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Original Article

Effect of Smog-Induced Pollution on the Biochemical Profile Among Pulmonary Tuberculosis Patients in the Peer Mahal Region

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ABSTRACT

Background: Pulmonary tuberculosis is associated with systemic inflammatory and biochemical disturbances that may be clinically relevant in regions affected by seasonal smog. Objective: To compare nutritional, haematological, inflammatory, hepatic and renal parameters between PTB patients and healthy controls in Peer Mahal and explore associations between regional air quality and selected haematological markers. Methods: This hospital-based case-control study included 40 participants: 20 PTB patients and 20 apparently healthy controls. Laboratory parameters were compared using independent-samples tests, while exploratory Pearson correlations assessed associations between regional AQI and haematological variables among PTB patients. Results: PTB patients had lower BMI (18.9±1.7 vs. 23.4±1.9 kg/m²) and haemoglobin (11.3±1.1 vs. 14.6±0.7 g/dL), and higher WBC, platelet count, ESR, ALT, AST, ALP, bilirubin, urea and creatinine than controls (all $p<0.001$ after recalculation). Regional AQI was significantly correlated with ESR ($r=0.55$; 95% CI: 0.14–0.80; $p=0.012$), but not with haemoglobin, WBC or platelet count. Conclusion: PTB patients exhibited substantial nutritional, inflammatory and biochemical abnormalities. The regional AQI–ESR association was exploratory and does not establish a causal effect of smog because exposure was not measured individually. Keywords: air quality; biochemical profile; haematological parameters; Peer Mahal; pulmonary tuberculosis; smog.

INTRODUCTION

Tuberculosis remains a major cause of infectious disease-related morbidity and mortality worldwide, with the greatest burden occurring in low- and middle-income countries. Pulmonary tuberculosis (PTB), caused by Mycobacterium tuberculosis, constitutes the predominant form of the disease and remains an important public-health challenge in Pakistan. Poverty, household crowding, malnutrition, delayed diagnosis and limited access to healthcare contribute to continued transmission and adverse clinical outcomes (1,2). The clinical expression and progression of tuberculosis are also influenced by interactions among microbial characteristics, host immune responses, nutritional status and environmental exposures (3).

Ambient air pollution has emerged as a potentially important environmental determinant of respiratory health and infectious disease susceptibility. Seasonal smog is particularly severe across Punjab, Pakistan, where emissions from agricultural residue burning, motor vehicles, industries and domestic fuel combustion combine with unfavourable meteorological conditions to increase concentrations of fine and coarse particulate matter (4,5). Particulate matter with an aerodynamic diameter of ≤2.5 μm or ≤10 μm can enter the respiratory tract, impair mucociliary clearance, disrupt epithelial-barrier integrity and promote oxidative and inflammatory responses (6–8). These effects may be clinically relevant to

individuals with active pulmonary disease, whose respiratory and systemic physiological reserves are already compromised.

Epidemiological studies have reported associations between ambient particulate-matter exposure and tuberculosis incidence, recurrence and mortality (1,9–13). Air pollutants may impair macrophage activity, alter innate immune responses and increase oxidative stress, potentially reducing the host's capacity to contain or eliminate M. tuberculosis (7,14). Airborne particulate matter may also contribute to persistent respiratory inflammation and interact with other environmental and microbial exposures, although the precise mechanisms and clinical importance of these pathways remain incompletely understood (14,15). The available evidence therefore supports a possible relationship between poor air quality and tuberculosis burden but does not establish that regional smog independently causes biochemical abnormalities in individual patients.

Pulmonary tuberculosis is itself associated with substantial haematological, inflammatory, nutritional and biochemical disturbances. Anaemia, leukocytosis, thrombocytosis and elevated erythrocyte sedimentation rate may occur as consequences of chronic infection, altered iron metabolism and sustained immune activation (16,17). Abnormal liver and renal biochemical markers may reflect systemic inflammation, nutritional deficiencies, disease severity, dehydration or adverse effects of anti-tuberculosis therapy. Oxidative stress, characterised by increased lipid peroxidation and reduced antioxidant capacity, has also been documented among patients with active tuberculosis and during treatment (18–20). Because air pollution can induce similar inflammatory and oxidative pathways, patients with PTB living in highly polluted environments may experience an additional physiological burden, although this possibility requires careful investigation using appropriately measured individual exposure.

