American Journal of Medicine and Medical Sciences 2025, 15(10): 3470-3473
DOI: 10.5923/j.ajmms.20251510.40

Biochemical and Immunological Parameters of Blood Serum of Patients with Tuberculosis in the Bukhara Region

Jalolitdin Sulton Nazarov

Department of Microbiology, Virology and Immunology, Bukhara State Medical Institute named after Abu Ali ibn Sino, Gijduvan St. 23, Bukhara, Uzbekistan

Abstract Objective: To characterize biochemical and immunological parameters in the blood serum of patients with a verified diagnosis of tuberculosis (TB) in the Bukhara region and to explore a multimarker model with emphasis on multidrug-resistant (MDR) and extensively drug-resistant (XDR) TB. Methods: We examined serum samples from 147 TB patients before initiation and after completion of anti-tuberculosis therapy at the Bukhara Regional Center of Phthisiology and Pulmonology; 20 practically healthy adults served as controls. Biochemical profiling (ALT, AST, glucose, creatinine, urea, total protein) and immunological assays (IgA, IgM, IgG, IgE; cytokines including IFNγ, IL-6, IL-1β, TNF-α) were performed using standard analyzers and ELISA. Results: Following therapy, ALT/AST elevations were common, consistent with treatment-related hepatocellular stress. Comorbidities were frequent—hypertension (33%), coronary heart disease (29%), angina (23%), and diabetes mellitus (27%). In the MDR/XDR subset (n=15), IFNγ and IL-6 were detected in all patients; IL-1β and TNF-α in 7 and 6 patients, respectively; anti-inflammatory cytokines were largely normal (IL-10 elevated in 2, IL-4 in none). Elevated immunoglobulins were observed predominantly for IgA (14/15), IgM (12/15), and IgE (12/15), with more modest IgG elevations (7/15). Conclusions: A composite of pro-inflammatory cytokines (IFNγ, IL-6, IL-1β) together with immunoglobulins (IgA, IgM, IgE) shows promise as a diagnostic and prognostic signature in MDR/XDR-TB. These markers may inform patient stratification and immunocorrective approaches alongside standard chemotherapy.

Keywords Tuberculosis, Biochemical markers, Cytokine profile, MDR-TB, XDR-TB, Immunoglobulins

1. Introduction

Tuberculosis (TB) remains among the most consequential infectious diseases, with billions living with latent infection and a substantial annual burden of active disease. Beyond microbiological confirmation, complementary biochemical and immunological markers can refine diagnosis, disease monitoring, and prognosis. Humoral responses—particularly serum immunoglobulins—show class- and antigen-specific patterns during active versus latent TB, and may modulate cellular immunity [1-3,7-9,11-13].

Cytokines orchestrate host-pathogen interactions in TB; dysregulated pro-inflammatory signaling (e.g., IFNγ, IL-6, TNF-α) can drive tissue damage and poor outcomes, whereas balanced responses are essential for control [5,6,17]. Serum biochemistry (e.g., acute-phase proteins, hepatocellular enzymes, renal indices) complements immune profiling by capturing systemic effects of disease and therapy [14-16].

* Corresponding author:
sultannazarov050@gmail.com (Jalolitdin Sulton Nazarov)
Received: Sep. 3, 2025; Accepted: Oct. 1, 2025; Published: Oct. 25, 2025
Published online at http://journal.sapub.org/ajmms

Here we report a single-center study from the Bukhara region evaluating serum biochemical parameters and a targeted panel of immunoglobulins and cytokines in TB patients before and after chemotherapy, with focused analyses in MDR/XDR subgroups. We propose a practical multimarker set for clinical use.

2. Materials and Methods

2.1. Study Design and Participants

We conducted a cross-sectional and longitudinal analyses of patients with bacteriologically confirmed TB treated at the Bukhara Regional Center of Phthisiology and Pulmonology (BrcPaP). A total of 147 patients provided venous blood samples at two time points: prior to initiation of anti-tuberculosis therapy and after completion of the treatment course. A control group of 20 practically healthy adults was also assessed.

Inclusion criteria: adult patients with verified TB diagnosis receiving care at BrcPaP and able to provide serum samples at the specified time points. Exclusion criteria: [to be specified, e.g., inability to provide consent, acute conditions precluding

phlebotomy, etc.].

Study period: 2022 – 2024 years.

2.2. Data Collection Instruments

Biochemical analyses were performed on a Biossays 240 plus automatic biochemical analyzer; erythrocyte sedimentation rate (ESR) was assessed on a Sysmex CA-600 series analyzer (Siemens). Leukocyte counts used an SL-1 manual counter (Kyiv, 1964) and a Zeiss Axiostar plus binocular optical microscope.

