Alqalam Journal of Medical and Applied Sciences. 2026;9(7):1896-1902
https://doi.org/10.54361/ajmas.269710

Original article

Hematological Phenotypes and Acute-Phase Kinetic Interactivity in Pulmonary versus Extrapulmonary Tuberculosis Across Native and Migrant Cohorts

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Abstract
Characterizing host hematologic and inflammatory kinetics during active Mycobacterium tuberculosis infection across different anatomical pathways is crucial for improving clinical triage. However, a critical analytical gap remains regarding how native Libyan versus migrant status independently influences these systemic inflammatory networks in Tripoli. To address this, this paper profiles baseline cellular damage and isolates the independent effects of these administrative backgrounds within the urban healthcare sectors. Retrospective data from Abu-Setta Hospital and Taj Al-Seha Clinic were analyzed using 200 participants: 100 active TB cases (50 Libyans, 50 migrants) and 100 healthy controls (86 Libyans, 14 migrants) whose eligibility required complete blood counts, lymphocyte percentages, acute-phase markers (CRP, ESR), and definitive microbiological/radiological confirmation. Results demonstrated that active infection induces a classic phenotype of anemia of chronic disease, significantly suppressing haemoglobin levels across infected Libyans (10.29 ± 2.57 g/dL) and migrants (9.91 ± 2.39 g/dL) compared to their healthy national controls (12.48 ± 2.05 and 12.31 ± 1.47 g/dL, $P < 0.001$), alongside severe innate leukocytosis. Crucially, when stratified by citizenship, native Libyan patients exhibited markedly higher baseline leukocyte counts (12.18 ± 7.47 vs. 9.58 ± 4.14 × 10^3/µL, $P = 0.029$) and profoundly elevated C-reactive protein concentrations (140.34 ± 98.80 mg/L) than their migrant counterparts (107.81 ± 54.17 mg/L, $P = 0.040$). Multivariable modelling confirmed host citizenship status acts as a standalone determinant of hepatic acute-phase reactivity ($P = 0.040$, partial $P = 0.042$), remaining uniform across both pulmonary and advanced extrapulmonary routes ($P > 0.05$). Overall, this paper presents a promising, cost-effective approach for accurately predicting mycobacterial severity and uncovering extended socio-behavioral diagnostic delays in native patients, highlighting the urgent need for decentralized screening in mobile communities to curb the 60.0% domestic relapse rate.

Keywords. Tuberculosis, Hematological Indices, Acute-phase kinetics, Citizenship status, Tripoli.

Introduction

Tuberculosis, driven by the intracellular pathogen *Mycobacterium tuberculosis*, remains a critical global infectious threat that consistently ranks among the leading causes of mortality from a single infectious agent globally (1). The systemic pathological progression of this infection is dictated entirely by the host's cellular immune efficiency (2). While a competent immune system successfully sequesters the bacilli within a protective cellular structure known as a granuloma to maintain a latent infection, immune dysregulation or systemic failure triggers the structural breakdown of this lesion (3). This structural failure allows the bacilli to multiply uncontrollably, cause tissue liquefaction, enter the bloodstream, and progress to active tuberculosis disease, which primarily affects the lungs but can disseminate to cause advanced extrapulmonary manifestations in various tissue niches (4).

The extensive systemic inflammatory response triggered by active mycobacterial replication profoundly alters the human hematopoietic system, shifting the clinical understanding of tuberculosis from a localized respiratory pathology to a complex systemic hematological disorder (5). On a cellular level, persistent chronic inflammation induces profound alterations in complete blood count indices, including leukocytosis and a marked depletion of circulating lymphocytes (6). Furthermore, this chronic inflammatory state induces a severe disruption of systemic iron trafficking, effectively trapping functional iron pools within tissue macrophages, withholding it from circulating red blood cell synthesis, and ultimately resulting in the development of anemia of chronic disease (7). Concurrently, this inflammatory signaling drives sharp increases in hepatic acute-phase reactants, particularly C-reactive protein and the erythrocyte sedimentation rate, which serve as highly sensitive indicators of physical stress and therapeutic response (8).

