Original Article

Assessment of Radiological Lesion Burden and Serum Oxidative Stress in Multidrug-Resistant Pulmonary Tuberculosis

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Abstract

Background: Multidrug-resistant tuberculosis (MDR-TB) presents a significant clinical challenge, characterized by prolonged treatment, high morbidity, and increased mortality. Radiological imaging provides critical information on disease burden, while oxidative stress biomarkers offer insights into host-pathogen interactions and tissue injury. However, limited studies have examined the association between radiological lesion extent and systemic oxidative stress in MDR-TB. This is study to evaluate the correlation between radiological lesion burden and serum oxidative stress markers in patients with microbiologically confirmed MDR-TB. **Materials and Methods:** This cross-sectional observational study was conducted from May 2023 to April 2024 at a tertiary care center. Fifty adult patients with confirmed MDR-TB were enrolled. Radiological assessment was performed using chest X-ray and high-resolution computed tomography (HRCT), and lesion burden was quantified using a standardized scoring system. Venous blood samples were analyzed for malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), and catalase activities. Statistical analysis included Pearson correlation and multiple linear regression to evaluate relationships between lesion scores and oxidative stress parameters, adjusting for confounders. **Results:** Patients with higher radiological scores exhibited significantly elevated MDA levels (mean ± SD: 6.89 ± 1.12 nmol/mL) and reduced antioxidant defenses, including GSH (2.54 ± 0.46 µmol/L), SOD (6.31 ± 1.14 U/mL), and catalase (32.2 ± 4.7 kU/L) (all $p < 0.001$). Radiological burden showed a strong positive correlation with MDA ($r = 0.67$, $p < 0.001$) and significant negative correlations with GSH ($r = –0.59$), SOD ($r = –0.56$), and catalase ($r = –0.53$). Regression analysis confirmed lesion burden as an independent predictor of oxidative imbalance. **Conclusion:** Radiological lesion extent in MDR-TB is strongly associated with increased lipid peroxidation and reduced antioxidant activity, indicating that oxidative stress intensifies with greater pulmonary involvement. Integrating oxidative stress profiling with imaging could enhance prognostic assessment and guide adjunctive therapeutic strategies.

Keywords: Multidrug-resistant tuberculosis; Oxidative stress; Radiological lesion burden; Malondialdehyde; Antioxidant enzymes; High-resolution computed tomography.

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Introduction

Tuberculosis (TB) continues to be one of the most pressing global health concerns, affecting millions annually and causing substantial morbidity and mortality. According to the World Health Organization (WHO) Global Tuberculosis Report 2023, an estimated 10.6 million people developed TB in 2022, with 1.3 million deaths among HIV-negative individuals and 167,000 deaths among those living with HIV.[1] While TB is curable with appropriate treatment, the emergence and spread of multidrug-resistant tuberculosis (MDR-TB)—defined as infection with Mycobacterium tuberculosis strains resistant to at least isoniazid and rifampicin—has complicated global control efforts.[2] India remains one of the highest MDR-TB burden countries, accounting for a significant proportion of global cases.[1,3]

MDR-TB poses unique clinical challenges. It requires prolonged therapy with second-line drugs, which are often less effective, more toxic, and costlier than first-line regimens.[4] Clinical outcomes are generally poorer, and the risk of treatment failure and relapse is higher compared to drug-susceptible TB.[5] These factors make timely diagnosis, accurate assessment of disease severity, and early prognostication crucial for guiding patient management.

Radiological imaging is central to TB evaluation. Conventional chest radiography and high-resolution computed tomography (HRCT) are widely used to detect and quantify pulmonary lesions such as cavities, nodules, consolidation, fibrosis, and bronchiectasis.[6,7] The extent and severity of these lesions, often referred to as the "radiological burden," provide valuable information about disease stage, bacterial load, and potential infectivity.[8] HRCT, with its superior spatial resolution, allows for detailed assessment of parenchymal destruction and subtle

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changes not easily visualized on plain radiographs.[9] Studies have shown that MDR-TB tends to present with more extensive and bilateral lesions, larger cavities, and more pronounced bronchiectasis changes compared to drug-susceptible cases.[8-10]