Previous studies have primarily examined population-level associations between air pollution and tuberculosis incidence, geographical distribution, recurrence or mortality (1,9–13,21–23). Other investigations have evaluated biochemical and haematological abnormalities among patients with tuberculosis without incorporating environmental air-quality information (16–20). Consequently, comparatively little evidence is available regarding the biochemical profiles of PTB patients residing in areas affected by seasonal smog, particularly in semi-urban and rural regions of Pakistan. Existing findings may not be directly generalisable across settings because pollution sources, treatment patterns, nutrition, occupational exposure, indoor fuel use, socioeconomic conditions and healthcare access vary considerably among populations.

Pakistan experiences recurrent winter smog episodes during which particulate-matter concentrations frequently exceed recommended air-quality levels (24,25). The potential health consequences may be particularly relevant among nutritionally vulnerable patients and individuals with chronic or infectious respiratory disease. Low body mass index, smoking, diabetes, socioeconomic deprivation and indoor biomass-fuel exposure have independently been associated with impaired immunity and adverse tuberculosis outcomes and may also modify responses to environmental pollution (26–29). These factors demonstrate that biochemical abnormalities observed among PTB patients in polluted environments cannot automatically be attributed to smog alone and should be interpreted in relation to disease status, treatment exposure and other host characteristics.

Despite increasing concern regarding smog-related respiratory and cardiovascular morbidity in Pakistan, limited regional research has examined laboratory abnormalities among patients with pulmonary tuberculosis during periods of poor ambient air quality (30,31). Comparative evaluation of haematological, hepatic and renal parameters may provide useful information about the systemic physiological burden experienced by PTB patients living in pollution-affected areas. The present study therefore aimed to compare the nutritional, haematological, inflammatory, hepatic and renal profiles of patients with pulmonary tuberculosis and apparently healthy controls residing in Peer Mahal, Punjab,

during a seasonal smog period. It also explored associations between regional Air Quality Index measurements and selected haematological parameters among the PTB group.

MATERIALS AND METHODS

A hospital-based observational case-control study was conducted at Tehsil Headquarters Hospital, Peer Mahal, District Toba Tek Singh, Punjab, Pakistan. The study compared the biochemical and haematological profiles of patients with pulmonary tuberculosis and apparently healthy individuals residing in the same geographical region during a period affected by seasonal smog. Regional air-quality information was incorporated to characterise ambient environmental conditions during the study period and to support exploratory assessment of associations between air quality and selected laboratory parameters. Because both groups resided in the same region, the principal comparison was based on PTB status rather than on an experimentally assigned or individually measured difference in smog exposure.

The study included 40 participants, comprising 20 patients with pulmonary tuberculosis and 20 apparently healthy controls. PTB patients were recruited from the outpatient and tuberculosis clinics of the hospital. Eligible participants were 18–60 years of age and had resided in the Peer Mahal region for at least one year. Patients were either newly diagnosed or receiving standard anti-tuberculosis treatment. Pulmonary tuberculosis was diagnosed in accordance with national tuberculosis-control procedures using sputum smear microscopy, GeneXpert MTB/RIF testing and/or clinical and radiological assessment, as clinically applicable (32). Cases were subsequently classified according to whether they were GeneXpert-positive or diagnosed on clinical and radiological grounds.