Despite the high clinical utility of these routine hematological and inflammatory biomarkers, a critical empirical gap exists within contemporary regional literature. While conventional studies have documented descriptive epidemiological trends of the infection (9). Comparative evidence clarifying how administrative migration status intersects with these specific physiological manifestations and blood cell phenotypes within North African transit zones remains critically limited (10). In post-conflict urban centers like Tripoli, the national tuberculosis control infrastructure must simultaneously manage two distinct clinical populations: native citizens with established local healthcare access, and highly vulnerable migrant cohorts operating within informal economies and overcrowded temporary centers without baseline health screenings (10). The scientific originality of this investigation lies in its specific utilization of multivariate analysis of variance to explicitly isolate the independent and interactive effects of citizenship status and anatomical infection site on acute-phase inflammatory networks, providing a novel pathophysiological framework that goes beyond traditional descriptive tracking.

Received*: 01-05-2026 -* ***Accepted****: 29-06-2026 -* ***Published****: 06-07-2026*

Understanding these distinct hematological configurations carries immense public health importance, as it provides objective laboratory benchmarks to identify clinical vulnerabilities, detect hidden diagnostic delays, and predict institutional therapeutic outcomes. To address these empirical and operational gaps within the regional healthcare framework, this study establishes three primary objectives. First, it aims to conduct a comparative retrospective analysis of baseline laboratory parameters, specifically evaluating complete blood count indices and acute-phase inflammatory reactants, between active tuberculosis patients and a matched cohort of healthy controls to quantify the precise systemic footprint of the infection. Second, it seeks to evaluate the direct impact of administrative migration status and anatomical site localization on individual laboratory profiles. Third, it implements advanced statistical modeling to isolate whether administrative background exerts an independent main effect on systemic inflammatory kinetics, thereby optimizing diagnostic and treatment pathways for both domestic and transient populations in urban centers.

Methods

Study Design and Setting

This research employed a retrospective, comparative design in order to assess the systemic hematological profiles and acute-phase inflammatory kinetics of individuals diagnosed as having active tuberculosis (TB). Clinical information regarding the active TB group was obtained from the institutional archives at two different hospitals located in Tripoli, Libya. Active TB cases were derived from the Abu-Setta Hospital for Chest Diseases, while healthy controls were generated from the Taj Al-Seha Clinic records. This dual-center archiving design allowed for a balanced cross-sectional comparison between individuals suffering from ongoing mycobacterial replication and an asymptomatic population representing a normal physiological baseline.

Sample Selection and Inclusion Criteria

The study included a total sample of 200 participants, divided into two main categories: Active TB Patients (n = 100) and Healthy Controls (n = 100). This group includes 50 Libyan national patients and 50 migrant patients diagnosed with active tuberculosis. Healthy Controls (n = 100): This group serves as the baseline comparison and consists of 86 healthy Libyan controls alongside 14 healthy migrant controls. The healthy individuals served as the normal physiological baseline, meaning they showed no clinical, radiological, or laboratory signs of mycobacterial infection or other acute inflammatory issues.

The criterion for inclusion was the total completeness of medical records. To qualify, records needed to contain, for each patient, a complete blood count, lymphocyte percentage, and acute phase marker (C-reactive protein and erythrocyte sedimentation rate), along with confirmatory radiological and microbiological diagnoses of active pulmonary or extrapulmonary tuberculosis.

Data Extraction Parameters and Laboratory Biomarkers

Comprehensive laboratory and clinical parameters were systematically extracted from the medical charts. The primary hematological phenotypes evaluated included total hemoglobin levels, total white blood cell counts, and absolute lymphocyte counts. The systemic inflammatory response was comprehensively monitored through two classic hepatic acute-phase reactants: serum C-reactive protein concentrations and the erythrocyte sedimentation rate. Additionally, vital physiological indicators, including baseline body weight, body temperature, and peripheral blood oxygen saturation, were compiled alongside clinical classifications such as the primary anatomical track of the infection (pulmonary versus extrapulmonary) and the patient's presentation status (newly diagnosed case versus clinical relapse).

Ethical Approval

This study relied exclusively on a retrospective archival review of historical medical records without any direct clinical intervention or direct contact with patients. Data collection protocols were conducted in full compliance with local institutional ethical guidelines and the principles of the Declaration of Helsinki. All retrieved patient indicators were completely anonymized, any personally identifiable information was removed, and they were securely coded prior to statistical analysis to ensure absolute patient confidentiality.

