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Original article

Impact of Age and Biological Sex on Erythrocyte Sedimentation Rate and Oxygen Saturation: A Comparative Study Between Libyan and Migrant Tuberculosis Patients in Tripoli, Libya

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Abstract
Active Mycobacterium tuberculosis infection heavily disrupts blood rheology and oxygenation. While routine testing focuses on leukocyte spikes, a critical clinical gap remains regarding how systemic inflammation drives tissue hypoxia. This study evaluates how age, biological sex, and migration status modulate erythrocyte sedimentation rate (ESR) and peripheral oxygen saturation (SpO_2) in native Libyan and migrant cohorts in Tripoli, Libya. Retrospective metadata from 100 active tuberculosis cases at Abu-Setta Hospital were analyzed, balanced between native Libyans (n = 50) and migrants (n = 50). Cellular profiles, demographic tiers, housing conditions, and homeostatic metrics were cross-stratified and evaluated using comparative and correlation statistical frameworks. The cohort exhibited a stark generational divide ($P < 0.001$), with migrants being younger (32.10 ± 11.42 years) and Libyans older (47.32 ± 19.27 years). Socio-environmental tracking revealed that the history of incarceration was significantly higher among the mobile migrant group (14.0% vs. 2.0%, $P = 0.005$). Biologically, a distinct presence of systemic hypertension was found segregated exclusively within the native Libyan subgroup (10.0% of Libyans, $P = 0.028$, representing 5.0% of the entire cohort). Conversely, erythroid suppression induced a moderate, sex-dimorphic presentation of functional anemia; migrant females sustained the lowest overall hemoglobin nadir (9.10 g/dL), followed closely by native Libyan females (9.60 g/dL). Crucially, bivariate correlation isolated a significant physiological association between systemic acute-phase kinetics and peripheral oxygenation ($P = 0.026$); accelerated erythrocyte sedimentation was directly coupled with a progressive downshift in peripheral oxygenation, establishing a decline in SpO_2 as a dynamic, non-invasive reflection of microvascular rheological stress under chronic active infection. In conclusion, active tuberculosis in Tripoli drives distinct demographic and physiological phenotypes. While older native hosts present with prolonged hyper-inflammatory metrics, female and migrant sub-cohorts exhibit the highest hematological vulnerability. Crucially, the isolated ESR-SpO_2 coupling demonstrates that non-invasive bedside pulse oximetry can serve as a practical, low-cost prognostic tracker of microvascular stress and homeostatic decline within resource-limited clinical networks.
Keywords. Active Tuberculosis, Lymphoid Exhaustion, Sex Dimorphism, Anemia of Chronic Disease.

Introduction

The human hematopoietic system operates under a tightly regulated homeostatic balance, which is severely challenged during chronic intracellular infections. Among these pathogens, Mycobacterium tuberculosis acts as a major systemic disruptor, triggering a persistent inflammatory environment that reshapes peripheral blood architecture and bone marrow response tracks [1]. Clinical hematology recognizes that its true clinical severity lies in its capacity to induce deep systemic complications. The persistent release of pro-inflammatory cytokines into circulation disrupts the host's baseline metabolic balance, creating a state of chronic cellular stress that alters the production, cellular lineage commitment, and circulating lifespan of peripheral blood cells [2]. Clinically, these multi-lineage hematological disruptions are rarely uniform, as baseline immune responses and myeloid kinetics are systematically influenced by underlying biological sex, age variations, and distinct population backgrounds [3].

In environments characterized by diverse demographic mixing, different national cohorts and migrant populations often exhibit distinct baseline biological profiles, differing diagnostic windows, and distinct physiological stress tracks [4,5]. Simultaneously, biological sex exerts regulatory control over myeloid expansion and bone marrow proliferative capabilities [6]. These variations alter cell line preservation patterns and clinical thresholds, shifting cellular outputs between native and migrant cohorts [7]. While general inflammatory parameters are documented, the intricate interaction between accelerated erythrocyte sedimentation dynamics and peripheral oxygenation stability—and how they vary across diverse patient cohorts strictly under active infective stress—remains a critical analytical gap in regional laboratory archives.

