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Biochemical Assessment of Oxidative Stress, Antioxidant Status, and Lipid Profile in Pulmonary and Abdominal Tuberculosis Patients Receiving Anti- Tubercular Therapy

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ABSTRACT

Background: Tuberculosis (TB) remains a significant global health burden, particularly in developing countries like Pakistan. Beyond its infectious nature, TB is associated with increased oxidative stress and metabolic alterations. However, limited data exist on the biochemical response to anti-tubercular therapy (ATT) in both pulmonary and abdominal TB cases. Therefore, this study aimed to evaluate the effect of ATT on oxidative stress markers, antioxidant enzymes, and lipid profiles in patients with pulmonary and abdominal TB.

Methods: A prospective observational study was conducted over a 12-month period (March 2023 to February 2024) on 83 patients with pulmonary or abdominal tuberculosis at multiple tertiary care hospitals across Pakistan. Patients were recruited using a non-probability purposive sampling technique. Blood samples were collected at baseline (before treatment) and after two months of standard ATT. Malondialdehyde (MDA), nitric oxide (NO), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), total antioxidant capacity (TAC), and lipid profile parameters were analyzed. Data were analyzed using SPSS version 26, and paired sample and independent t-tests were applied with a significance level set at $p < 0.05$.

Results: Among the 83 tuberculosis patients enrolled, 47 (56.6%) were males and 36 (43.4%) females, with 47 (56.6%) having pulmonary TB and 36 (43.4%) abdominal TB. Significant reductions in oxidative stress markers were observed post-treatment: MDA decreased from 5.26 ± 1.31 to 2.79 ± 1.01 and NO from 4.82 ± 1.00 to 3.95 ± 1.02 ($p < 0.001$ for both). Antioxidant enzymes showed marked improvement: SOD increased from 2.02 ± 0.65 to 3.28 ± 0.87, CAT from 2.54 ± 0.99 to 4.46 ± 1.00, GPx from 1.91 ± 0.57 to 3.08 ± 0.72, and TAC from 0.94 ± 0.31 to 1.62 ± 0.32 (all $p < 0.001$). Lipid markers also improved, with total cholesterol rising from 127.71 ± 18.18 to 154.53 ± 19.75, LDL from 71.27 ± 15.05 to 88.05 ± 16.61, HDL from 31.59 ± 5.95 to 39.54 ± 7.23, TG from 107.98 ± 19.84 to 122.97 ± 20.15, and VLDL from 22.35 ± 3.81 to 25.69 ± 4.62 (all $p < 0.001$). No significant differences were found in these biochemical changes between pulmonary and abdominal TB groups ($p > 0.05$).

Conclusion: Anti-tubercular therapy leads to a marked reduction in oxidative stress and improvement in antioxidant and lipid profiles in both pulmonary and abdominal TB patients. These findings highlight the systemic impact of TB and underscore the biochemical recovery that follows treatment

Keywords: Lipid Metabolism, Abdominal TB with Tuberculosis, Abdominal, Anti-Tubercular Therapy, Antitubercular Agents.

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INTRODUCTION

Tuberculosis (TB) continues to be one of the top ten causes of death worldwide, with an estimated 10.6 million people developing the disease annually, according to the World Health Organization (WHO, 2023)[1,2]. Despite global efforts to control TB, countries like Pakistan remain among the top five contributors to the global TB burden, where both pulmonary and extrapulmonary forms of the disease are prevalent[3,4].

While TB is classically characterized by its infectious and pulmonary manifestations, accumulating evidence suggests that it also causes profound biochemical disturbances[5]. The chronic inflammatory state associated with TB leads to an increase in reactive oxygen species (ROS), resulting in oxidative damage to lipids, proteins, and DNA. This imbalance between oxidants and antioxidants is well-documented in international studies, which reported elevated levels of MDA and nitric oxide (NO) in TB patients[6,7]. Similarly, local studies from Pakistan have highlighted antioxidant enzyme depletion in untreated TB cases, suggesting a clear oxidative burden[8,9].