Immunological assays included enzyme-linked immunosorbent assays (ELISA) for IgA, IgM, IgG, IgE and selected cytokines using: ELISA Reader (DR-200B), ELISA Microplate Washer (DRW-320, Diatek), and ELISA Thermo Shaker (MB-100-2A, Allsheng); devices manufactured in 2023.

2.3. Procedures

Whole blood was collected between 07:00–08:00 while fasting, via antecubital venipuncture (5 mL) using disposable syringes. Samples were transported to the laboratory within 1.5 hours. After centrifugation at room temperature (Cence TDZ4K, 1500 rpm, 10–15 minutes), serum was aliquoted into sterile 2 mL PP cryovials using a filtered single-channel pipette (1000 μL tips). Serum aliquots were stored at −30 ℃ (Liebherr) until analysis.

Clinical biochemistry and ELISAs were performed according to reagent and instrument manufacturer instructions.

2.4. Ethical Considerations

The study protocol was approved by the Institutional Review Board of the Bukhara State Medical Institute (BSMI), Bukhara, Uzbekistan; approval code [1847/45 issued on [18 March 2023]. All procedures complied with the Declaration of Helsinki (2013). Written informed consent was obtained from all participants; data were anonymized prior to analysis.

2.5. Data Analysis

Analyses were conducted using [software, e.g., SPSS vXX or R v4.X]. Continuous variables are reported as mean ± standard error (SE) unless otherwise specified. Within-patient pre/post differences were assessed using paired tests (paired t-test for approximately normal data; Wilcoxon signed-rank otherwise). Categorical comparisons used χ^2 or Fisher's exact tests. Statistical significance was set at $p<0.05$ (two-tailed).

3. Results

3.1. Biochemical Parameters

Elevations in ALT and AST were common after therapy, consistent with antibiotic-related hepatocellular stress; the predominance of AST over ALT suggests concomitant myocardial involvement in a subset of patients.

Glycemic abnormalities were frequent: 51 patients had elevated glucose prior to treatment, 36 of whom had known diabetes mellitus. Low glucose levels occurred in association with cardiac, renal, or hepatic failure and in individuals with anemia or alcohol misuse.

Renal indices reflected heterogeneous patterns: a modest post-treatment decline in creatinine among men (possibly due to lower in-facility physical activity) and increased creatinine among women (potentially diet-related). One patient with nephrosclerosis and chronic renal failure exhibited markedly elevated creatinine and urea.

Nearly one-fifth of patients had elevated urea both before and after therapy, plausibly linked to reduced renal function and/or high-protein diet during hospitalization. Patients with hepatitis C generally showed reduced urea; 17 patients had confirmed hepatitis B/C.

Hypoproteinemia was common and may reflect immunodeficiency states with increased antibody production and/or hepatic, renal, or pancreatic involvement.

Table 1. Biochemical parameters of 147 BrcPaP patients before and after treatment (as reported)

Biochemical Indicators	Before Treatment (Increased Level) ± Error	Before Treatment (Reduced Level) ± Error	After Treatment (Increased Level) ± Error	After Treatment (Reduced Level) ± Error	Normal Range
ALT (<40 U/L)	19 ± 2	131 ± 4	33 ± 3	117 ± 2	<40 U/L
AST (<35 U/L)	20 ± 3	130 ± 5	36 ± 4	114 ± 3	<35 U/L
Glucose (32–61 mmol/L)	51 ± 6	7 ± 1	41 ± 5	19 ± 2	32–61 mmol/L
Creatinine (Male: 44–115 μmol/L)	6 ± 0.5	1 ± 0.2	2 ± 0.4	2 ± 0.4	44–115 μmol/L
Creatinine (Female: 44–97 μmol/L)	2 ± 0.3	9 ± 1.2	5 ± 0.8	8 ± 1.0	44–97 μmol/L
Urea (25–83 mmol/L)	29 ± 2.5	116 ± 5.1	26 ± 2.3	120 ± 5.4	25–83 mmol/L
Total Protein (66–85 g/L)	3 ± 0.5	46 ± 4	1 ± 0.3	58 ± 5	66–85 g/L

3.2. Comorbidities

Cardiovascular comorbidity was prevalent: hypertension in 49 patients (33%; 29 women [19.7%], 20 men [13.6%]); coronary heart disease in 43 (29.2%; 24 women [16.3%], 19 men [12.9%]); angina pectoris in 34 (23.1%).

Diabetes mellitus ranked second among comorbidities (40 patients; 27.2%; similar distribution across sexes). Hepatitis B/C was confirmed in 17 patients (11.5%).