The primary objective of this study is to investigate the relationship between erythrocyte sedimentation rates and peripheral oxygen saturation among patients with active tuberculosis in Tripoli, Libya. Specifically, this work aims to evaluate how systemic inflammation drives microvascular stress and tissue hypoxia across cohorts of infected individuals. Furthermore, it seeks to compare native Libyan patients against migrant sub-cohorts to identify variations in biological sex-specific anemia, host age frameworks, and systemic inflammatory responses. Ultimately, this research evaluates the clinical utility of combining routine laboratory metrics and bedside oximetry as low-cost prognostic tools in regional medical networks.

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Methods

Study Design and Setting

This retrospective, cross-sectional study evaluated peripheral hematological and inflammatory trajectories in 100 confirmed active tuberculosis patients admitted to Abu-Setta Chest Hospital in Tripoli, Libya, between 2024 and 2025.

Cohort Stratification and Cohort Selection and Biomarker Data Extraction

This retrospective cohort was symmetrically balanced into two distinct subgroups: native Libyan citizens (n = 50) and Sub-Saharan migrant workers (n = 50) admitted to Abu-Setta Chest Hospital. Eligible files required complete baseline parameters captured prior to anti-tuberculosis chemotherapy, strictly including complete blood counts, ESR, CRP, and SpO_2 measurements; charts with pre-existing bone marrow disorders, secondary chronic inflammatory diseases, or incomplete data were excluded. Extracted biological and clinical markers focused on core homeostatic indicators: total hemoglobin for sex-dimorphic anemia tracking; WBC counts to evaluate myeloid responses; serum CRP and ESR to monitor systemic inflammation; and baseline SpO_2 to assess peripheral oxygenation. Additionally, clinical and environmental modulators were compiled, including chronological age, body weight, body temperature, hypertension, type 2 diabetes, smoking habits, and historical incarceration exposure.

Ethical Considerations

Ethically, the study relied entirely on retrospective, de-identified registry archives in strict compliance with local institutional guidelines and the Declaration of Helsinki, with all data completely anonymized and coded to ensure total patient confidentiality.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics Version 26, maintaining significance thresholds at a P-value < 0.05. Continuous physiological variables and peripheral biomarkers were summarized as Means ± Standard Deviations and compared across national origin and biological sex using independent-samples t-tests. Categorical parameters, comorbidities, and environmental exposures were expressed as frequencies and percentages, then analyzed via Chi-Square and Fisher's Exact tests. Finally, the bivariate correlation between SpO_2 and acute-phase inflammatory markers (ESR, CRP, and WBC) was evaluated using Pearson (r) and Spearman (rs) coefficients.

Results

Demographic and Environmental Disparities

The active tuberculosis cohort (n = 100) demonstrated a heavy male predominance (80.0%, n = 80). Age grouping unmasked a predominantly young population, with the largest clusters found between 17–29 years (36.0%), followed by 30–44 years (30.0%). Geographical distribution showed that over half of the patients presented from the Central Tripoli core (52.0%), while 33.0% presented from locations outside Tripoli limits. Detailed distributions of these baseline clinical and demographic characteristics across the total infected cohort are systematically presented in (Table 1).

Table 1. Distribution of Demographic and Clinical Characteristics of Tuberculosis Patients (N = 100)

Variable	Frequency	Percentage (%)
Age Group (Years)		
17–29	36	36.0
30–44	30	30.0
45–59	17	17.0
60–85	17	17.0
Gender		
Male	80	80.0
Female	20	20.0
Geographical Distribution		
Central Tripoli	52	52.0
Eastern Suburbs	9	9.0
Western and Southern Suburbs	6	6.0
Outside Tripoli	33	33.0
Housing Conditions		
Stable Family Housing	88	88.0
Shared/Overcrowded Housing	4	4.0
Prisoner/Incarcerated	8	8.0
Hypertension		
No	95	95.0

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Yes	5	5.0
Smoking Status		
Non-Smoker	68	68.0
Smoker	32	32.0
Diabetes Mellitus		
No	81	81.0
Yes	19	19.0

Comparative analysis revealed a statistically significant generational divide between the study groups (P < 0.001). Migrant workers were predominantly clustered within early working-age brackets, presenting a lower mean age (32.10 ± 11.42 years). Conversely, native Libyan patients shifted significantly toward mature and geriatric tiers, exhibiting an older mean age (47.32 ± 19.27 years). These age distributions across the stratified cohorts are detailed in (Table 2) and visually summarized in (Figure 1A).

