

Original article:

Study on biochemical parameters in chronic obstructive pulmonary diseases

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ABSTRACT:

Introduction: The study of thoracic medicine has experienced revolutionary transformations, and in recent years, our understanding of the pathophysiology of lung disorders has grown significantly. The lungs are unusually prone to infection and damage from ambient air. When all other parts of a medical or surgical disease are improving, pulmonary problems are dreaded occurrences that frequently result in mortality.

Materials and methods: The present study was conducted at department of Biochemistry, Fathima Institute of Medical Sciences, Kadapa, India. Study was conducted in the months of April-May 2016. The patients selected for study were admitted in the TB wards or attended O.P.D. of Hospital FIMS Kadapa. Total 60 patients were taken into this study in that 30 were diagnosed as TB, and 30 were controls.

Conclusion: By examining the CD4 and CD8 populations of lymphocytes, the activity of adenosine deaminase (ADA), and the production of antibodies against the 38 kDa antigen of Mycobacterium tuberculosis, this study has greatly aided our understanding of some immunological phenomena that are urgently needed today in the big "killer disease" - pulmonary tuberculosis.

Key words: Adenosine deaminase, Chronic obstructive pulmonary bronchitis, TB.

Introduction:

The study of thoracic medicine has experienced revolutionary transformations, and in recent years, our understanding of the pathophysiology of lung disorders has grown significantly. The lungs are unusually prone to infection and damage from ambient air. When all other parts of a medical or surgical disease are improving, pulmonary problems are dreaded occurrences that frequently result in mortality.[1] The lungs perform a number of other tasks besides breathing, such as maintaining water balance, preserving pH, expelling organisms and particles from the air we breathe, filtering particulate matter from the blood, and metabolising specific medications and enzymes.[2] Chronic in the group of illnesses known as chronic obstructive pulmonary bronchitis and emphysema are included disease (COPD). Breathing becomes more difficult with COPD with time. Although lung damage cannot be reversed, lifestyle adjustments and medication adjustments can help you manage the symptoms.[3]

The natural history of COPD is punctuated by exacerbations which have major implications on the patient and healthcare system. In this review we provide a concise overview of COPD exacerbations and their impact, outlining the population at risk, etiology and current management and preventive strategies.[4]

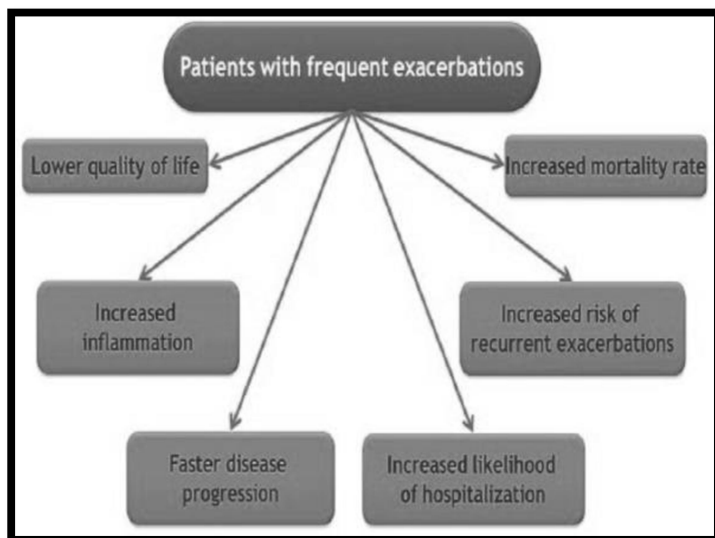


Figure 1: Impact of chronic obstructive pulmonary disease exacerbations.