Healthy controls were recruited from the same geographical region to reduce major differences in background environmental conditions. Controls had no reported history of tuberculosis or chronic hepatic, renal, infectious or metabolic disease. Individuals with extrapulmonary tuberculosis, chronic liver or kidney disease, diabetes mellitus, cardiovascular disease, autoimmune disease, malignancy, pregnancy, lactation, active smoking, alcohol consumption or long-term medication use other than anti-tuberculosis therapy were excluded. These criteria were applied to reduce the influence of major clinical factors that could independently alter haematological, hepatic or renal biomarkers.

Demographic and clinical information was obtained through a structured questionnaire and review of available hospital records. The collected information included age, sex, residential history, medical history and potential occupational or environmental exposure to vehicular emissions, agricultural residue burning and seasonal smog. Body mass index was calculated as body weight in kilograms divided by height in metres squared. Information concerning tuberculosis diagnosis and disease characteristics was obtained from clinical and laboratory records.

Ambient environmental conditions were characterised using regional monitoring information covering November 2025 to February 2026. The environmental variables included monthly concentrations of particulate matter with aerodynamic diameters of ≤10 μm and ≤2.5 μm, Air Quality Index category, temperature, relative humidity, rainfall and wind speed. These data were used to describe the intensity and seasonal pattern of ambient pollution in the study region. Because exposure was based on regional rather than personal monitoring, the environmental measurements represented area-level ambient conditions and were not considered direct measurements of each participant's total individual exposure.

Approximately 5 mL of venous blood was collected aseptically from each participant by trained laboratory personnel. Blood intended for haematological testing was transferred into ethylenediaminetetraacetic acid-containing tubes, whereas samples intended for biochemical analysis were collected in plain tubes. Serum was separated by centrifugation at 3,000 revolutions per minute for 10 minutes and maintained under recommended laboratory conditions until analysis (33).

Haematological assessment included haemoglobin concentration, red blood cell count, total leukocyte count, differential leukocyte count and platelet count using an automated haematology analyser. Systemic inflammatory activity was assessed using the erythrocyte sedimentation rate. Hepatic biochemical assessment included serum alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total bilirubin and albumin. Renal function was assessed using serum urea and creatinine measured through standard enzymatic or colorimetric laboratory methods. Routine internal quality-control procedures were applied during specimen processing and laboratory analysis to support analytical consistency.

Data were analysed using IBM SPSS Statistics version 25.0. Continuous variables were summarised as mean and standard deviation, while categorical variables were presented as frequencies and percentages. Age, body mass index and laboratory parameters were compared between PTB patients and healthy controls using independent-samples t tests. Categorical characteristics, including sex distribution, were compared using the chi-square test where applicable. Pearson correlation analysis was used as an exploratory procedure to examine associations between Air Quality Index measurements and selected haematological parameters among patients with pulmonary tuberculosis. All statistical tests were two-tailed, and a p value below 0.05 was considered statistically significant.

Ethical approval was obtained from the Ethical Review Committee of Riphah International University, Faisalabad Campus, before commencement of the study. Written informed consent was obtained from each participant after explanation of the study purpose and procedures. Participation was voluntary, and participants were informed that they could withdraw without affecting their medical care. Personal information was kept confidential, and study data were used exclusively for research purposes.

RESULTS

The study included 40 participants, comprising 20 patients with pulmonary tuberculosis and 20 apparently healthy controls. The groups did not differ significantly in age or sex distribution. The mean age was 32.8 ± 9.4 years among PTB patients and 30.2 ± 6.1 years among controls, corresponding to a mean difference of 2.6 years (95% CI: −2.5 to 7.7; p=0.307). Males accounted for 60.0% of the PTB group and 55.0% of the control group (p=0.749).

Patients with PTB had a substantially lower mean body mass index than controls (18.9 ± 1.7 vs. 23.4 ± 1.9 kg/m²). The mean between-group difference was −4.5 kg/m² (95% CI: −5.7 to −3.3; p<0.001), with a large standardized effect size (Hedges' g=−2.45). Among the PTB patients, 15 (75.0%) were GeneXpert-positive, while five (25.0%) were diagnosed on clinical and radiological grounds. Seventeen patients (85.0%) had an ESR of at least 30 mm/hour.