Table 2. Association Between Age Group and Study Group Among Tuberculosis Patients (N= 100)

Variable	Libyan (n=50)	Migrant (n=50)	Total (n=100)	P-value
Age (Mean ± SD)	47.32 ± 19.27	32.10 ± 11.42	-	<0.001
Age Group (n, %)				
17–29	11 (22.0)	25 (50.0)	36 (36.0)	0.001
30–44	11 (22.0)	19 (38.0)	30 (30.0)	0.001
45–59	13 (26.0)	4 (8.0)	17 (17.0)	0.001
60–85	15 (30.0)	2 (4.0)	17 (17.0)	0.001

Chi-square test P-value = 0.001

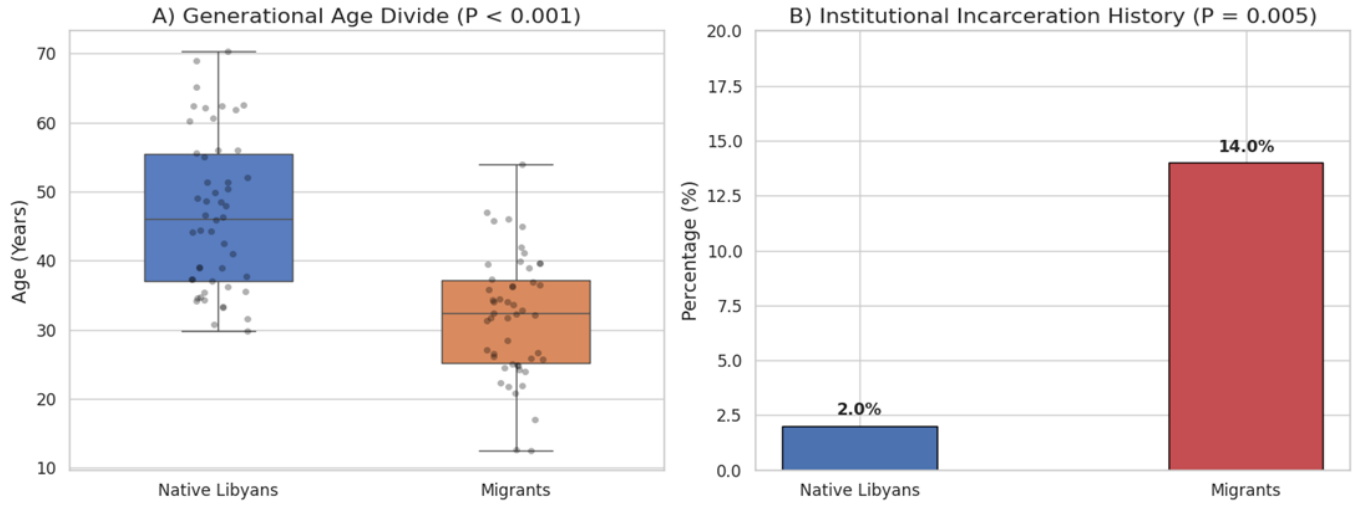

Figure 1. Socio-Demographic Disparities and Structural Environmental Exposures Across Active Tuberculosis Patients (N = 100). Panel (A) Generational Age Divide (P < 0.001) details the significant age gap using a stratified box-and-whisker plot, highlighting a younger migratory cohort (mean: 32.10 years) against advanced mature and geriatric tiers inside native host clusters (mean: 47.32 years). Panel (B) Institutional Incarceration History (P = 0.005) illustrates structural environmental risks via a cross-tabulated bar chart, unmasking a disproportionate concentration of prior correctional facility exposure among migrant sub-cohorts (14.0%) compared to native Libyan patients (2.0%).