Furthermore, TB also affects lipid metabolism. Several studies have reported decreased levels of HDL and total cholesterol in TB patients, likely due to malnutrition and immune activation[10,11,12]. However, data comparing pulmonary and abdominal TB in terms of biochemical response to treatment remain sparse. While most available literature has focused on pulmonary TB, there is a lack of comparative studies evaluating oxidative and lipid profile changes in extrapulmonary cases, particularly abdominal TB.

This gap in evidence limited the understanding of whether the site of tuberculosis influenced systemic oxidative or metabolic outcomes and whether both forms of the disease responded similarly to treatment. Hence, the present study was undertaken to evaluate the changes in oxidative stress markers, antioxidant enzymes, and lipid profiles before and after antitubercular therapy in patients with pulmonary and abdominal tuberculosis. By addressing this gap, the study aimed to provide meaningful insight into the systemic effects of antitubercular therapy and to determine whether therapeutic outcomes differed by tuberculosis type.

METHODS

This was a prospective, observational study designed to assess the impact of antitubercular therapy (ATT) on oxidative stress levels, antioxidant enzyme status, and lipid profiles in patients diagnosed with either pulmonary or abdominal tuberculosis. The study was conducted over 12 months, from March 2023 to

February 2024, at multiple tertiary care hospitals across Pakistan, including the Armed Forces Combined Military Hospital (CMH) Rawalakot, Sheikh Khalifa Bin Zayed Al-Nahyan Medical Complex Rawalakot, Khyber Teaching Hospital Peshawar, Rawal General and Dental Hospital Islamabad, Combined Military Hospital Rawalakot, Ayub Teaching Hospital Abbottabad, and the National University Hospital Islamabad, all of which are actively involved in the diagnosis and management of both pulmonary and extrapulmonary tuberculosis. Ethical approval for the research was obtained from the hospital's Ethics Review Committee under reference number 617/SKBZ/CMH/RKT, dated 02-02-2023.

The required sample size was calculated using **G*Power software** for a paired sample *t*-test, assuming a medium effect size (Cohen's $d = 0.5$)[13], a 95% confidence level, and 80% statistical power. This yielded a minimum sample size of 67 participants. To account for potential dropouts or incomplete data, a total of 83 patients were recruited. A non-probability purposive sampling technique was employed, including only those who met the inclusion criteria and provided informed consent.

Inclusion Criteria were patients aged between 18 and 65 years. Confirmed diagnosis of either pulmonary TB (via sputum AFB/GenXpert and chest imaging) or abdominal TB (via ultrasound, CT scan, ascitic fluid analysis, or histopathology). Patients who had not previously received ATT. And illingness to provide written informed consent and comply with follow-up.

Exclusion Criteria were patients with chronic illnesses such as diabetes mellitus, chronic kidney disease, liver failure, cardiovascular disease, or HIV. Individuals already taking antioxidants, lipid-lowering agents, or steroids. Pregnant or lactating women. Cases of multi-drug-resistant (MDR) tuberculosis. Patients who failed to complete the intensive phase of therapy or were lost to follow-up.

At the time of enrollment (before the start of ATT), each participant's demographic data (age, gender, smoking status, BMI, TB type) were recorded on a structured proforma. Two blood samples (5 ml each) were collected from each patient: Before starting therapy (baseline). After 2 months of therapy (end of intensive phase)

Blood was collected under aseptic conditions and analyzed for Oxidative Stress Markers: MDA, Nitric Oxide (NO). Antioxidant Enzymes: Superoxide Dismutase (SOD), Catalase (CAT), Glutathione Peroxidase (GPx), and Total Antioxidant Capacity (TAC) and Lipid Profile Components: Total Cholesterol, LDL, HDL, Triglycerides (TG), and Very-Low-Density Lipoprotein (VLDL)

All biochemical assessments were carried out in the hospital laboratory using standardized spectrophotometric and colorimetric kits as per the manufacturer's protocols.To ensure reliability, all tests were conducted in duplicate, and mean values were used to reduce random measurement error. The same equipment and technician were used throughout the study period to minimize inter-observer and procedural variability. Internal quality control samples were run alongside every batch to maintain assay consistency.

