Ninety percent of these deaths occur in low- and middle-income countries. It is projected to be the 3rd leading cause of death by 2030.¹ Its chronic nature causes disruption of normal social roles, reduced workability, and poses massive burden on direct and indirect costs.²

Lactate dehydrogenase (LDH) is an intracellular cytoplasmic enzyme found in all tissues of the human body. There are five LDH isoenzymes present in blood, which are classified according to their electrophoretic movement. LDH-1 moves faster while LDH-5 is the slowest one. Elevated LDH isoenzymes levels indicate the organ specific origin of disease such as LDH-1, LDH-2 in heart, kidneys, erythrocytes and brain; LDH-3 in lungs, thyroid, pancreas, adrenals, spleen, thymus, lymph nodes and leukocytes; LDH-4 in skeletal muscles and the LDH-5 in hepatic system.³⁻⁴

Normal concentration LDH in the serum is due to normal tissue breakdown which increases significantly after tissue damage. LDH being a cytoplasmic cellular enzyme if increased in serum serve as indicator suggestive of disturbance of cellular integrity induced by pathological conditions.⁵ LDH is raised in number of pathological conditions like hematological disorders acute myocardial

infarction, liver diseases and several respiratory conditions. Respiratory conditions include bronchial asthma, bronchopneumonia, pulmonary tuberculosis, chronic obstructive pulmonary disease (COPD). All these conditions have inflammation, cell damage or both as underlying pathological mechanism.⁶

Material and Methods

This cross-sectional study was conducted on the inpatients and outpatients attending the Department of Medicine. Therefore, a total of 50 COPD patients and including 50 controls were enrolled in the study.

The exclusion criteria included

1. Uncooperativeness and unwillingness to participate in the study,
2. Being seriously ill,
3. Known case of carcinoma, bronchial asthma, active tuberculosis, diabetes, hypertension, collagen vascular disease,
4. Inability to properly perform spirometry,
5. The presence of congenital or valvular cardiomyopathy or other familial hyperlipidemias.

After the informed consent was obtained from all the participants. Subsequently, the COPD patients were segregated and diagnosed based on the GOLD guidelines with such risk factors as dyspnea, chronic cough, chronic sputum production, history of exposure to the respective

risk factors, and age of > 40 years. a detailed history was obtained from all the patients; furthermore, clinical examination and radiological tests were performed to confirm the presence of COPD and the associated cardiovascular complications.

After a 12-hour overnight fast, 5 ml fasting blood samples were collected from all the participants in the morning. Grossly hemolysed and lipemic samples were excluded. The LDH were directly analyzed using the standard enzymatic techniques

Results

Mean age in COPD patients was 41.25± 13.15 years and control patients was 40.23± 12.30 years and age range was 20-60 years. Both groups were well matched for age and sex distribution.

Table 1: LDH level in case and controls.

Parameters	Case (n=50)	Control (n=50)	p-value
Serum LDH(U/L)	346.23± 71.12	265.20± 78.12	<0.05

The statistically significant increased value of serum lactate dehydrogenase in cases as compared to control group with p value<0.05.

Discussion

Pulmonary system related disorders as possible sources of serum LDH abnormalities have been underreported, and isoenzyme patterns are seldom measured. This is the first study of its own kind in India to assess serum LDH level and lipid profile in patients with COPD. We found significant increase in Serum LDH level in the patients with COPD. This elevation is because of a predominant increase in serum LDH 3 isoenzymes which is released from cells of lung and airway origin.⁷ Airway mucosal changes consisting of increased broncho-alveolar mast cells, mononuclear phagocytic cells and epithelial shedding have been observed in chronic cough. In patients with chronic cough, a homogenous rise in cellular markers of inflammation has been observed in the bronchoalveolar lavage fluid⁸.

It is possible that persistent coughing may itself induce a degree of inflammation because of the trauma of the lining epithelium of the respiratory tract as well as that of the lung parenchyma. It is likely that the inflammatory process in patients with chronic cough is the cause of the increase in LDH.

Conclusion

COPD patients showed significantly higher serum levels of LDH to controls.

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