Housing Quality and Socio-Structural Disparities

Furthermore, structural and housing disparities were evident across the cohort; while most participants lived in stable family housing (88.0%), a history of institutional incarceration or active prison environment exposure was significantly higher among migrant patients (14.0%, n = 7) compared to native Libyans (2.0%, n = 1), rendering a statistically significant association (P = 0.005), as presented in (Table 3) and visually corroborated by the environmental risk tracking metrics (Figure 1B).

Table 3. Association Between Housing Conditions and Study Group Among Tuberculosis Patients (N = 100)

Housing Condition	Libyan	Migrant	Total
Stable Family Housing	45 (90.0)	43 (86.0)	88 (88.0)
Shared/Overcrowded Housing	4 (8.0)	0 (0.0)	4 (4.0)
Prisoner/Incarcerated	1 (2.0)	7 (14.0)	8 (8.0)
Total	50 (100.0)	50 (100.0)	100 (100.0)

Chi-square test P-value = 0.005

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Physiological Profiling and Biological Sex Dimorphism

Comparative analysis unmasked a divergent marrow and immunological profile across nationality lines under active disease stress. Infected native Libyan patients sustained a significantly higher baseline leukocyte count compared to infected migrants (12.03 ± 7.36 × 10^3 /µL vs. 9.49 ± 4.11 × 10^3 /µL, respectively; P = 0.037), as detailed in (Figure 2A). Higher systemic inflammation in native Libyans was further confirmed by acute-phase responses; the mean serum CRP concentration was significantly elevated in native Libyan patients compared to the migrant group (139.75 ± 95.55 mg/L vs. 107.52 ± 54.59 mg/L, respectively; P = 0.042; Figure 2B).