All patients received the standard first-line anti-tubercular therapy (ATT) regimen recommended by the National TB Control Programme of Pakistan, consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol during the intensive phase (first two months), followed by isoniazid and rifampicin in the continuation phase. The regimen was uniform for both pulmonary and abdominal TB patients, ensuring treatment consistency across the cohort.Validity was ensured by selecting widely accepted biochemical markers relevant to oxidative stress and lipid metabolism. Diagnostic accuracy for TB type was confirmed by established clinical and laboratory criteria, reducing the risk of misclassification.

Data entry and analysis were performed using IBM SPSS Statistics version 26. Categorical variables (gender, age group, BMI category, smoking status, TB type) were presented as frequencies and percentages, while continuous variables were expressed as means and standard deviations (mean ± SD). Before running statistical tests, all continuous variables were assessed for normality using the **Shapiro–Wilk test.** As most variables followed a normal distribution ($p > 0.05$), parametric tests were applied. Paired Sample t-test was used to assess changes in oxidative stress markers, antioxidant enzymes, and lipid profiles before and after ATT within the same group. Chi-square test was used to examine associations between TB type and categorical variables like age group, gender, BMI, and smoking status. An Independent Sample t-test was used to compare mean change scores (post–pre) of oxidative and lipid markers between pulmonary and abdominal TB groups. A p-value less than 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Characteristics and Association with TB Type (n = 83)

Variable	Subgroup	Pulmonary TB (n=52)	Abdominal TB (n=31)	χ^2 (df)	p-value
Gender	Male	31 (59.62%)	16 (51.61%)	1.137 (1)	0.286
	Female	21 (40.38%)	15 (48.39%)		
Smoking Status	Smoker	20 (38.46%)	12 (38.71%)	0.003 (1)	0.956
	Non-smoker	32 (61.54%)	19 (61.29%)		
Age Group	<30	12 (23.08%)	8 (25.81%)	0.634 (2)	0.728
	30–50	21 (40.38%)	11 (35.48%)		
	>50	19 (36.54%)	12 (38.71%)		
BMI Category	Underweight	12 (23.08%)	4 (12.90%)	2.675 (2)	0.262
	Normal	32 (61.54%)	20 (64.52%)		
	Overweight	8 (15.38%)	7 (22.58%)		
Abdominal TB Subtypes	Peritoneal	–	12 (38.71%)	–	–
	Intestinal	–	9 (29.03%)	–	–
	Hepatic	–	5 (16.13%)	–	–

	Renal/Adrenal	–	5 (16.13%)	–	–

Among the 83 patients studied, 52 (62.65%) had pulmonary TB and 31 (37.35%) had abdominal TB. Males were slightly more common in pulmonary TB (59.62%) compared to abdominal TB (51.61%), while females were more frequent in abdominal TB (48.39%) than pulmonary TB (40.38%), with no significant association (p=0.286). Smoking status was nearly identical between groups (38.46% vs. 38.71%, p=0.956). The majority of patients were aged 30–50 years (40.38% pulmonary vs. 35.48% abdominal), followed by >50 years (36.54% vs. 38.71%) and <30 years (23.08% vs. 25.81%), showing no significant difference (p=0.728). Most patients were in the normal BMI category (61.54% pulmonary vs. 64.52% abdominal), with fewer being underweight (23.08% vs. 12.90%) or overweight (15.38% vs. 22.58%), also non-significant (p=0.262). Among abdominal TB subtypes, peritoneal TB was most frequent (38.71%), followed by intestinal (29.03%), hepatic (16.13%), and renal/adrenal involvement (16.13%).