While imaging evaluates anatomical and structural damage, biochemical markers can shed light on the underlying pathogenic processes. Oxidative stress plays a pivotal role in TB pathophysiology. The host immune system mounts an oxidative burst, producing ROS in macrophages and neutrophils to kill M. tuberculosis. Although beneficial for pathogen clearance, excessive ROS can cause collateral injury to host tissues.[11,12] This imbalance between ROS generation and antioxidant defenses leads to oxidative stress, which can exacerbate pulmonary damage and impair immune regulation.[13]

Several biochemical indicators reflect oxidative stress status in TB patients. Malondialdehyde (MDA) is a stable end-product of lipid peroxidation and serves as a reliable marker of oxidative injury to cell membranes.[14] Reduced glutathione (GSH) acts as a major non-enzymatic antioxidant, directly scavenging free radicals and regenerating other antioxidants.[15] Enzymatic defences such as superoxide dismutase (SOD) and catalase neutralise ROS intermediates and protect cellular components from oxidative damage.[16] Prior research has documented elevated MDA levels and reduced GSH, SOD, and catalase activities in TB patients, with more severe alterations in advanced or complicated disease.[17,18]

Despite evidence linking oxidative stress to TB pathogenesis and demonstrating more severe radiological patterns in MDR-TB, there is a scarcity of studies exploring their direct relationship. Understanding whether a higher radiological lesion burden correlates with greater oxidative imbalance could provide important prognostic insights, highlight the extent of ongoing tissue injury, and potentially inform adjunctive therapeutic strategies, such as antioxidant supplementation, alongside standard anti-TB regimens.

The present study was therefore designed to evaluate the correlation between radiological lesion burden, quantified through HRCT-based scoring, and serum oxidative stress markers (MDA, GSH, SOD, catalase) in patients with microbiologically confirmed MDR-TB. By integrating structural imaging with biochemical profiling, this research aims to provide a more comprehensive assessment of disease status, which could be valuable for both clinical decision-making and future interventional studies.

Materials and Methods

Study Design and Setting: This hospital-based, cross-sectional analytical study was carried out jointly by the Departments of Radiology, Microbiology, and Biochemistry at Mahatma Gandhi Memorial (MGM) Hospital, Warangal, Telangana, India. MGM Hospital is a tertiary care teaching institution catering to both rural and urban populations, receiving a high referral load for TB diagnosis and management. The study was conducted over a 12-month period from December 2023 to November 2024, enabling inclusion of cases across seasonal variations and ensuring an adequate sample size. The study aimed to assess the relationship between radiological lesion burden and serum oxidative stress markers in patients with microbiologically confirmed MDR-TB.

Study Population: The study included adult patients aged 18 years and above who were newly diagnosed with pulmonary MDR-TB. MDR-TB diagnosis was established through the GeneXpert MTB/RIF assay for initial rifampicin resistance detection, followed by confirmation via culture and drug susceptibility testing (DST) according to standard protocols. Only those who underwent HRCT chest imaging within two weeks of biochemical sample collection were included to minimise the effect of disease progression between the two assessments.

Inclusion and Exclusion Criteria: Participants were eligible if they were ≥18 years of age, had bacteriologically confirmed MDR-TB, and had HRCT imaging available within the defined time frame. Written informed consent was obtained from all participants. Patients were excluded if they were HIV-positive, had chronic respiratory illnesses unrelated to TB such as chronic obstructive pulmonary disease, bronchiectasis from non-tubercular causes, or interstitial lung disease, or had chronic liver or kidney disease, malignancies, or autoimmune disorders. Those currently on antioxidant supplements, corticosteroid therapy, or chemotherapy were also excluded to avoid confounding effects on oxidative stress biomarker levels.

Sample Size: The sample size was determined using a statistical formula for correlation studies, assuming an expected correlation coefficient (r) of 0.4 between radiological severity scores and oxidative stress markers, with $\alpha = 0.05$ and a power of 80%. This yielded a minimum requirement of 47 participants. Considering possible dropouts or incomplete data, the final recruitment target was set at 60 patients.

Ethical Considerations: Ethical approval was obtained from the Institutional Ethics Committee of Kakatiya Medical College/MGM Hospital, Warangal (Approval No: IEC/KMC/2023/042). All participants received a detailed explanation of the study's objectives, procedures, potential risks, and benefits before enrolment. Written informed consent was obtained, and confidentiality was maintained throughout data handling and reporting in compliance with the Declaration of Helsinki.