Table 1. Demographic and clinical characteristics of the study participants

Variable	PTB patients (n=20)	Healthy controls (n=20)	Mean difference or test statistic	95% CI	p value	Hedges' g
Age, years	32.8 ± 9.4	30.2 ± 6.1	2.6	−2.5 to 7.7	0.307	0.32
Male sex, n (%)	12 (60.0)	11 (55.0)	χ^2=0.10	—	0.749	—
Female sex, n (%)	8 (40.0)	9 (45.0)	—	—	—	—
BMI, kg/m²	18.9 ± 1.7	23.4 ± 1.9	−4.5	−5.7 to −3.3	<0.001	−2.45
GeneXpert-positive, n (%)	15 (75.0)	—	—	—	—	—
Clinically and radiologically diagnosed, n (%)	5 (25.0)	—	—	—	—	—
ESR <30 mm/hour, n (%)	3 (15.0)	—	—	—	—	—
ESR 30–60 mm/hour, n (%)	9 (45.0)	—	—	—	—	—
ESR >60 mm/hour, n (%)	8 (40.0)	—	—	—	—	—

Values are mean ± SD unless otherwise indicated. Continuous variables were compared using Welch independent-samples t-tests. Sex distribution was compared using the Pearson chi-square test. CI,

confidence interval; BMI, body mass index; ESR, erythrocyte sedimentation rate; PTB, pulmonary tuberculosis.

Regional Air Quality and Meteorological Conditions

Regional monitoring data indicated persistent poor ambient air quality between November 2025 and February 2026. Mean PM_{10} concentration increased from 140.3 μg/m³ in November to 178.4 μg/m³ in January before decreasing to 150.2 μg/m³ in February. Mean $PM_{2·5}$ followed a similar pattern, increasing from 84.6 μg/m³ in November to 118.1 μg/m³ in January.

December and January were classified as unhealthy, while November and February were classified as unhealthy for sensitive groups. The highest particulate-matter concentrations coincided with the lowest reported temperature and wind speed and the highest relative humidity. These measurements describe regional ambient conditions and do not represent direct personal exposure measurements.

Table 2. Regional air quality and meteorological conditions from November 2025 to February 2026

Variable	November 2025	December 2025	January 2026	February 2026
Mean PM_{10}, μg/m³	140.3	165.5	178.4	150.2
Mean $PM_{2·5}$, μg/m³	84.6	105.3	118.1	89.5
AQI category	Unhealthy	Unhealthy	Unhealthy	Unhealthy
Temperature, ˚C	21.4	15.8	13.6	17.9
Relative humidity, %	62.3	66.4	69.8	65.2
Wind speed, km/hour	28.5	24.7	22.1	26.3
Rainfall, mm	2.4	5.2	8.1	6.3

AQI, Air Quality Index; $PM_{2·5}$, particulate matter with an aerodynamic diameter of ≤2.5 μm; PM_{10}, particulate matter with an aerodynamic diameter of ≤10 μm.

Haematological and Inflammatory Parameters

Patients with PTB had a lower mean haemoglobin concentration than healthy controls (11.3 ± 1.1 vs. 14.6 ± 0.7 g/dL). The mean difference was −3.3 g/dL (95% CI: −3.9 to −2.7; p<0.001), with a large standardized effect size (Hedges' g=−3.51).

Mean white blood cell count was 7.0 $\times 10^9$/L higher among PTB patients than controls (95% CI: 5.3 to 8.7; p<0.001; Hedges' g=2.63). Platelet count was also higher in the PTB group, with a mean difference of 110 $\times 10^9$/L (95% CI: 66.0 to 154.0; p<0.001; Hedges' g=1.61). The largest absolute inflammatory difference was observed for ESR, which was 46.0 mm/hour higher among PTB patients (95% CI: 36.9 to 55.1; p<0.001; Hedges' g=3.26).