Table 2: Comparison of Oxidative Stress Markers Before and After Anti-Tubercular Therapy (n = 83)

Marker	Mean ± SD (Before)	Mean ± SD (After)	Mean Difference	t (df)	*p*-value
Malondialdehyde (MDA)	5.26 ± 1.31	2.79 ± 1.01	2.47	14.213 (82)	< 0.001
Nitric Oxide (NO)	4.82 ± 1.00	3.95 ± 1.02	0.87	5.872 (82)	< 0.001

Values are presented as mean ± standard deviation. Statistical analysis was performed using the paired sample t-test

The levels of oxidative stress markers significantly decreased after completion of anti-tubercular therapy. Mean MDA levels dropped from 5.26 ± 1.31 to 2.79 ± 1.01, showing a significant mean difference of 2.47 ($t(82) = 14.213$, $p < 0.001$). Similarly, NO levels declined from 4.82 ± 1.00 to 3.95 ± 1.02 with a mean difference of 0.87 ($t(82) = 5.872$, $p < 0.001$). These results reflect a marked reduction in oxidative damage following treatment, supporting the role of ATT in oxidative stress resolution in TB patients.

Table 3: Comparison of Antioxidant Markers Before and After Anti-Tubercular Therapy (n = 83)

Marker	Mean ± SD (Before)	Mean ± SD (After)	Mean Difference	t (df)	*p*-value
Superoxide Dismutase (SOD)	2.02 ± 0.65	3.28 ± 0.87	-1.27	-11.568 (82)	< 0.001

Catalase (CAT)	2.54 ± 0.99	4.46 ± 1.00	-1.92	-12.558 (82)	< 0.001
Glutathione Peroxidase (GPx)	1.91 ± 0.57	3.08 ± 0.72	-1.17	-12.031 (82)	< 0.001
Total Antioxidant Capacity (TAC)	0.94 ± 0.31	1.62 ± 0.32	-0.68	-13.585 (82)	< 0.001

Values are expressed as Mean ± SD. Statistical analysis was performed using paired sample t-test

Following anti-tubercular therapy, significant improvements were observed in all measured antioxidant markers. Superoxide dismutase (SOD) levels rose from 2.02 ± 0.65 to 3.28 ± 0.87 ($t(82) = -11.568$, $p < 0.001$), and catalase (CAT) increased from 2.54 ± 0.99 to 4.46 ± 1.00 ($t(82) = -12.558$, $p < 0.001$). Similarly, GPx levels improved from 1.91 ± 0.57 to 3.08 ± 0.72 ($t(82) = -12.031$, $p < 0.001$). TAC also showed a marked rise from 0.94 ± 0.31 to 1.62 ± 0.32 ($t(82) = -13.585$, $p < 0.001$). These findings collectively indicate a strong enhancement of the body's antioxidant defenses after treatment.

Table 4: Comparison of Lipid Profile Before and After Anti-Tubercular Therapy (n = 83)

Lipid Marker	Mean ± SD (Before)	Mean ± SD (After)	Mean Difference	t (df)	*p*-value
Total Cholesterol	127.71 ± 18.18	154.53 ± 19.75	-26.82	-8.510 (82)	< 0.001
LDL	71.27 ± 15.05	88.05 ± 16.61	-16.78	-6.639 (82)	< 0.001
HDL	31.59 ± 5.95	39.54 ± 7.23	-7.95	-7.711 (82)	< 0.001
Triglycerides (TG)	107.98 ± 19.84	122.97 ± 20.15	-15.00	-4.889 (82)	< 0.001
VLDL	22.35 ± 3.81	25.69 ± 4.62	-3.34	-4.984 (82)	< 0.001

Values are expressed as Mean ± SD. Statistical analysis was performed using paired sample t-test.