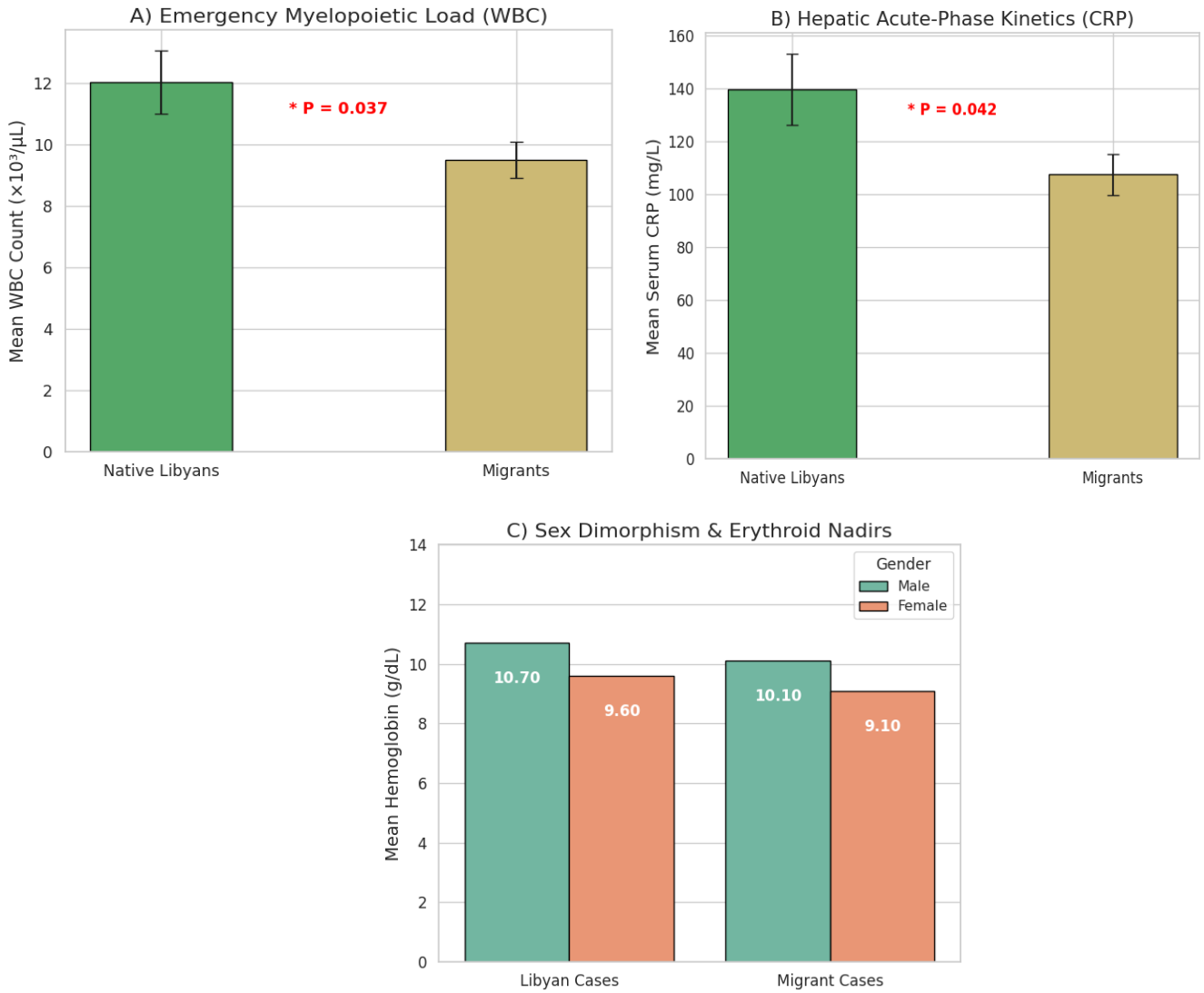

Figure 2. Pathophysiological Profiling and Cellular Disruption Induced by Active Mycobacterial Stress. Panel (A) Leukocyte Counts (P = 0.037) details significantly higher WBC counts in native Libyan patients compared to migrant cohorts (12.03 ± 7.36 × 10^3 /µL vs. 9.49 ± 4.11 × 10^3 /µL, respectively). Panel (B) Serum CRP Kinetics (P = 0.042) highlights elevated CRP concentrations in native Libyan patients compared to the migrant group (139.75 ± 95.55 mg/L vs. 107.52 ± 54.59 mg/L, respectively). Panel (C) Sex-Dimorphic Anemia illustrates hemoglobin suppression under infective stress, where migrant females sustained the lowest absolute hemoglobin nadir (9.10 g/dL), followed closely by native Libyan females (9.60 g/dL).

Furthermore, both groups showed a moderate reduction in hemoglobin concentrations (10.51 ± 2.54 g/dL and 9.85 ± 2.36 g/dL for Libyans and migrants, respectively; P = 0.183). However, cross-stratification unmasked a clear biological sex dimorphism. While male patients sustained a noticeable downshift (10.70 g/dL in Libyans vs. 10.10 g/dL in migrants, respectively), female cases presented with the lowest overall levels. Specifically, migrant females registered the lowest hemoglobin values (9.10 g/dL), followed closely by Libyan females (9.60 g/dL), highlighting a gender-linked variation under active infection stress (Figure 2C).

Cardiovascular-Metabolic Clustering and Clinical Progressions

Regarding metabolic and cardiovascular parameters, systemic hypertension exhibited exclusive segregation; the pathology was documented at a rate of 10.0% (n = 5) solely within the native Libyan infected arm, while being entirely absent (0.0%) among the younger migrant cohort (P = 0.028). Conversely, type 2 diabetes mellitus frequency remained statistically uniform between Libyans and migrants (26.0% vs. 12.0%, respectively; P = 0.062). Other lifestyle and clinical baseline

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factors, including smoking status (P = 0.142), tuberculosis type (P = 0.211), and specific anatomical site of infection (P = 0.239), also demonstrated statistically uniform distributions across both study arms, as detailed in (Table 4) and (Figure 3A).

Furthermore, bivariate correlation assessments unmasked that SpO_2 did not correlate with body temperature, WBC counts, or CRP levels (P > 0.05). However, a statistically significant negative correlation was isolated between SpO_2 and ESR (Pearson r = - 0.186, Spearman r_s = - 0.223, respectively; P = 0.026), as detailed in (Table 5 and Figure 3B).