Statistical Analysis

Statistical analyses were performed to evaluate the differences between the cohorts. Continuous physiological and laboratory biomarkers were expressed as means and standard deviations, while categorical clinical features were summarized as frequencies and percentages. Students' independent t-tests were utilized to compare mean values between the primary disease groups and citizenship sub-cohorts. Inferential associations for categorical risk factors, structural housing qualities, and comorbidities were evaluated via Chi-Square and Fisher's Exact tests.

Crucially, a multivariable Two-way analysis of variance was conducted to isolate the main effects of administrative group and tuberculosis manifestation type and their interaction on systemic inflammatory kinetics. Finally, a binary logistic regression model was constructed to identify standalone predictors of clinical survival and institutional mortality, establishing a robust clinical baseline. All statistical significance thresholds were maintained at a p-value of less than 0.05.

Received*: 01-05-2026 -* ***Accepted****: 29-06-2026 -* ***Published****: 06-07-2026*

Results

Baseline Hematological Profiles: Cases versus Healthy Controls

The study revealed significant hematological variations between active TB patients and healthy controls, with distinct patterns observed based on administrative background. As presented in Table 1, both Libyan and migrant TB cohorts exhibited significantly lower hemoglobin levels compared to their respective healthy counterparts (P < 0.001). Notably, Libyan TB patients demonstrated a statistically significant elevation in white blood cell (WBC) counts compared to the control group (P < 0.001), whereas this trend did not reach statistical significance among the migrant cohort (P = 0.100). These findings suggest that TB infection exerts a systemic impact on haematological homeostasis, with administrative status potentially modulating the intensity of the leukocyte response.

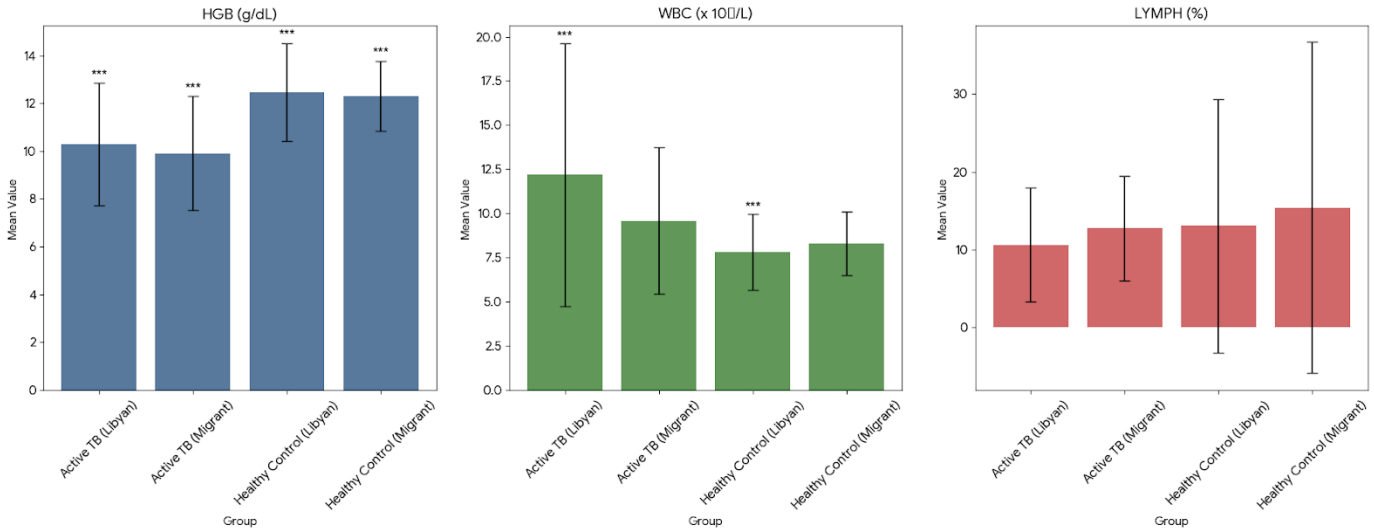

Figure 1. Comparative Hematological Parameters (HGB, WBC, and LYMPH) in TB Patients versus Healthy Controls, Stratified by Nationality. Data are presented as mean ± SD. Error bars indicate standard deviation. Statistical significance is denoted by (P < 0.001) compared to the control group.