Significant changes were observed in the lipid profile of tuberculosis patients following anti-tubercular therapy. Total cholesterol levels increased from 127.71 ± 18.18 to 154.53 ± 19.75 ($t(82) = –8.510$, $p < 0.001$), and LDL levels rose from 71.27 ± 15.05 to 88.05 ± 16.61 ($t(82) = –6.639$, $p < 0.001$). HDL, which is protective, improved significantly from 31.59 ± 5.95 to 39.54 ± 7.23 ($t(82) = –7.711$, $p < 0.001$). Triglyceride and VLDL values also increased significantly, with TG rising from 107.98 ± 19.84 to 122.97 ± 20.15 ($t(82) = –4.889$, $p < 0.001$) and VLDL from 22.35 ± 3.81 to

25.69 ± 4.62 ($t(82) = –4.984$, $p < 0.001$). These shifts may reflect an overall nutritional recovery post-therapy.

Table 5: Comparison of Mean Change in Biochemical Markers Between Pulmonary and Abdominal TB Patients (n = 83)

Marker Change	Pulmonary (n = 47) Mean ± SD	Abdominal (n = 36) Mean ± SD	*t* (df)	*p*-value
MDA_Change	2.64 ± 1.64	2.24 ± 1.50	1.14 (81)	0.256
NO_Change	0.79 ± 1.40	0.98 ± 1.30	–0.65 (81)	0.520
SOD_Change	1.33 ± 0.95	1.19 ± 1.06	0.65 (81)	0.519
CAT_Change	2.09 ± 1.34	1.69 ± 1.44	1.30 (81)	0.199
GPx_Change	1.17 ± 0.97	1.17 ± 0.77	–0.04 (81)	0.969
TAC_Change	0.66 ± 0.48	0.70 ± 0.43	–0.42 (81)	0.674
LDL_Change	18.93 ± 25.03	13.98 ± 20.13	0.97 (81)	0.335
Cholesterol_Change	28.26 ± 28.76	24.93 ± 28.95	0.52 (81)	0.603
HDL_Change	7.52 ± 8.29	8.52 ± 10.77	–0.48 (81)	0.632
TG_Change	15.78 ± 28.44	13.97 ± 27.65	0.29 (81)	0.771
VLDL_Change	3.82 ± 6.64	2.71 ± 5.34	0.82 (81)	0.415

Values are expressed as Mean ± SD. Statistical analysis was performed using an independent sample t-test.

No statistically significant differences were observed in the mean change of biochemical markers between pulmonary and abdominal TB groups. While the pulmonary group showed slightly higher reductions in oxidative stress markers (MDA and NO) and moderate gains in antioxidant enzymes (SOD, CAT), these differences did not reach statistical significance ($p > 0.05$). Similarly, the lipid profile changes, including LDL, HDL, cholesterol, triglycerides, and VLDL, did not differ

significantly between the two TB types. These findings suggest that both forms of tuberculosis responded similarly to anti-tubercular therapy in terms of oxidative stress and metabolic changes.

As shown in **Figure 1,** there was a substantial decrease in oxidative stress markers, with MDA levels falling from 5.26 to 2.79 and NO from 4.82 to 3.95 after treatment. Concurrently, antioxidant enzymes displayed marked improvement. SOD increased from 2.02 to 3.28, CAT from 2.54 to 4.46, GPx from 1.91 to 3.08, and TAC from 0.94 to 1.62. The relatively low standard deviations indicate consistent responses among patients. These trends clearly demonstrate the beneficial effect of ATT in reducing oxidative damage and restoring antioxidant defense mechanisms in tuberculosis patients.