3.3. Cytokine Profile in MDR/XDR Subset (n=15)

In patients with MDR-TB and XDR-TB (n=15), pro-inflammatory cytokines demonstrated the highest diagnostic yield: IFNγ and IL-6 were detected in all patients, IL-1β in 7, and TNF-α in 6.

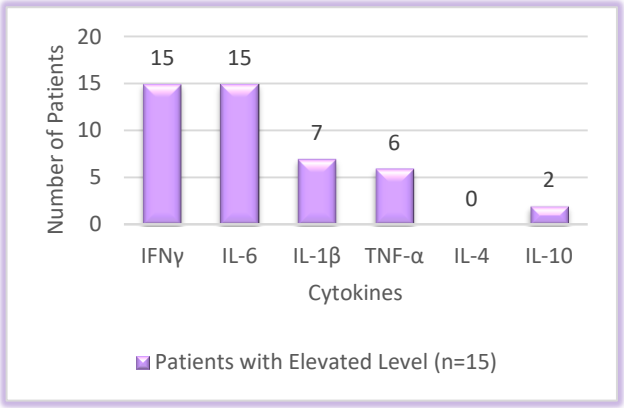


Figure 1. Cytokine profile (counts of patients with elevated levels) in MDR/XDR-TB (n=15)

Anti-inflammatory cytokines were largely within normal ranges: IL-4 was not elevated in any case, and IL-10 was elevated in two MDR cases.

3.4. Immunoglobulins in MDR/XDR Subset (n=15)

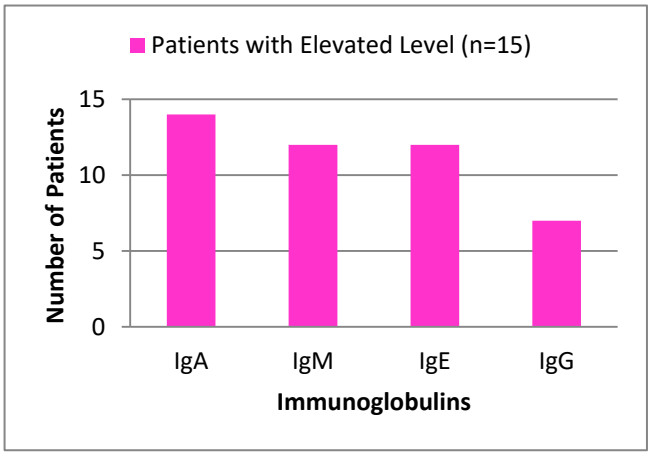


Figure 2. Immunoglobulin titer (counts of patients with elevated levels) in MDR/XDR-TB (n=15)

Elevated IgA was observed in 14/15 patients, followed by IgM and IgE (each 12/15). IgE showed considerable intra-individual variability across time points, potentially reflecting responses to chemotherapy. IgG elevations were less frequent (7/15), with normalization in some patients after treatment.

4. Discussion

This study demonstrates that routine biochemical parameters and targeted immunological markers provide complementary insights into TB pathobiology and treatment response. Post-therapy elevations in ALT/AST are consistent with known hepatic stress from anti-TB regimens and warrant monitoring. The high prevalence of cardiovascular disease and diabetes mellitus underscores a syndemic burden that can complicate management and outcomes.

The MDR/XDR subset revealed a robust pro-inflammatory signature dominated by IFNγ and IL-6, in line with established roles in TB immunity and pathology [5,6,17]. Serological markers—especially IgA, IgM, and IgE—showed discriminative potential, echoing prior reports that combinations of immunoglobulin classes against selected mycobacterial antigens can improve diagnostic performance [1-3,11,12].

Integrating these signals into a composite clinical algorithm could facilitate early risk stratification, therapeutic monitoring, and potentially guide immunocorrective strategies alongside chemotherapy.

5. Study Limitations

- Single-center design; limited generalizability beyond the Bukhara region.
- Incomplete standardization of timing for follow-up sampling; potential confounding from comorbidities and concomitant medications.
- Small MDR/XDR sample (n=15) limits power for subgroup analyses and dynamic (pre/post) modeling.
- Some laboratory reference intervals and unit notations require verification against the local laboratory's standards prior to resubmission (e.g., glucose and urea ranges).

6. Conclusions

Serum biochemical indices together with a focused panel of pro-inflammatory cytokines (IFNγ, IL-6, IL-1β) and immunoglobulins (IgA, IgM, IgE) may form a practical multimarker signature for TB—particularly MDR/XDR—supporting diagnosis, monitoring, and prognostication alongside standard therapy.

ACKNOWLEDGEMENTS

We thank the Bukhara Regional Center of Phthisiology and Pulmonology and the Bukhara branch of the Institute of Human Immunology and Genomics, Academy of Sciences of the Republic of Uzbekistan, for laboratory support and collaboration.