Table 1. Comparison of Hematological Characteristics Between Cases and Healthy Controls

Group	Nationality	HGB (g/dL) (Mean ± SD)	WBC (× 10^9/L) (Mean ± SD)	LYMPH (%) (Mean ± SD)
Active TB	Libyan (n=50)	10.29 ± 2.57	12.18 ± 7.47	10.63 ± 7.31
Control	Libyan (n=86)	12.48 ± 2.05	7.81 ± 2.14	13.05 ± 16.34
P-value		P < 0.001	P < 0.001	P = 0.233
Active TB	Migrant (n=50)	9.91 ± 2.39	9.58 ± 4.14	12.76 ± 6.76
Control	Migrant (n=14)	12.31 ± 1.47	8.30 ± 1.81	15.40 ± 21.28
P-value		P < 0.001	P = 0.100	P = 0.655

Immunopathological Variations Across Administrative Subgroups

Classifying patients with active tuberculosis by migration status allowed for the identification of different inflammatory patterns, as shown in Table 2. Native Libyan patients exhibited a significantly elevated cellular inflammatory response, with a higher mean white blood cell count (12.18 ± 7.47 × 10^3 µL) compared to the migrant group (9.58 ± 4.14 × 10^3 µL) (P=0.029). Furthermore, higher levels of systemic inflammation in native Libyans were confirmed by acute-phase protein responses; the mean serum C-reactive protein concentration was significantly higher in native Libyan patients compared to the migrant group (140.34 ± 98.80 mg/L vs. 107.81 ± 54.17 mg/L) (P=0.040). No statistically significant differences were observed regarding hemoglobin levels, lymphocyte percentage, or erythrocyte sedimentation rate (P>0.05).

Table 2. Comparison of Clinical and Inflammatory Markers Between Libyan and Migrant TB Patients

Variable	Native Libyans (n=50) (Mean ± SD)	Migrant Patients (n=50) (Mean ± SD)	P-value
Hemoglobin (g/dL)	10.29 ± 2.57	9.91 ± 2.39	0.431
White Blood Cells (× 10^3/µL)	12.18 ± 7.47	9.58 ± 4.14	0.029
Lymphocyte Count (%)	10.63 ± 7.31	12.76 ± 6.76	0.125
Erythrocyte Sedimentation Rate (mm/hr)	92.83 ± 41.14	88.90 ± 27.36	0.567
C-Reactive Protein (mg/L)	140.34 ± 98.80	107.81 ± 54.17	0.040

Received: *01-05-2026 -* ***Accepted:*** *29-06-2026 -* ***Published:*** *06-07-2026*

https://doi.org/10.54361/ajmas.269710

Multivariable Analysis of Acute-Phase Inflammatory Kinetics

To evaluate whether systemic tissue alterations were independently regulated or driven by localized anatomical involvement, a multivariable assessment was implemented (Table 3). The analysis isolated a statistically significant independent main effect of the study group on both systemic C-reactive protein (CRP) kinetics (P = 0.040) and white blood cell (WBC) counts (P = 0.037). Crucially, the primary anatomical track of the infection (pulmonary vs. extrapulmonary) exerted no significant main effect on inflammatory markers, and no significant interaction effect was identified between citizenship and disease type (P > 0.05). This demonstrates that the heightened inflammatory response observed in native Libyans remains robust across diverse clinical presentations.

Table 3. Independent and Interactive Effects of Study Group and Pathology Type on Systemic Inflammatory Markers

Dependent Variable	Source of Variation	P-value	Partial Eta Squared (η^2)
C-Reactive Protein (CRP)	Study Group (Libyan vs. Migrant)	0.040	0.042
	Tuberculosis Type (Pulmonary vs. Extrapulmonary)	0.681	0.002
	Study Group × Tuberculosis Type	0.171	0.019
White Blood Cells (WBC)	Study Group (Libyan vs. Migrant)	0.037	0.043
	Tuberculosis Type (Pulmonary vs. Extrapulmonary)	0.741	0.001
	Study Group × Tuberculosis Type	0.721	0.001