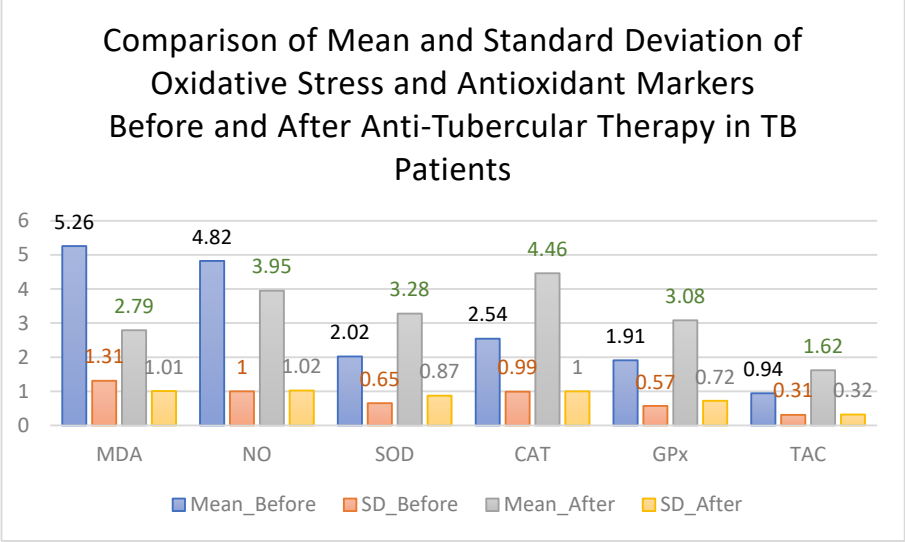

Figure 1: Comparison of Mean and Standard Deviation of Oxidative Stress and Antioxidant Markers Before and After Anti-Tubercular Therapy in TB Patients (n = 83).

The bar chart illustrates the changes in oxidative stress markers (MDA, NO) and antioxidant enzymes (SOD, CAT, GPx, TAC) before and after the completion of intensive-phase anti-tubercular therapy. Each marker is represented with its corresponding mean and standard deviation before and after treatment

DISCUSSION

This study investigated the impact of anti-tubercular therapy (ATT) on oxidative stress, antioxidant defense mechanisms, and lipid profiles in patients with pulmonary and abdominal tuberculosis. The findings consistently showed that ATT leads to significant biochemical recovery in both TB types, reinforcing the dual role of therapy in not only eradicating Mycobacterium tuberculosis but also mitigating systemic oxidative and metabolic damage.

A marked reduction in oxidative stress was observed following ATT, as reflected by the significant decrease in MDA and NO levels. These findings align with previous reports that tuberculosis infection increases lipid peroxidation and reactive nitrogen species due to chronic inflammation, which gradually normalizes as the infection is controlled14,15. The substantial drop in MDA and NO after therapy thus suggests effective suppression of infection-associated oxidative damage.

Concomitant with this reduction, antioxidant markers such as SOD, CAT, GPx, and TAC improved significantly. This enhancement indicates a restoration of endogenous antioxidant defenses, consistent with previous research that reported antioxidant system rebound after microbial clearance16,17. The simultaneous decrease in oxidative markers and increase in enzymatic antioxidants collectively highlight a favorable oxidative-antioxidative shift during recovery.

Interestingly, lipid parameters also changed significantly post-treatment. Increases in total cholesterol, LDL, and HDL levels may appear paradoxical but likely reflect a return toward metabolic normalcy. TB-associated cachexia often results in depressed lipid levels due to malnutrition and infection-related catabolism18-20. As patients recover and regain weight, these values rebound. The observed HDL increase is particularly encouraging, as HDL plays a protective role in immune regulation and lipid transport, both essential during immune recovery.

Despite clear within-subject improvements, no statistically significant differences in treatment response were found between pulmonary and abdominal TB groups21,22. Both types of TB showed comparable reductions in MDA and NO, and similar improvements in antioxidant and lipid markers. This suggests that the systemic biochemical burden of tuberculosis may be similar regardless of primary disease site, and that ATT yields broadly effective results across TB subtypes. This finding is in agreement with previous research studies that found no significant variance in oxidative stress responses between extrapulmonary and pulmonary TB cases23-25.

The demographic analysis also revealed no significant association between TB type and factors like gender, age, BMI, or smoking status, suggesting an equitable distribution of these baseline characteristics. The relatively high proportion of underweight patients and smokers highlights known TB risk factors and underscores the importance of nutritional and behavioral interventions in TB management.

In addition to biochemical and metabolic recovery, clinical improvements were also observed in the majority of patients. Most reported resolution of hallmark symptoms such as fever, night sweats, and cough, along with improved appetite, weight gain, and overall well-being by the end of the intensive phase. These outcomes were noted during follow-up but were not formally included in statistical

analysis, as the primary focus of this study was biochemical. We acknowledge this as a limitation and suggest that future research should integrate both laboratory and clinical endpoints for a more comprehensive assessment of treatment outcomes.