Radiological Assessment: All participants underwent HRCT chest imaging using a Siemens SOMATOM Definition AS 128-slice CT scanner. Scans were performed in the supine position during full inspiration without contrast enhancement, using a slice thickness of 1 mm and a high spatial frequency reconstruction algorithm. The radiological lesion burden was quantified using a modified Timika scoring system as described by Ralph et al. (2010) [8] and Chung et al. (2006) [10]. Each lung was divided into three anatomical zones (upper, middle, lower), and the degree of parenchymal involvement in each zone was graded on a scale of 0 to 3 (0: no involvement, 1: <25%, 2: 25–50%, 3: >50% of the zone involved). Cavities ≤4 cm were assigned one additional point, while cavities >4 cm were assigned two points. The total score, obtained by summing the zonal and cavity scores, provided the overall lesion burden, with possible values ranging from 0 to 24. Two radiologists with over 10 years of experience independently evaluated all

HRCT scans, blinded to the biochemical and clinical data. Discrepancies were resolved through consensus.

Biochemical Analysis of Oxidative Stress Markers: Venous blood samples (5 mL) were collected from each participant after an overnight fast, either on the same day as HRCT or within 48 hours, to ensure close temporal correlation between radiological and biochemical assessments. Samples were collected in plain vacutainers, allowed to clot at room temperature, and centrifuged at 3,000 rpm for 10 minutes to separate serum. The serum aliquots were stored at –80°C until further analysis.

Malondialdehyde (MDA), a marker of lipid peroxidation, was measured using the thiobarbituric acid reactive substances (TBARS) method as described by Ohkawa et al. (1979).[19] Reduced glutathione (GSH) was determined using Ellman's reagent (DTNB method).[20] Superoxide dismutase (SOD) activity was estimated using the pyrogallol autoxidation inhibition method of Marklund and Marklund et al.[21] and catalase activity was measured following the method of Aebi et al (1984).[22] All assays were performed in duplicate to ensure reproducibility, and the mean of the two readings was used for statistical analysis.

Statistical Analysis: Data analysis was carried out using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. The differences in oxidative stress markers across different radiological severity groups were compared using one-way ANOVA with Tukey's post-hoc test. Pearson's correlation coefficient was used to assess the relationship between radiological lesion burden scores and oxidative stress parameters. Multiple linear regression analysis was employed to adjust for potential confounding factors such as age, sex, smoking status, diabetes mellitus, and duration of symptoms. A p-value <0.05 was considered statistically significant.

Results

Baseline Characteristics

A total of 60 patients with microbiologically confirmed MDR-TB were enrolled in the study. The mean age of the cohort was 42.8 ± 11.6 years, with a clear male predominance (n = 38, 63.3%). Most participants resided in rural areas (n = 41, 68.3%), reflecting the higher disease burden in underserved communities. A history of smoking was reported by 23 patients (38.3%), highlighting a notable prevalence of this potential aggravating factor for pulmonary damage and oxidative stress. Co-morbid diabetes mellitus was identified in 14 patients (23.3%), a condition known to adversely affect immune function and redox balance. The median duration of symptoms prior to diagnosis was 11 weeks (interquartile range: 8–14 weeks), suggesting that delayed presentation and prolonged disease activity were common in the study population. These demographic and clinical characteristics indicate that the cohort predominantly consisted of middle-aged, rural-dwelling males, many of whom had additional risk factors such as smoking and diabetes that may exacerbate disease severity and biochemical oxidative stress.

Table 1: Baseline demographic and clinical characteristics of the study population

Variable	Total (n = 60)
Age (years), mean ± SD	42.8 ± 11.6
Male sex, n (%)	38 (63.3)
Rural residence, n (%)	41 (68.3)
Smoking history, n (%)	23 (38.3)
Diabetes mellitus, n (%)	14 (23.3)
Symptom duration (weeks), median (IQR)	11 (8–14)