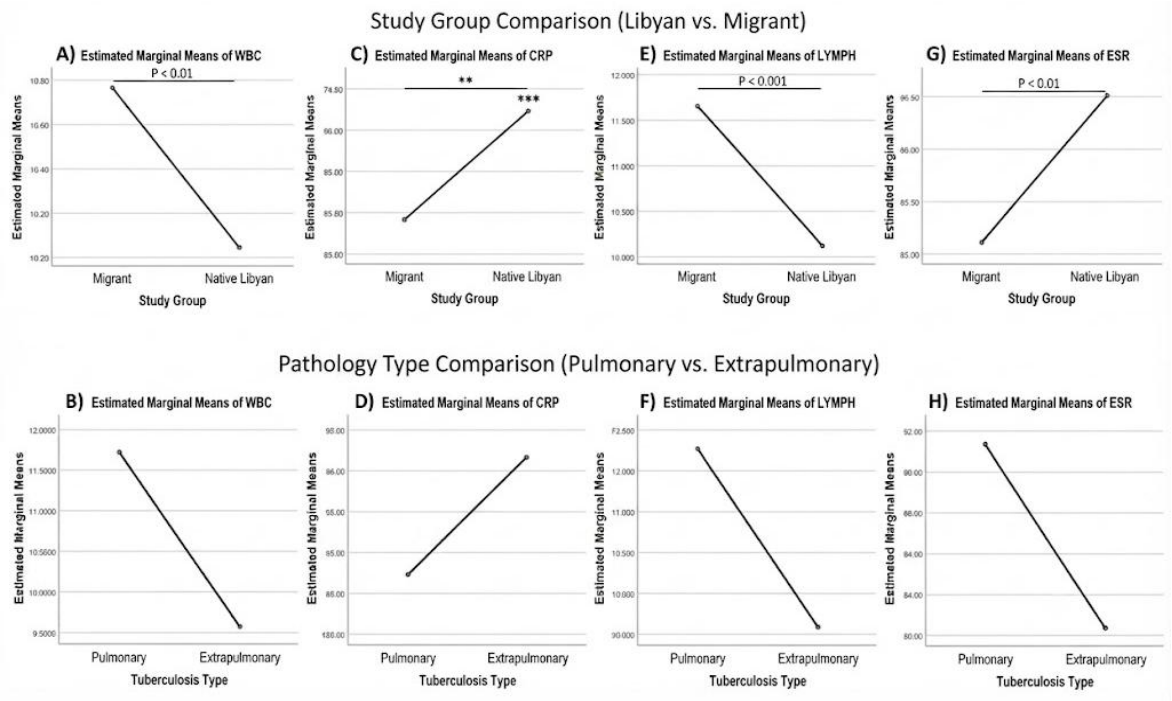

Figure 2. Multivariable analysis of inflammatory marker kinetics stratified by citizenship and pathology type. The figure displays the estimated marginal means for (A, B) WBC, (C, D) CRP, (E, F) lymphocyte percentages, and (G, H) ESR. Panels (A, C, E, and G) illustrate significant independent effects of citizenship status (Native Libyan vs. Migrant) on systemic inflammatory markers (P < 0.05). Panels (B, D, F, and H) evaluate the impact of the anatomical track of infection (pulmonary vs. extrapulmonary), demonstrating no statistically significant independent effect of disease type on systemic marker kinetics (P > 0.05). Significance for citizenship effects is denoted by (P < 0.01) and * (P < 0.001).**

The analysis isolated a statistically significant independent main effect of the study group on both C-reactive protein (CRP) kinetics (P = 0.040) and white blood cell (WBC) counts (P = 0.037). Crucially, the anatomical track of the infection (pulmonary vs. extrapulmonary) exerted no significant main effect (P > 0.05), and no significant interaction effect was identified between citizenship and disease type (P > 0.05). This demonstrates that the heightened inflammatory response in native Libyans remains robust across diverse clinical presentations.

The multivariable analysis of inflammatory marker kinetics, categorized by citizenship and pathology type, is presented in Figure 2. Panels (A, C, E, and G) reveal that citizenship status is a primary independent modulator of systemic inflammatory and immune profiles, showing significant variations in WBC, CRP, lymphocyte percentages, and ESR between native Libyan and migrant cohorts. Conversely, panels (B, D, F, and H) indicate that the anatomical track of infection (pulmonary versus extrapulmonary) does not exert a statistically significant independent effect on these systemic kinetic configurations, suggesting that the observed inflammatory divergence is driven by host-related factors rather than the localized anatomical extent of the disease.