While this study provides valuable insights into the biochemical response of TB patients to anti-tubercular therapy, certain limitations should be acknowledged. Firstly, the sample size was relatively modest and drawn from a single center, which may limit the generalizability of the findings to broader populations. Secondly, the study only assessed short-term biochemical changes after two months of treatment; long-term follow-up was not included to observe sustained effects or relapse. Third, although nutritional status likely influenced oxidative and lipid markers, dietary intake and supplementation were not systematically recorded or controlled. Additionally, the study did not account for possible confounders such as subclinical comorbidities, socioeconomic status, or variations in drug adherence. Lastly, while oxidative stress markers were measured, inflammatory markers and immune status indicators were not included, which could have provided a more comprehensive understanding of treatment impact.

Future studies should aim to include larger, multicentric cohorts to validate these findings across diverse populations. It is also recommended to conduct long-term follow-up assessments to evaluate whether the improvements in oxidative and lipid markers are sustained after treatment completion. Incorporating dietary assessments and controlling for nutritional interventions could help clarify the role of nutrition in biochemical recovery. Furthermore, the inclusion of inflammatory and immune biomarkers would provide a more holistic view of the physiological response to anti-tubercular therapy. Lastly, randomized controlled trials investigating the role of adjunct antioxidant supplementation alongside ATT are encouraged to explore potential benefits in enhancing recovery and reducing oxidative damage.

CONCLUSION

In conclusion, this study demonstrates that anti-tubercular therapy significantly reduces oxidative stress and improves antioxidant capacity and lipid metabolism in TB patients, regardless of disease location. Both pulmonary and abdominal TB cases responded similarly to treatment, highlighting the systemic nature of tuberculosis-induced oxidative and metabolic derangements. These findings reinforce the need to monitor oxidative and nutritional status in TB patients and support the incorporation of antioxidant and dietary support strategies alongside standard ATT to enhance recovery outcomes. Future studies with longer follow-up periods and inclusion of nutritional supplementation data are warranted to expand on these insights.

LIST OF ABBREVIATIONS

TB: Tuberculosis

ATT: Anti-Tubercular Therapy

MDA: Malondialdehyde

NO: Nitric Oxide

SOD: Superoxide Dismutase

CAT: Catalase

GPx: Glutathione Peroxidase

TAC: Total Antioxidant Capacity

LDL: Low-Density Lipoprotein

HDL: High-Density Lipoprotein

TG: Triglycerides

VLDL: Very-Low-Density Lipoprotein

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FUNDING

None

CONFLICT OF INTEREST

None

ETHICAL APPROVAL

This study was approved by the Institutional Review Board (IRB) (Approval No: 617/SKBZ/CMH/RKT, dated 02-02-2023). Written informed consent was obtained from all participants prior to enrollment. The study was conducted in accordance with the principles of the Declaration of Helsinki.

PATIENT CONSENT

Written informed consent was obtained from all study participants before inclusion in the study. Patients were assured of confidentiality, and their data were anonymized for analysis and publication purposes.

AUTHORS' CONTRIBUTION

SFF: Conceived and designed the study, analyzed and interpreted the biochemical and pharmacological data, and was a major contributor in writing the manuscript. **SR:** Assisted in study design, contributed to pharmacological data interpretation, and critically reviewed the manuscript for intellectual content. **RM:** Contributed to literature review, data collection, and manuscript drafting. **ZR:** Performed biochemical assays, analyzed laboratory data, and contributed to the results section. **MAQ:** Assisted in clinical evaluation of patients, provided gastroenterology input, and contributed to patient recruitment and clinical data interpretation. **ZKK:** Assisted in biochemical data analysis, contributed to manuscript writing, and reviewed the final draft for accuracy. All authors read and approved the final manuscript.

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