Authors' Contribution

JSN conceived the study, collected and processed samples, performed analyses, interpreted data, and drafted the manuscript.

Conflicts of Interest

The author declares no conflicts of interest.

Funding

No specific funding was received for this work. [If applicable, insert funding details here].

REFERENCES

[1] Alvarez N., Otero O., Camacho F., Borrero A., Tirado Y. et al. Passive administration of purified secretory IgA from human colostrum induces protection against Mycobacterium tuberculosis in a murine model of progressive pulmonary infection. BMC Immunology. 2013; 14: 1–4.

[2] Awoniyi D., Baumann R., Chegou N., Kriel B., Jacobs A. et al. Detection of a combination of serum IgG and IgA antibodies against selected mycobacterial targets provides promising diagnostic signatures for active TB. Oncotarget. 2017; 23: 37525–37537.

[3] Baumann R., Kaempfer S., Chegou N.N., Oehlmann W., Loxton A.G. Serologic diagnosis of tuberculosis by combining Ig classes against selected mycobacterial targets. Journal of Infection. 2014; 69(6): 581–589.

[4] Cambier C., Falkow S., Ramakrishnan L. Host evasion and exploitation schemes of Mycobacterium tuberculosis. Cell. 2014; 159(7): 1497–1509.

[5] Chernushenko E.F., Kadan L.P., Panasukova O.R., Petishkina V.N., Cygankova L.M. Cytokines in assessing the immune system in patients with pulmonary tuberculosis. Ukrainian Pulmonological Journal. 2010; (2): 39–43.

[6] Domingo-Gonzales R., Prince O., Cooper A., Khader S.A. Cytokines and chemokines in Mycobacterium tuberculosis infection. Microbiology Spectrum. 2016; 4(5): 1–37.

[7] Feris E., Encinales L., Awad C., Stern J., Tabansky I. et al. High levels of anti-tuberculin (IgG) antibodies correlate with the blocking of T-cell proliferation in individuals with high exposure to Mycobacterium tuberculosis. International Journal of Infectious Diseases. 2016; 43: 21–24.

[8] Galson J., Pollard A., Truck J., Kelly D. Studying the antibody repertoire after vaccination: practical applications. Trends in Immunology. 2014; 35(7): 319–331.

[9] Hur Y.-G., Kim A., Kang Y.A., Kim A.S., Kim D.Y. Evaluation of antigen-specific immunoglobulin G responses in pulmonary tuberculosis patients and contacts. Journal of Clinical Microbiology. 2015; 53(3): 904–909.

[10] Nazarov J.S. Features and causes of drug resistance in pathogenic Mycobacteria today. European Journal of Modern Medicine and Practice. 2024; 4(4): 113–119.
ISSN: 2795-921X.

[11] Lee J., Kim B., Koo H., Kim J., Kim M. et al. Diagnostic potential of IgG and IgA responses to Mycobacterium tuberculosis antigens for discrimination among active tuberculosis, latent tuberculosis infection, and non-infected individuals. Microorganisms. 2020; 8(7): 1–17.

[12] Liu L., Zhang W., Zheng J., Fu H., Chen Q. et al. Exploration of novel cellular and serological antigen biomarkers in the ORFeome of Mycobacterium tuberculosis. Molecular & Cellular Proteomics. 2014; 13(3): 897–906.

[13] Lu L.L., Chung A.W., Rosebrock T.R. A functional role for antibodies in tuberculosis. Cell. 2016; 167(2): 433–443.

[14] Niki M., Yoshiyama T., Nagai H., Miyamoto Y., Niki M. Nutritional status positively impacts humoral immunity against Mycobacterium tuberculosis, disease progression, and vaccine development. PLoS One. 2020; 15(8): 1–16.

[15] Perley C.C., Frahm M., Click E.M., Dobos K.M., Ferrari G. The human antibody response to the surface of Mycobacterium tuberculosis. PLoS One. 2014; 9(6): 1–12.

[16] Slogotskaya L.V., Sinitsyn M.V., Kudlay D.A. Possibilities of immunological tests in the diagnosis of latent tuberculosis infection and tuberculosis. Tuberculosis and Lung Diseases. 2019; 97(11): 46–58.

[17] Yusupalieva M.M., Zaurova M.B., Enzel D.A. The role of interleukin-6 in the cytokine storm in tuberculosis. Modern Science (Moscow). 2022; 4(3): 181–184.

Copyright © 2025 The Author(s). Published by Scientific & Academic Publishing
This work is licensed under the Creative Commons Attribution International License (CC BY). http://creativecommons.org/licenses/by/4.0/