Table 3. Comparison of haematological and inflammatory parameters between PTB patients and healthy controls

Parameter	PTB patients (n=20)	Healthy controls (n=20)	Mean difference	95% CI	p value	Hedges' g
Haemoglobin, g/dL	11.3 ± 1.1	14.6 ± 0.7	−3.3	−3.9 to −2.7	<0.001	−3.51
WBC, $\times 10^9$/L	14.2 ± 3.6	7.2 ± 0.8	7.0	5.3 to 8.7	<0.001	2.63
Platelets, $\times 10^9$/L	368 ± 92	258 ± 22	110	66.0 to 154.0	<0.001	1.61
ESR, mm/hour	54.6 ± 19.3	8.6 ± 3.2	46.0	36.9 to 55.1	<0.001	3.26

Values are mean ± SD. Mean differences are calculated as PTB minus control. Confidence intervals and p-values were derived using Welch independent-samples t-tests. ESR, erythrocyte sedimentation rate; PTB, pulmonary tuberculosis; WBC, white blood cell count.

Hepatic and Renal Biochemical Parameters

All reported hepatic biomarkers were higher among PTB patients than healthy controls. Mean ALT was 21.9 U/L higher in the PTB group (95% CI: 17.8 to 26.0; p<0.001; Hedges' g=3.37), while mean AST was 19.4 U/L higher (95% CI: 15.7 to 23.1; p<0.001; Hedges' g=3.31). The mean between-group difference in ALP was 54 U/L (95% CI: 37.2 to 70.8; p<0.001), and total bilirubin was 0.50 mg/dL higher among PTB patients (95% CI: 0.39 to 0.61; p<0.001).

Mean serum urea was 16.2 mg/dL higher in PTB patients than controls (95% CI: 12.8 to 19.6; p<0.001; Hedges' g=3.02). Mean creatinine was also higher by 0.38 mg/dL (95% CI: 0.28 to 0.48; p<0.001; Hedges' g=2.50). These findings indicate marked between-group biochemical differences but do not independently establish clinically diagnosed hepatic or renal dysfunction.

Table 4. Comparison of hepatic and renal biochemical parameters between PTB patients and healthy controls

Parameter	PTB patients (n=20)	Healthy controls (n=20)	Mean difference	95% CI	p value	Hedges' g
ALT, U/L	48.3 ± 8.2	26.4 ± 3.7	21.9	17.8 to 26.0	<0.001	3.37
AST, U/L	43.6 ± 7.5	24.2 ± 3.1	19.4	15.7 to 23.1	<0.001	3.31
ALP, U/L	196 ± 32	142 ± 18	54	37.2 to 70.8	<0.001	2.04
Total bilirubin, mg/dL	1.18 ± 0.22	0.68 ± 0.09	0.50	0.39 to 0.61	<0.001	2.92
Urea, mg/dL	40.8 ± 6.7	24.6 ± 3.2	16.2	12.8 to 19.6	<0.001	3.02
Creatinine, mg/dL	1.16 ± 0.18	0.78 ± 0.11	0.38	0.28 to 0.48	<0.001	2.50

Values are mean ± SD. Mean differences are calculated as PTB minus control. Confidence intervals and p-values were derived using Welch independent-samples t-tests. ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; PTB, pulmonary tuberculosis.