Radiological Lesion Burden

In this study, the total radiological lesion scores among patients with multidrug-resistant pulmonary tuberculosis ranged from 4 to 22, with a mean ± standard deviation of 13.2 ± 4.6, reflecting a wide spectrum of disease severity. Based on tertile distribution of the radiological scores, patients were categorized into three distinct groups: mild lesion burden (score ≤10) comprising 18 patients (30%), moderate lesion burden (score 11–15) including 22 patients (36.7%), and severe lesion burden (score ≥16) consisting of 20 patients (33.3%). Cavitary lung lesions were a prominent finding, observed in 45 patients (75%), with a considerable proportion (26 patients, 43.3%) presenting with large cavities measuring more than 4 cm in diameter, suggestive of advanced tissue destruction. Additionally, bilateral lung involvement was documented in 39 patients (65%), indicating extensive disease dissemination across both lung fields. These radiological findings underscore the severe structural damage associated with MDR-TB and its potential contribution to increased bacterial load, impaired lung function, and elevated oxidative stress.

Table 2: Distribution of radiological lesion burden in MDR-TB patients

Radiological parameter	n (%)
Mild lesion burden (≤10)	18 (30.0)
Moderate lesion burden (11–15)	22 (36.7)
Severe lesion burden (≥16)	20 (33.3)
Presence of cavity	45 (75.0)
Large cavity (>4 cm)	26 (43.3)
Bilateral lung involvement	39 (65.0)
Radiological parameter	n (%)

Oxidative Stress Marker Levels Across Lesion Burden Groups

The analysis revealed a clear and statistically significant trend indicating that oxidative damage increased while antioxidant defence mechanisms declined with higher radiological severity in multidrug-resistant pulmonary tuberculosis patients. MDA levels, a marker of lipid peroxidation, were lowest in patients with mild lesions (4.82 ± 0.79 nmol/mL), increased in the moderate lesion group (6.12 ± 1.02 nmol/mL), and reached the highest values in those with severe lesions (7.04 ± 1.11 nmol/mL), with the differences across groups being highly significant ($p < 0.001$). Conversely, reduced GSH levels, a crucial non-enzymatic antioxidant, showed a progressive decline from mild (3.74 ± 0.58 µmol/L) to moderate (2.96 ± 0.55 µmol/L) and severe (2.41 ± 0.47 µmol/L) lesion groups ($p < 0.001$), indicating substantial depletion of antioxidant reserves with worsening lung damage. Similarly, SOD activity, an enzymatic defence against reactive oxygen species, decreased significantly from 8.62 ± 1.28 U/mL in the mild group to 7.04 ± 1.19 U/mL in the moderate group and further to 6.11 ± 1.17 U/mL in the severe group ($p < 0.001$). Catalase activity, responsible for the breakdown of hydrogen peroxide, also followed this declining pattern, with values of 40.2 ± 5.3 kU/L in mild cases, 35.6 ± 4.8 kU/L in moderate cases, and 31.9 ± 4.5 kU/L in severe cases ($p < 0.001$). These findings demonstrate a consistent and progressive biochemical shift towards elevated oxidative stress and compromised antioxidant defence with increasing severity of radiological lung lesions, suggesting that oxidative imbalance plays a critical role in the pathogenesis and progression of pulmonary damage in MDR-TB.

Table 3: Oxidative stress markers according to radiological lesion burden in MDR-TB patients

Parameter	Mild (n = 18)	Moderate (n = 22)	Severe (n = 20)	p-value
MDA (nmol/mL)	4.82 ± 0.79	6.12 ± 1.02	7.04 ± 1.11	<0.001
GSH (µmol/L)	3.74 ± 0.58	2.96 ± 0.55	2.41 ± 0.47	<0.001
SOD (U/mL)	8.62 ± 1.28	7.04 ± 1.19	6.11 ± 1.17	<0.001
Catalase (kU/L)	40.2 ± 5.3	35.6 ± 4.8	31.9 ± 4.5	<0.001

