

# Changes in Serum Oxidative Stress Biomarkers during Initial Antituberculosis Therapy: A Pilot

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## Introduction

The study of oxidative stress biomarkers in patients undergoing antituberculosis treatment provides valuable insights into the complex biological changes occurring during the therapeutic process. Oxidative stress refers to the imbalance between the productions of reactive oxygen species and the body's ability to detoxify these harmful molecules or repair the damage they cause. In the context of tuberculosis, oxidative stress has been implicated in the disease's pathogenesis, affecting both the infection itself and the subsequent healing process during treatment. This pilot study aimed to investigate the variations in serum oxidative stress biomarkers in patients receiving first-line antituberculosis therapy, contributing to a deeper understanding of how such therapies influence the body's oxidative status. Tuberculosis remains one of the leading infectious diseases globally, primarily affecting the lungs but capable of impacting other organs. The standard treatment for TB involves a combination of antibiotics known as first-line therapy, which typically includes drugs such as isoniazid, rifampin, pyrazinamide, and ethambutol. While these drugs are effective in killing the *Mycobacterium tuberculosis* bacteria, they also have an impact on the body's biochemical processes, including the modulation of oxidative stress. Oxidative stress is known to play a role in various diseases, including infectious diseases like TB, and it has been suggested that the imbalance between ROS and antioxidants in TB patients may contribute to disease progression and treatment outcomes. Therefore, understanding how serum biomarkers of oxidative stress fluctuate during the course of antituberculosis treatment is crucial for assessing the broader effects of the therapy on patients' health [1].

In this pilot study, a cohort of TB patients undergoing standard first-line antituberculosis treatment was monitored for changes in specific oxidative stress biomarkers in their serum over the course of their therapy. The biomarkers of interest included malondialdehyde, a product of lipid peroxidation; superoxide dismutase, an enzyme involved in the detoxification of superoxide radicals; and glutathione peroxidase, an enzyme that plays a key role in reducing hydrogen peroxide levels in cells. These markers were selected due to their central role in the antioxidant defense system and their known involvement in the oxidative stress response. The results of the study revealed significant variations in the levels of these biomarkers as patients progressed through their treatment. MDA levels, which reflect the extent of lipid damage caused by oxidative stress, were found to increase during the early stages of treatment, suggesting a heightened oxidative state at the beginning of therapy. This could be a result of the body's response to the bacterial infection and the inflammatory processes triggered by the *Mycobacterium tuberculosis* bacteria. However, as treatment continued, MDA levels began to decrease, indicating a reduction in lipid peroxidation and a potential improvement in the body's oxidative balance. This decline in MDA was consistent with the therapeutic effects of the antituberculosis drugs, which may have helped to reduce the bacterial load and inflammation, leading to a lower oxidative burden [2].

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Received: 02 November, 2024, Manuscript No. PE-24-156345; Editor Assigned: 04 November, 2024, PreQC No. P-156345; Reviewed: 18 November, 2024, QC No. Q-156345; Revised: 23 November, 2024, Manuscript No. R-156345; Published: 30 November, 2024, DOI: 10.37421/2472-1042.2024.9.252

## Description

In contrast, the levels of antioxidant enzymes such as SOD and GPx showed a different pattern. Initially, both SOD and GPx levels were relatively low, indicating that the antioxidant defense mechanisms were overwhelmed by the oxidative stress induced by the TB infection. However, as treatment progressed, the levels of these enzymes gradually increased, suggesting that the body's antioxidant response was activated in response to the oxidative damage caused by the disease and the ongoing treatment. The increase in these antioxidant biomarkers may be a sign of the body's adaptive response to the treatment, as the enzymes work to neutralize ROS and prevent further cellular damage [3].

The variations in oxidative stress biomarkers observed in this study provide important clues about the role of oxidative stress in tuberculosis and its modulation during treatment. The initial increase in MDA levels suggests that oxidative stress is a prominent feature of TB infection, likely exacerbated by the inflammatory processes associated with the disease. The subsequent decrease in MDA levels during treatment, coupled with the rise in antioxidant enzyme activity, points to the effectiveness of the antituberculosis drugs in not only killing the bacteria but also in modulating the oxidative stress response. These findings highlight the complex interplay between oxidative stress and the body's immune response during TB treatment, emphasizing the need for a balanced oxidative state to support recovery [4].

While the results of this pilot study are promising, it is important to acknowledge the limitations of the research. The sample size was relatively small, and further studies with larger cohorts would be necessary to validate these findings and assess their generalizability. Additionally, the study did not explore the relationship between oxidative stress biomarkers and clinical outcomes such as treatment success or relapse, which could provide further insights into the clinical relevance of these biomarkers. Future research could also investigate the potential therapeutic benefits of antioxidant supplementation in conjunction with antituberculosis treatment, as this could help to optimize patient outcomes by reducing oxidative damage and supporting the body's natural defense mechanisms [5].

## Conclusion

This pilot study provides valuable data on the variations in serum oxidative stress biomarkers during the course of first-line antituberculosis treatment. The findings suggest that oxidative stress plays a significant role in the pathogenesis of tuberculosis and that antituberculosis therapy may help to modulate the oxidative balance in the body. The increase in antioxidant enzyme activity observed during treatment further supports the idea that the body's defense mechanisms are activated in response to the oxidative damage caused by the infection. These insights into the biochemical changes occurring during TB treatment could inform future research on improving treatment strategies and managing oxidative stress in TB patients.

## Acknowledgement

None.

## Conflict of Interest

None.

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**How to cite this article:** Boonyuen, Kristiina. "Changes in Serum Oxidative Stress Biomarkers during Initial Ant tuberculosis Therapy: A Pilot." *Pharmacoeconomics* 9 (2024): 252.