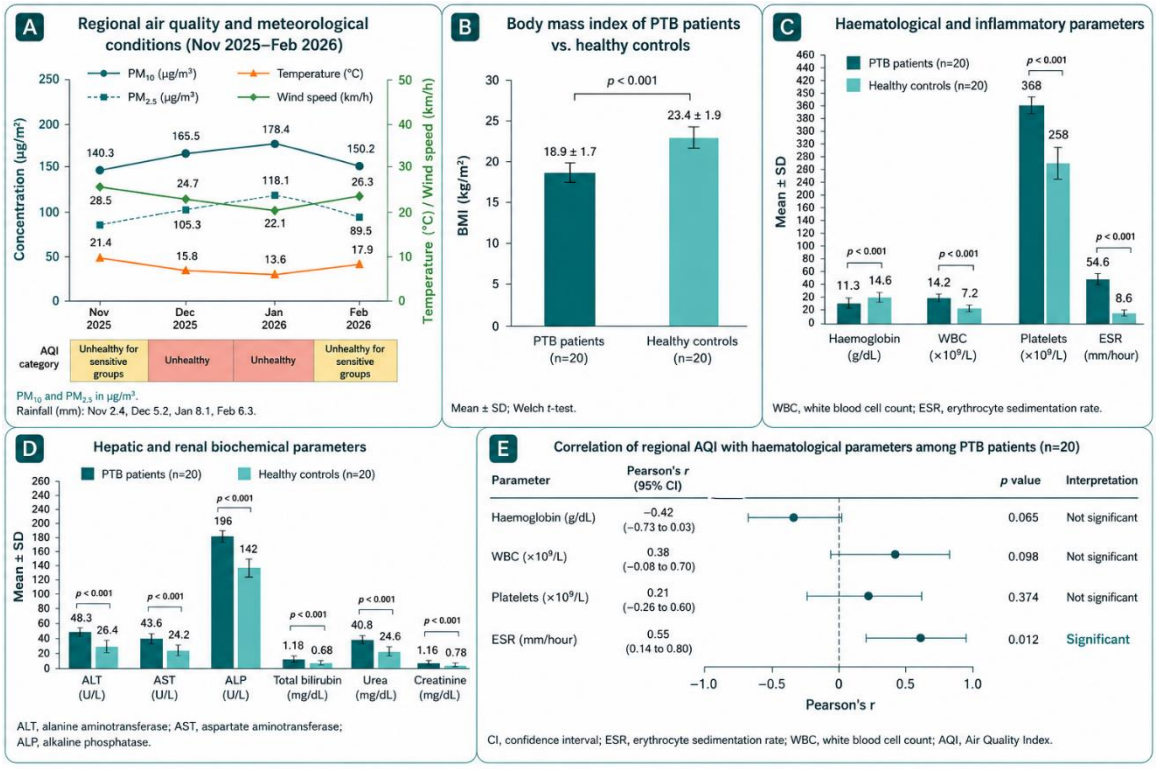

Figure 1 The multipanel figure summarizes regional winter air quality and the principal clinical findings. PM_{10} and $PM_{2.5}$ concentrations peaked in January 2026, while PTB patients showed significantly lower BMI and haemoglobin and markedly higher WBC, platelet count, ESR, hepatic enzymes, bilirubin, urea, and creatinine than healthy controls. In exploratory correlation analysis, regional AQI was significantly associated only with ESR (r=0.55, p=0.012), whereas correlations with haemoglobin, WBC, and platelet count were not statistically significant.

Exploratory Associations Between Air Quality and Haematological Parameters

Exploratory Pearson correlation analysis was reported for regional AQI and selected haematological parameters among 20 PTB patients. After recalculation using the stated coefficients and sample size, AQI showed a moderate inverse correlation with haemoglobin, although the association did not reach statistical significance (r=−0.42; 95% CI: −0.73 to 0.03; p=0.065).

The correlation between AQI and white blood cell count was positive but non-significant (r=0.38; 95% CI: −0.08 to 0.70; p=0.098). Platelet count was weakly and non-significantly correlated with AQI (r=0.21; 95% CI: −0.26 to 0.60; p=0.374). ESR demonstrated a moderate positive correlation with AQI (r=0.55; 95% CI: 0.14 to 0.80; p=0.012).

Table 5. Exploratory correlations between regional AQI and haematological parameters among PTB patients

Parameter	n	Pearson's r	95% CI	p value
Haemoglobin	20	−0.42	−0.73 to 0.03	0.065
White blood cell count	20	0.38	−0.08 to 0.70	0.098
Platelet count	20	0.21	−0.26 to 0.60	0.374
ESR	20	0.55	0.14 to 0.80	0.012

Confidence intervals were calculated using Fisher's z transformation. All p-values are two-tailed. AQI, Air Quality Index; ESR, erythrocyte sedimentation rate; PTB, pulmonary tuberculosis.