When a patient with tuberculosis sneezes or coughs, bacteria are released into the air and can spread to other people. Overall, 1.7 billion people worldwide—or one-third of the population—have Mycobacterium TB infection (220a). Most healthy individuals who are infected manage it thanks to their robust immune systems. The cobra appears when the immune system becomes compromised because to starvation or advanced age. A person who has been infected runs the risk of having active TB for the rest of their lives. Up to 10% of people who contain the bacterium go on to develop the disease's active form.[4-6]

Materials and methods:

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Using sterile plastic syringes and adhering to all aseptic procedures, venous blood samples were taken. To measure Superoxide dismutase and Glutathione peroxidase, whole blood samples were drawn using EDTA as an anticoagulant. Sera that had not yet undergone hemolysis was used for the remaining investigations, and any sample that had even a hint of hemolysis was excluded. Within 48 hours following an asthma attack, blood samples of asthma patients were taken.

Results:

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Table 1: Pleural and serum ADA and their ratio in Pul. TB. with pleurisy and pleurisy due to other aetiology.

S.NO	Aetiology of Pleurisy	Pleural ADA U/L mean ± SD	Serum ADA U/L mean ± SD	Ratio of Pleural to serum ADA
01	Pulmonary TB.[n= 30]	68.33 ± 17.22	39.42±12.8	1.9±0.8
02	Non tubercular effusion[n 30]	15.23± 5.8	15.89±6.4	0.9± 0.24

When compared to group 2, or nontubercular effusion, the mean value of ADA of both serum and pleural fluid is considerably higher in group 1 (P 0.001), or effusion with pul TB. However, the gap between serum ADA and pleural.ADA* is much larger (372 percent) (210 percent) In 1 compared to 2, the ratio of pleural to serum ADA is considerably larger (P 0.001).

Table 2 : Erythrocyte superoxide Dismutase and Glutathione Peroxidase in pul TB :

S.NO	GROUP	SOD Levels in U/mg of Hb. Mean ± SD	GSHPx in U/g of Hb mean ± SD
01	Healthy controls[30]	4.19 ± 0.29	55.52 ± 7.85
02	Pul.TB Total[30]	2.5 ± 0.15	47.40 ± 4.9

Significant lowering of mean level of erythrocyte SOD is observed in total Pul. TB group compared to controls. A significant reduction in glutathione Peroxidase activity is found in pulmonary TB - whole group.

Discussion:

Disease develops in every organ as a result of either an external pathogen's introduction or an intrinsic failing of the tissue's structure and function. Pathogens that are carried by the air and in the blood are necessarily and naturally exposed to the lungs.[7] The most prevalent pulmonary diseases are: a) infectious diseases brought on by viruses, bacteria, mycobacteria, fungi, and parasites; b) obstructive diseases like asthma, chronic bronchitis, bronchiectasis, emphysema, and lung tumours; c) pulmonary circulation disorders like pulmonary embolism; d) infiltrative and

interstitial diseases like sarcoidosis. Mycobacterium tuberculosis is an intracellular bacteria that causes pulmonary tuberculosis, a serious infectious disease in India.[8-9]

We still don't fully understand the aetiology, immunology, and molecular biology of it. The range of different clinical and pathological symptoms that have been identified is mostly caused by variances in "host response. Currently, doing immunologic research is a key objective in tuberculosis research.[10-11]

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Conclusion:

By examining the CD4 and CD8 populations of lymphocytes, the activity of adenosine deaminase (ADA), and the production of antibodies against the 38 kDa antigen of Mycobacterium tuberculosis, this study has greatly aided our understanding of some immunological phenomena that are urgently needed today in the big "killer disease" - pulmonary tuberculosis. The activity of ADA rose in both the diseases, but the proportion of rise was quite high in pulmonary TB, which is an infectious disease, and asthma, which is an obstructive condition. Both disorders are observed to have elevated levels of lipid peroxidation together with decreased SOD activity. Although asthma does not affect GSHPx activity, pulmonary TB does. In contrast to asthma, where there was no change in the levels of these trace elements, pulmonary TB was characterised by an increase in serum Cu concentration and a decrease in serum Zn level.

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