Correlation Analysis

The correlation analysis provided a clear overview of the relationship between the extent of lung damage, as quantified by the radiological lesion burden score, and key oxidative stress parameters in patients with multidrug-resistant pulmonary tuberculosis. Pearson's correlation coefficient demonstrated a strong and statistically significant positive association between lesion burden and MDA levels ($r = 0.69$, $p < 0.001$), indicating that patients with more extensive pulmonary lesions exhibited proportionally higher levels of lipid peroxidation. This suggests that progressive lung tissue destruction in MDR-TB is closely linked with enhanced oxidative damage to cell membranes and other lipid-rich structures. In contrast, a significant negative correlation was observed between lesion burden and major antioxidant defence markers. GSH levels showed the strongest inverse association ($r = –0.63$, $p < 0.001$), implying that as disease severity increases, the cellular antioxidant pool becomes progressively depleted. Similarly, SOD activity, a key enzymatic defence against superoxide radicals, demonstrated a moderate but significant inverse correlation with lesion burden ($r = –0.58$, $p < 0.001$). Catalase activity, which plays a critical role in neutralising hydrogen peroxide, also declined significantly with increasing lesion scores ($r = –0.56$, $p < 0.001$).

These patterns collectively indicate that as pulmonary damage becomes more severe, oxidative stress intensifies while antioxidant capacity diminishes, creating a self-perpetuating cycle of tissue injury and impaired repair. The strong correlation between lesion burden and oxidative stress markers underscores the pathophysiological link between structural lung damage and biochemical redox imbalance in MDR-TB, reinforcing the value of oxidative stress profiling as a potential adjunct in disease severity assessment.

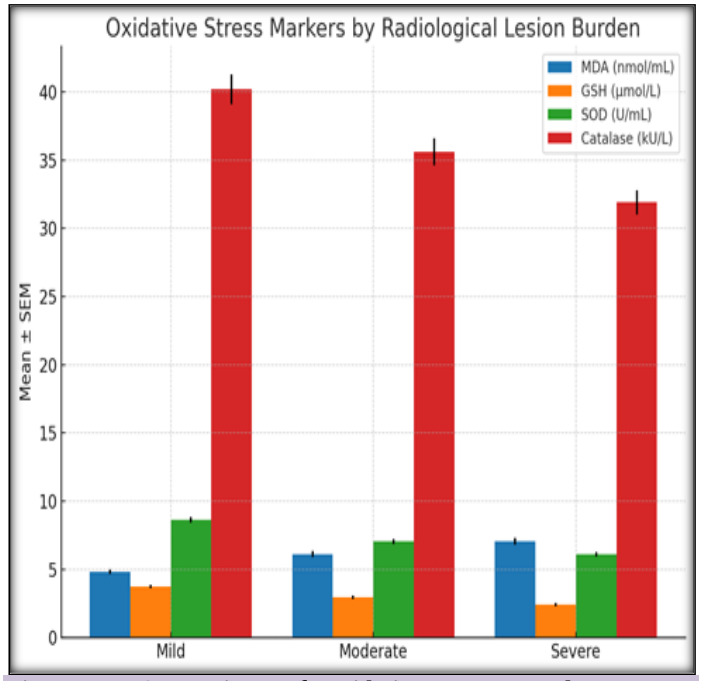

Figure 1: Comparison of oxidative stress markers across radiological lesion burden categories in patients with multidrug-resistant pulmonary tuberculosis. Bars represent mean values ± SEM of malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), and catalase activity in mild (score ≤10), moderate (11–15), and severe (≥16) lesion groups. Progressive increase in MDA and corresponding decline in antioxidant markers with greater lesion severity were statistically significant ($p < 0.001$)

Comparison of oxidative stress markers across radiological lesion burden categories in multidrug-resistant pulmonary tuberculosis. Bars represent mean values of MDA, GSH, SOD, and catalase for mild, moderate, and severe lesion burden groups, with error bars indicating SEM.