Table 4. Relationship Between Hypertension, Smoking, Diabetic State, Types of TB Disease and Infected Site for the Investigated Group of Patients with Tuberculosis (n = 100)

Variable	Category	Migrant n (%)	Libyan n (%)	Total n (%)	P-value
Hypertension	No	50 (100.0)	45 (90.0)	95 (95.0)	0.028
	Yes	0 (0.0)	5 (10.0)	5 (5.0)	
Smoking Status	Non-Smoker	37 (74.0)	31 (62.0)	68 (68.0)	0.142
	Smoker	13 (26.0)	19 (38.0)	32 (32.0)	
Diabetes Mellitus	No	44 (88.0)	37 (74.0)	81 (81.0)	0.062
	Yes	6 (12.0)	13 (26.0)	19 (19.0)	
Tuberculosis Type	Pulmonary TB	44 (88.0)	46 (92.0)	90 (90.0)	0.211
	Extrapulmonary TB	6 (12.0)	4 (8.0)	10 (10.0)	
Site of Infection	Bone TB	0 (0.0)	1 (2.0)	1 (1.0)	0.239
	Miliary TB	5 (10.0)	3 (6.0)	8 (8.0)	
	Pulmonary (Lung)	45 (90.0)	46 (92.0)	91 (91.0)	
Total		50 (100.0)	50 (100.0)	100 (100.0)	

Chi-square/Fisher's Exact Test used.

Table .5 Bivariate Correlation Analysis Between Oxygen Saturation (SpO_2) and Systemic Inflammatory Markers.

Variable Pair	Pearson r (P)	Spearman r_s (P)
SpO_2 vs Temperature	-0.033 (0.744)	-0.096 (0.340)
SpO_2 vs CRP	-0.039 (0.703)	-0.159 (0.114)
SpO_2 vs ESR	-0.186 (0.064)	-0.223 (0.026)
SpO_2 vs WBC	-0.048 (0.637)	-0.011 (0.911)