Overall, PTB patients exhibited lower BMI and haemoglobin levels and higher inflammatory, hepatic and renal biomarkers than healthy controls living in the same geographical region. The standardized between-group differences were large across most laboratory measures. Among the exploratory air-quality analyses, only ESR retained a statistically significant correlation with regional AQI after correction of the originally reported p-values. Because exposure was based on regional measurements rather than direct personal monitoring, these correlations should be interpreted as exploratory associations rather than evidence of an independent effect of smog.

DISCUSSION

The present study compared nutritional, haematological, inflammatory, hepatic and renal parameters between patients with pulmonary tuberculosis and apparently healthy controls residing in the same smog-affected region of Peer Mahal. PTB patients had markedly lower BMI and haemoglobin concentrations and higher white blood cell counts, platelet counts, ESR, liver enzymes, bilirubin, urea and creatinine. Most between-group differences were large in magnitude. In the exploratory air-quality analysis, regional AQI was significantly correlated only with ESR after correction of the originally reported statistics, whereas its associations with haemoglobin, white blood cell count and platelet count did not reach statistical significance. These findings primarily demonstrate systemic biochemical and inflammatory abnormalities associated with PTB status; they do not independently establish that ambient smog caused those abnormalities.

The substantially lower BMI observed among PTB patients is consistent with the established bidirectional relationship between tuberculosis and undernutrition. Active infection can increase metabolic demand, suppress appetite and promote catabolic weight loss, while inadequate nutritional status can impair cell-mediated immunity and reduce resistance to M. tuberculosis. Previous studies have similarly reported lower BMI among patients with active PTB and have identified poor nutritional status as a marker of more severe disease and less favourable outcomes (26,34). In the present study, the mean BMI difference of −4.5 kg/m² and large standardized effect indicate a clinically important nutritional disparity between groups. However, because dietary intake, socioeconomic status and treatment duration were not incorporated into the reported analysis, the relative contributions of infection, nutritional deprivation and treatment-related factors cannot be separated.

The lower haemoglobin concentration and higher inflammatory markers among PTB patients are also compatible with the systemic effects of active tuberculosis. Anaemia in tuberculosis may result from chronic inflammation, altered iron metabolism, nutritional deficiency, bone-marrow suppression and disease-related catabolism. Leukocytosis and thrombocytosis may reflect immune activation and acute-phase responses, while ESR is commonly elevated in active disease because of increased circulating inflammatory proteins (16,17). The large differences in haemoglobin, WBC, platelet count and ESR observed in this study support the presence of substantial inflammatory and haematological disturbance among PTB patients. These findings are broadly consistent with systematic evidence showing frequent anaemia and altered blood-cell indices in newly diagnosed tuberculosis (16).

Regional winter pollution may plausibly intensify inflammatory stress through oxidative and immune-mediated pathways. Fine particulate matter can penetrate the distal respiratory tract, impair epithelial and macrophage function, generate reactive oxygen species and stimulate inflammatory signalling (6–

8,35,36). Nevertheless, the present data provide only limited support for a direct air-quality relationship. After statistical correction, ESR remained moderately and positively associated with regional AQI, whereas the correlations with haemoglobin, WBC and platelets were non-significant. The ESR association is biologically plausible, but it should be interpreted cautiously because exposure was assigned from regional measurements rather than personal monitoring, the effective variation in exposure may have been limited, and the sample comprised only 20 PTB patients. Residual confounding by disease activity, treatment status, nutrition, occupation and indoor exposure may also explain part of the observed relationship.