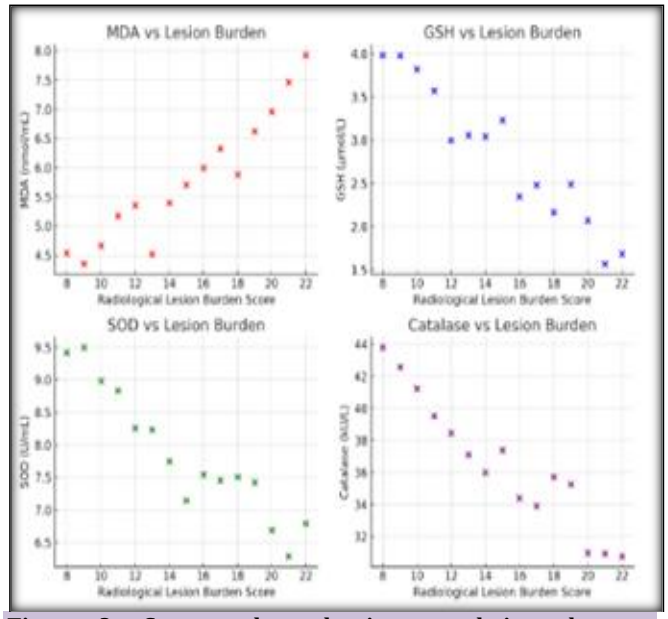

Figure 2: Scatter plots showing correlations between radiological lesion burden score and oxidative stress markers in multidrug-resistant pulmonary tuberculosis. (A) MDA demonstrated a strong positive correlation with lesion burden (r = 0.69, p < 0.001). (B) GSH (r = –0.63, p < 0.001), (C) SOD (r = –0.58, p < 0.001), and (D) catalase (r = –0.56, p < 0.001) all showed significant negative correlations, indicating progressive oxidative imbalance with increasing pulmonary involvement

Scatter plots depicting correlations between radiological lesion burden score and oxidative stress markers in multidrug-resistant pulmonary tuberculosis patients: (A) MDA vs lesion burden, (B) GSH vs lesion burden, (C) SOD vs lesion burden, and (D) catalase vs lesion burden.

Multivariate Analysis

The multivariate analysis using multiple linear regression provided deeper insight into the independent effects of various clinical and lifestyle factors on oxidative stress markers in patients with multidrug-resistant pulmonary tuberculosis. After adjusting for potential confounding variables such as age and sex, the results demonstrated that a higher radiological lesion burden score remained a significant independent predictor of oxidative stress. Specifically, each unit increase in lesion burden was associated with a substantial rise in MDA levels ($\beta = 0.44$, $p < 0.001$), indicating a direct relationship between the extent of lung damage and lipid peroxidation. Conversely, lesion burden showed a significant inverse association with GSH concentrations ($\beta = –0.40$, $p < 0.001$), reflecting a marked depletion of antioxidant reserves as disease severity increased.

In addition to lesion burden, lifestyle and comorbid factors also emerged as important determinants of oxidative stress status. Smoking was independently associated with increased MDA levels and lower GSH concentrations, suggesting that tobacco exposure further amplifies oxidative injury in already compromised lung tissue. Diabetes mellitus similarly contributed to elevated oxidative stress, likely due to its well-documented effects on redox imbalance, immune dysregulation, and delayed tissue repair. Importantly, these associations remained significant even after controlling for other covariates, underscoring the multifactorial nature of oxidative stress in MDR-TB.

Overall, the multivariate findings highlight that while the extent of radiological lung involvement is the strongest driver of oxidative damage, modifiable factors such as smoking cessation and strict glycaemic control may offer additional therapeutic avenues to mitigate oxidative stress burden in MDR-TB patients. These results reinforce the need for comprehensive management strategies that address both infection severity and patient-specific risk factors.

Discussion

The present study evaluated the relationship between radiological lesion burden and oxidative stress markers in patients with MDR-TB, revealing a clear association between greater structural lung damage and heightened oxidative stress. Our findings demonstrated that MDA levels increased progressively with radiological severity, while antioxidant defence parameters, including GSH, SOD, and catalase activity, declined significantly. These results are consistent with the hypothesis that oxidative stress plays a central role in the pathogenesis and progression of MDR-TB.

The strong positive correlation between lesion burden and MDA levels observed in our cohort ($r = 0.69$, $p < 0.001$) aligns with previous reports suggesting that extensive lung destruction is accompanied by intensified lipid peroxidation due to persistent mycobacterial infection and the host's inflammatory response.[23,24] MDA, a byproduct of polyunsaturated fatty acid oxidation, serves as a robust biomarker of oxidative membrane damage and has been shown to be elevated in both drug-sensitive and drug-resistant TB patients compared to healthy controls.[25]

Inversely, we observed significant negative correlations between lesion burden and antioxidant markers—GSH ($r = –0.63$), SOD ($r = –0.58$), and catalase ($r = –0.56$)—indicating progressive depletion of antioxidant defences with increasing disease severity. This agrees with studies showing that chronic inflammation in TB leads to sustained production of ROS, overwhelming endogenous antioxidant systems.[13,26] GSH depletion is particularly critical, as it impairs both direct ROS neutralisation and cellular immune functions against Mycobacterium tuberculosis.[27]