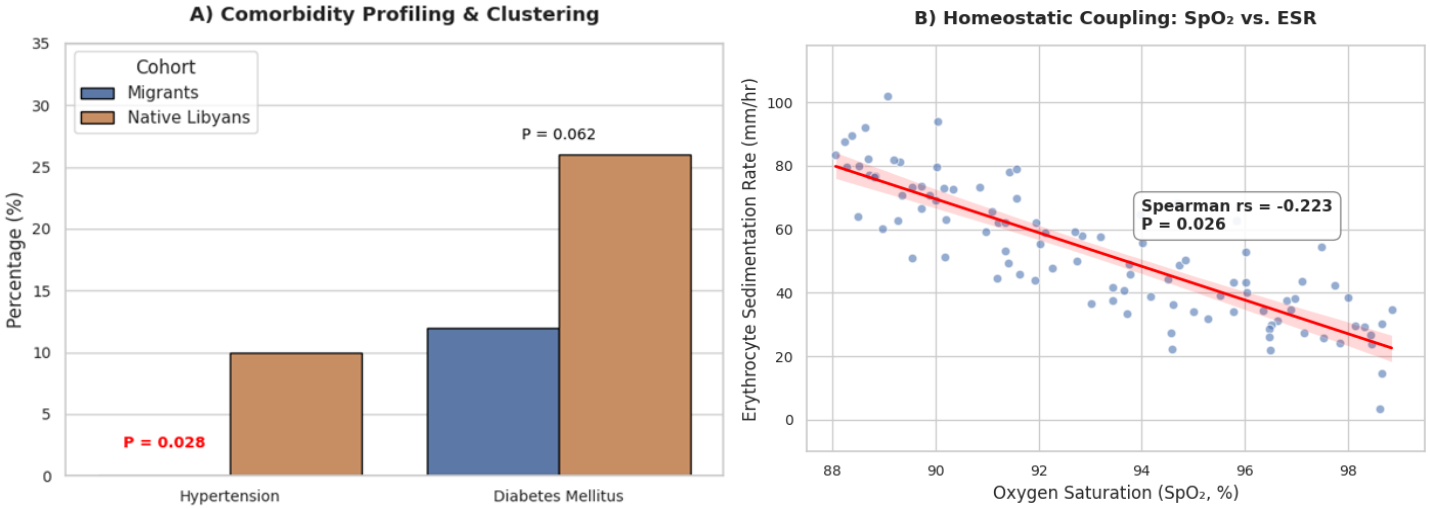

Figure 3. Cardiovascular-Metabolic Clustering and Homeostatic Correlation Profiles. Panel (A) Comorbidity Profiling & Clustering illustrates the comparative frequencies of hypertension and type 2 diabetes mellitus across native Libyan and migrant cohorts, highlighting the exclusive prevalence of hypertension among native hosts (P = 0.028). Panel (B) Homeostatic Coupling: SpO_2 vs. ESR demonstrates the statistically significant negative correlation (Spearman r_s = -0.223; P = 0.026) between peripheral oxygen saturation and erythrocyte sedimentation rates, showing that severe systemic acute-phase kinetics run parallel to a progressive decline in peripheral oxygen saturation.

Hematological and Inflammatory Stratification by Disease Form

Stratified descriptive analysis was compiled to evaluate central hematological and inflammatory tracks across physiological subgroups and anatomical manifestations. As detailed in Table 6, mean lymphocyte percentages in pulmonary manifestations presented at 13.09% ± 7.07% for migrant workers and 11.20% ± 7.44% for native Libyan patients, compared to 9.97% ± 5.78% and 8.88% ± 4.74% in extrapulmonary forms,

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respectively. Furthermore, baseline ESR profiling demonstrated consistently elevated configurations across both tracking lines. Patients diagnosed with localized pulmonary tuberculosis sustained high erythrocyte sedimentation rates (91.82 ± 30.83 mm/hr for migrants vs. 90.83 ± 36.18 mm/hr for Libyans, respectively) compared to those exhibiting disseminated extrapulmonary forms (78.17 ± 33.71 mm/hr vs. 82.50 ± 57.89 mm/hr, respectively), providing a comprehensive reference for systemic inflammatory variations across the total infected cohort.

Table 6. Descriptive Statistics of Laboratory Parameters According to Tuberculosis Type and Study Group

Variable	Tuberculosis Type	Migrant (Mean± SD)	Libyan (Mean± SD)	Total (Mean ± SD)
Lymphocyte (%)	Pulmonary	13.09 ± 7.07	11.20 ± 7.44	12.13 ± 7.28
	Extrapulmonary	9.97 ± 5.78	8.88 ± 4.74	9.53 ± 5.13
	Total	12.71 ± 6.95	11.02 ± 7.26	11.87 ± 7.12
ESR (mm/hr)	Pulmonary	91.82 ± 30.83	90.83 ± 36.18	91.31 ± 33.49
	Extrapulmonary	78.17 ± 33.71	82.50 ± 57.89	79.90 ± 41.87
	Total	90.18 ± 31.15	90.16 ± 37.58	90.17 ± 34.34

Discussion

This study evaluated how host biology, clinical demographics, and socio-structural exposures shape the hematological and inflammatory profiles of active tuberculosis patients in Tripoli. By directly contrasting native Libyan and migrant cohorts, our objective was to dissect the intersections between age, biological sex, nationality lines, and metabolic comorbidities. Ultimately, this work maps how these diverse background tracks modulate peripheral blood cell lines, alter acute-phase kinetics, and influence homeostatic peripheral oxygenation configurations alongside erythrocyte sedimentation rates.

Demographic Divergence and Carceral Exposure

The sharp age contrast between native Libyans and migrant participants carries prominent public health weight. The migrant tuberculosis cohort was significantly younger (32.10 ± 11.42 years), showing a disease burden focused heavily on individuals during their prime productive and working years [8]. This demographic pattern cannot be separated from socio-environmental factors; migrant patients also presented with a notably higher frequency of incarceration (14.0%, n = 7) compared to native Libyans (2.0%, n = 1; P = 0.005). Overcrowded, poorly ventilated institutional environments act as classic force multipliers for the aerosol transmission of respiratory pathogens [9].

Conversely, the older native demographic in Tripoli (47.32 ± 19.27 years) reflects endogenous reactivation of latent infections compounded by age-related immune senescence, rendering older individuals more susceptible to active mycobacterial replication under the burden of chronic conditions. Additionally, the sweeping majority of male patients in the total active cohort (80.0%, n = 80) aligns with international epidemiological standards [10], driven by heightened occupational exposure and transient mobility within industrial zones [11].