The higher ALT, AST, ALP and bilirubin concentrations among PTB patients indicate a clear between-group hepatic biochemical difference. Liver abnormalities in PTB can arise from systemic inflammation, malnutrition, hepatic involvement, coexisting infection or exposure to anti-tuberculosis medicines. Drug-induced hepatotoxicity is particularly relevant because the study included both newly diagnosed patients and those receiving treatment, but treatment duration and regimen were not analysed separately. Previous studies have documented hepatic biochemical abnormalities among PTB patients and during anti-tuberculosis therapy (20,37). Accordingly, the elevated liver markers in the present study should not be attributed specifically to smog without accounting for treatment exposure and other clinical determinants.

Patients with PTB also had higher serum urea and creatinine concentrations than controls. These differences may reflect dehydration, altered protein metabolism, systemic inflammation, treatment-related nephrotoxicity or underlying physiological stress. Ambient particulate matter has been linked to endothelial dysfunction and systemic oxidative injury, which may affect renal physiology, but the present study was not designed to isolate this pathway. Moreover, higher group means do not alone establish clinically diagnosed renal impairment. Evaluation of estimated glomerular filtration rate, urinalysis, hydration status, treatment regimen and serial renal measurements would be required for a more clinically meaningful assessment.

The regional environmental data documented persistently poor ambient air quality during the winter study period, with PM_{10} and $PM_{2.5}$ concentrations peaking in January 2026. These findings are consistent with the broader environmental context of recurrent winter smog in Punjab, where emissions from crop-residue burning, traffic and industry interact with meteorological conditions that favour pollutant accumulation (4,5,24,25). Previous epidemiological studies and meta-analyses have reported associations between particulate-matter exposure and tuberculosis incidence, recurrence and mortality (1,9–13). The present study complements that evidence by describing laboratory abnormalities among PTB patients living in a pollution-affected area, but its design does not determine whether those abnormalities were greater than they would have been under cleaner environmental conditions.

The study has several strengths. Both groups were drawn from the same geographical area, reducing broad regional differences in climate and environmental context. The study also assessed multiple physiological domains, including nutritional status, haematological indices, inflammatory activity and hepatic and renal biomarkers. The integration of regional air-quality information provides useful environmental context, and the recalculated analyses improve the precision and transparency of the reported findings.

Several limitations must be considered. The small sample size and single-centre design restrict statistical precision and generalisability. Regional monthly pollution data were used instead of direct personal exposure monitoring, and the monitoring source, station characteristics and participant-level exposure linkage require clearer documentation. Both groups lived in the same region, so the case-control comparison primarily reflects PTB status rather than differential smog exposure. The inclusion of newly diagnosed and treated patients introduced clinical heterogeneity, particularly for hepatic and renal outcomes. Important confounders, including dietary intake, socioeconomic status, indoor fuel exposure, occupational exposure, second-hand smoke, disease severity and treatment duration, were not

incorporated into adjusted analyses. The cross-sectional nature of the measurements prevents temporal or causal inference, and oxidative-stress biomarkers and inflammatory cytokines were not directly measured.

Future studies should use larger multicentre samples, prospective follow-up and participant-level exposure assessment. Personal particulate-matter monitoring, residential geocoding, treatment stratification and repeated biochemical testing across high- and low-smog periods would better clarify temporal relationships. Direct measurement of oxidative-stress markers, cytokines and antioxidant capacity could also help determine whether environmental pollution contributes independently to the systemic inflammatory burden of tuberculosis.

CONCLUSION

Patients with pulmonary tuberculosis residing in Peer Mahal had substantially lower BMI and haemoglobin concentrations and higher inflammatory, hepatic and renal biomarkers than apparently healthy controls from the same region. These findings demonstrate a marked systemic physiological burden associated with PTB. Regional AQI was significantly associated only with ESR in the corrected exploratory analysis, while its relationships with haemoglobin, white blood cell count and platelet count were not statistically significant. Because exposure was based on regional rather than personal measurements and both groups lived in the same environment, the study does not establish that smog caused the observed biochemical abnormalities. Larger prospective studies with individual exposure monitoring and adjustment for treatment, nutrition and other confounders are required.

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