Received*: 01-05-2026 -* ***Accepted****: 29-06-2026 -* ***Published****: 06-07-2026*

Discussion

The primary objective of this investigation was to characterize the systemic hematological footprints and acute-phase inflammatory kinetics in patients presenting with active tuberculosis, specifically isolating how administrative migration status intersects with these laboratory profiles across differing anatomical infection tracks in post-conflict North African transit environments. Upon comparative stratification of the active cohorts, native Libyan patients exhibited significantly higher baseline white blood cell counts of 12.18 ± 7.47 × 10^3/μL and profoundly elevated serum C-reactive protein concentrations of 140.34 ± 98.80 mg/L than their migrant counterparts. This biochemical divergence was statistically validated by our multivariable analysis, which isolated a significant independent main effect of citizenship status on C-reactive protein kinetics (P = 0.040), demonstrating a robust metabolic phenotype that remained uniform regardless of whether the infection tracked along a localized pulmonary or an advanced extrapulmonary anatomical route (11, 12). These findings collectively suggest that host-related socio-environmental variables serve as primary modulators of systemic inflammatory intensity, necessitating a re-evaluation of current diagnostic benchmarks to account for administrative migration status in regional clinical settings.

Systemic Hematological Disruptions and Kinetic Mechanisms of Anemia

These baseline configurations align closely with international clinical cohorts, reflecting a classic phenotypic manifestation of anemia of chronic disease combined with adaptive immune exhaustion. Pathophysiologically, our documented hemoglobin suppression mirrors trends reported by Tiu et al. (11), emphasizing that active mycobacterial replication shifts host hematopoiesis toward systemic cytopenia (1, 13). This red blood cell restriction is directly driven by persistent systemic cytokine release, which suppresses erythropoietin production and induces hyperhepcidinemia, a kinetic shift that degrades ferroportin channels and traps functional iron inside tissue macrophages (14) . Concurrently, our documented peripheral lymphopenia matches the pathways of T-cell exhaustion and granulomatous sequestration described by Lombardi et al. (15), where functional lymphocytes are recruited directly into active parenchymal lesions, a process accelerated by persistent antigen exposure and functional immunosenescence.

Host-Immune Heterogeneity and Systemic Inflammatory Modulation

While the anatomical progression of mycobacterial infection (pulmonary versus extrapulmonary) does not independently dictate the magnitude of the host response, citizenship status serves as a decisive modulator of immune kinetics. The significantly heightened leukocyte mobilization and elevated serum C-reactive protein concentrations observed in native Libyan patients suggest a profound acute-phase hepatic activation that operates independently of the infection site. This biochemical divergence implies that host-related socio-environmental variables—rather than intrinsic mycobacterial virulence patterns—are the primary drivers of the observed systemic inflammatory intensity. Our documented patterns suggest that the immunological footprint is shaped significantly by the timing of clinical presentation and long-standing socio-behavioral barriers, which exacerbate systemic inflammatory networks before institutional intervention can mitigate the cellular stress tracks. This interpretation is supported by the work of Arnold et al., (16) which links functional lymphocyte depletion in peripheral blood to continuous cellular sequestration into parenchymal lesions, a process that appears to be accelerated by the delayed diagnostic pathways we identified within the local transit ecosystem.

Socio-Behavioral Stigma and Healthcare Access Dynamics in Tripoli

This phenomenon stands in sharp contrast to established European cohorts evaluated by Lebano et al. (17), where migrant populations consistently present with markedly higher absolute inflammatory loads due to severe physical transit exhaustion, compromised environmental conditions, and lower baseline adaptive immunity. Within the specific regional transit ecosystem of Tripoli, this heightened acute-phase hepatic response among native nationals points toward extended, socio-behavioral diagnostic delays rather than lower baseline disease severity among the mobile migrant sub-cohorts. Driven by the intense social stigma surrounding mycobacterial infections in domestic circles (9), local patients frequently defer seeking tertiary clinical care, resorting to prolonged self-management and empirical community treatments until systemic inflammatory networks and cellular stress tracks peak immediately before institutional admission. Conversely, the lower baseline inflammatory indices observed among the younger migrant workers match the clinical reality that stringent workplace medical mandates and employment screening protocols often capture the pathogen at an earlier, sub-clinical physiological stage before extensive systemic tissue destruction or severe hematological exhaustion occurs (1).