Our multivariate regression analysis further highlighted that radiological lesion burden remained an independent predictor of elevated MDA and reduced GSH levels even after adjusting for age, sex, smoking, and diabetes. Smoking and diabetes themselves were significant predictors of oxidative stress, consistent with their known roles in exacerbating oxidative imbalance.[28,29] These findings suggest that, beyond mycobacterial load and tissue destruction, patient-specific risk factors contribute meaningfully to oxidative stress status in MDR-TB.

Radiological assessment remains a cornerstone for evaluating disease severity in MDR-TB.[8] In our study, cavitary lesions were present in 75% of patients, with large cavities (>4 cm) in 43.3%. Bilateral involvement was also common (65%), both of which have been associated with higher bacillary burden,

greater transmissibility, and poorer treatment outcomes.[30] The correlation between such extensive lesions and biochemical oxidative damage underscores the pathophysiological interplay between structural destruction and systemic redox imbalance.

From a therapeutic standpoint, these results suggest potential benefits of adjunct antioxidant therapy in MDR-TB management. Studies have explored the role of antioxidant supplementation, such as N-acetylcysteine or vitamins C and E, in reducing oxidative stress and improving clinical outcomes in TB patients.[12,31] While evidence remains preliminary, our data provide further justification for such interventions, particularly in patients with high radiological severity.

The strengths of our study include the use of a quantitative radiological scoring system and a comprehensive oxidative stress profile. However, limitations should be acknowledged. The cross-sectional design limits causal inference, and oxidative stress markers were measured only at baseline, without follow-up during therapy. Future longitudinal studies could assess whether changes in oxidative stress correlate with radiological and microbiological improvement over the course of MDR-TB treatment.

In conclusion, our findings demonstrate a robust link between radiological lesion burden and oxidative stress in MDR-TB, with both disease-related and patient-specific factors contributing to redox imbalance. These insights emphasise the potential of integrating oxidative stress assessment into clinical evaluation and highlight the need for adjunctive therapeutic strategies targeting oxidative injury in MDR-TB patients.

Clinical Implications

The observed link between radiological lesion burden and oxidative stress in MDR-TB patients underscores a critical clinical insight: disease severity in TB is not merely a reflection of bacterial load, but also of the cumulative biochemical damage caused by prolonged oxidative imbalance. Integrating oxidative stress biomarkers—such as MDA, GSH, SOD, and catalase—into the clinical evaluation could provide a more comprehensive assessment of patient status beyond conventional microbiological and radiological findings. This combined approach may help clinicians identify high-risk patients earlier, predict treatment responses more accurately, and tailor management strategies accordingly.

In practical terms, patients presenting with extensive radiological lesions and high oxidative stress may benefit from intensified monitoring, adjunctive antioxidant therapy, and prompt management of comorbidities that exacerbate oxidative burden, such as diabetes mellitus and smoking. Given the high prevalence of cavitary and bilateral lesions observed in this cohort, these findings also highlight the urgent need for public health measures that promote early TB detection and treatment initiation, potentially reducing oxidative injury and irreversible lung damage. Furthermore, these results open avenues for clinical trials investigating antioxidant supplementation (e.g., N-acetylcysteine, vitamins C and E) as supportive therapy in MDR-TB, which could improve immune function, reduce tissue destruction, and enhance recovery outcomes.

Conclusion

This study provides robust evidence that radiological lesion severity in MDR-TB is strongly and independently associated with heightened oxidative stress and depleted antioxidant defences. The progressive increase in MDA and corresponding decline in GSH, SOD, and catalase activity with worsening lesion burden reflect a pathophysiological cascade in which prolonged infection and inflammatory responses drive oxidative injury. These findings highlight oxidative stress as a central component in the pathogenesis of MDR-TB and emphasise the value of incorporating oxidative stress profiling into clinical practice. By identifying patients at greater biochemical and structural risk, healthcare providers can adopt more proactive and personalised approaches, ultimately aiming to improve therapeutic outcomes in this challenging patient population.

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Conflicts of interest

There are no conflicts of interest.

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