Methodological Strengths, Novelty, and Educational Utilization

Ultimately, the true scientific novelty of this investigation stretches far beyond the boundaries of routine epidemiology, carving out an original biochemical framework that reshapes how we view host homeostatic collapse in transit environments. By weaving together advanced biostatistical modeling with clinical cell biology, this study successfully isolated a compelling regional paradox: while international cohorts frequently picture highly mobile migrants as the standard for extreme clinical severity, the reality within Tripoli's landscape inverts this completely, proving that the citizenship status of the host acts as a standalone, independent governor of hepatic acute-phase reactivity. This pivotal discovery uncovers a deep diagnostic gap in patient care, exposing how deep-seated domestic social stigma quietly drives local patients into extended self-management, meaning they only reach tertiary clinical facilities after systemic cellular

Received: *01-05-2026 -* ***Accepted:*** *29-06-2026 -* ***Published:*** *06-07-2026*

stress networks and tissue destruction tracks have already peaked. Methodologically, the strength of this work delivers a powerful, low-cost clinical blueprint ready for immediate integration into the regional medical laboratory curriculum. Rather than viewing standard hematological configurations as static, back-end diagnostic readouts, this research proves they can function as dynamic, forward-looking prognostic trackers. Harnessing these accessible kinetic markers allows clinicians to catch sub-clinical mycobacterial replication early and map out advanced extrapulmonary risks, handing local healthcare entities an objective, data-driven weapon to bypass behavioral delays, optimize proactive interventions, and ultimately save lives where it matters most.

Limitations

While this investigation provides significant insights into the systemic hematological footprints and acute-phase inflammatory kinetics of active tuberculosis, several methodological limitations must be acknowledged. On the one hand, since existing institutional medical records were used for data collection due to the retrospective design of this study, they were unable to accommodate more advanced and current biomarkers of immune and metabolic processes, namely: serum hepcidin levels, specific pro-inflammatory interleukin concentrations, and a complete iron panel (e.g., ferritin, total iron-binding capacity) could not be followed or quantified over time. On the other hand, the use of a single cross-section of each hematological phenotype at the time of clinical admission means the longitudinal characteristics of these blood cells and acute phase reactants could not be followed through subsequent phases of anti-TB chemotherapy.

Also, although this study demonstrated clear and substantial physiological differences between citizen groupings, there was no clarification on the sociological reasons for those differences, nor the duration of symptomology at the time of hospital admission, nor the length of the migrants' previous transit times. This lack of detailed documentation (therefore, possible confounding of these administrative-biochemical associations through differing histories of all migrant-in-transit subject sub-groups) enabled the researcher to assess the extent to which the study results would be generalizable to any other region, specifically transcontinental transit locations.

Conclusion

This research demonstrates that *Mycobacterium tuberculosis* infection induces profound systemic disruptions across both acute-phase inflammatory and hematopoietic networks, confirming that tuberculosis is a complex systemic illness rather than a localized pulmonary pathology. The observed clinical and hematological manifestations—characterized by severe anemia, myeloid leukocytosis, and dramatic peripheral lymphopenia—underscore the intense immunopathological challenge posed by ongoing mycobacterial replication. A pivotal finding of this study is the identified clinical paradox regarding host-immune expression: native Libyan patients exhibit significantly higher baseline inflammatory markers (CRP, $p = 0.040$) compared to migrant counterparts, regardless of the anatomical route of infection. This biochemical divergence indicates that host-related socio-environmental variables, particularly domestic social stigma, drive significant delays in diagnosis and medical intervention for the native population. Conversely, the comparatively lower inflammatory indices among migrant cohorts highlight the protective role of early detection through stringent employment-based screening. These findings underscore the need for public health authorities to prioritize decentralized, non-stigmatized screening networks for native populations to mitigate the systemic tissue destruction associated with advanced inflammatory peaks and ensure timely clinical intervention.

Acknowledgments

The authors express their sincere gratitude to the National Tuberculosis Program Manager at the National Center for Disease Control in Tripoli, Libya, for providing invaluable administrative guidance, facilitating logistics, and ensuring essential technical support throughout the duration of this study. Additionally, we extend our appreciation to the clinical and laboratory staff at Abu-Setta Hospital and Taj Al-Seha Clinic for their cooperation, which was vital to the successful completion of this investigation.

Conflicts of Interest

The authors declare no conflicts of interest. The research was conducted in the absence of any commercial, financial, or personal relationships that could be construed as a potential conflict of interest. The institutional and administrative support provided during data collection did not inappropriately influence the representation, analysis, or interpretation of the reported clinical results.

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Received*: 01-05-2026 -* ***Accepted****: 29-06-2026 -* ***Published****: 06-07-2026*

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Received*: 01-05-2026 -* ***Accepted****: 29-06-2026 -* ***Published****: 06-07-2026*