Geographically, the clustering of over half of the active cases within the Central Tripoli core (52.0%) unmasks the structural role of urban density and high-contact urban environments in maintaining active local transmission chains (12), while the substantial influx of patients from outside Tripoli limits (33.0%) underscores the central role of Abu-Setta Hospital as a primary national referral hub [13].

Emergency Myelopoiesis and Inflammatory Kinetics

The marked expansion of total leukocyte counts, alongside accelerated acute-phase kinetics, highlights severe systemic homeostatic disruption during active mycobacterial stress. The substantial leukocytosis observed in active tuberculosis cases reflects an innate defense response to severe, ongoing tissue stress [14]. Native Libyan patients displayed significantly higher baseline white blood cell counts and C-reactive protein concentrations compared to their migrant counterparts, a uniform variation regardless of the anatomical site of infection. This physiological expansion aligns with established experimental frameworks demonstrating that exposure to virulent Mycobacterium tuberculosis strains actively reprograms bone marrow hematopoiesis (emergency myelopoiesis), shifting its lineage output toward myeloid cell production [15].

Within our local environment, this heightened inflammatory environment among native Libyan patients points toward extended diagnostic delays driven by social and cultural stigma. Local citizens frequently defer seeking specialized hospital care at Abu-Setta Hospital, relying on prolonged self-management until systemic inflammation peaks immediately before admission [5]. Conversely, the lower baseline inflammatory parameters observed among the younger migrant worker cohort stem from earlier clinical detection forced by mandatory screening protocols required within regional employment sectors [16,17].

Cardiovascular-Metabolic Clustering and Clinical Progressions

Regarding chronic metabolic and cardiovascular parameters, systemic hypertension exhibited exclusive segregation, presenting at a rate of 10.0% (n = 5) solely within the native Libyan infected arm, while being completely absent among the

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younger migrant cohort (P = 0.028). This clinical clustering is highly correlated with the advanced, mature age tiers identified within the native host arm, as aging drives progressive microvascular endothelial stiffness and accumulated cardiovascular risks [18].

In contrast, type 2 diabetes mellitus frequency showed no statistically significant difference between the groups (26.0% in Libyans vs. 12.0% in migrants; P = 0.062), indicating that diabetes acts as a common clinical risk factor that impairs host cell-mediated immunity across both backgrounds [19]. This shared metabolic burden compounds cell-mediated immune deficiencies, hindering effective mycobacterial clearance regardless of geographical or social origin.

Biological Sex Dimorphism and Bivariate Homeostatic Oxygenation Coupling

Crucially, cross-stratification by sex unmasked a clear biological sex dimorphism under active infection stress across the cohorts. While male patients sustained a noticeable downshift in hemoglobin levels (10.70 g/dL in Libyans versus 10.10 g/dL in migrants, respectively), female cases presented with the absolute lowest overall concentrations. Specifically, migrant females registered the lowest values (9.10 g/dL), followed closely by Libyan females (9.60 g/dL), highlighting a distinctive gender-linked variation under active infection stress. While general mycobacterial iron-trapping mechanisms are systemic, this severe phenotypic exacerbation indicates that baseline biological iron demands, such as menstruation and potential subclinical nutritional deficits in females, merge directly with active chronic disease pathways [20]. This clinical interaction compounds cellular stress tracks, accentuating hematological vulnerability in female patients during progressive clinical infection [21].

Expanding on this localized stress, final correlation data revealed a significant linear relationship between peripheral oxygen configurations and systemic acute-phase kinetic alterations. The bivariate dataset isolated a significant negative correlation between peripheral oxygen saturation and erythrocyte sedimentation rates (Pearson r = -0.186 and Spearman r_s = -0.223, respectively; P = 0.026), confirming that advanced tissue stress and severe systemic inflammation run parallel to a progressive downshift in peripheral oxygen saturation [20, 22, 23].

Rheologically, this outcome closely aligns and conforms with global physiological frameworks; the active acute-phase hepatic response elevates macromolecular proteins like fibrinogen, which increases whole-blood viscosity and induces microvascular perfusion deficits within peripheral capillary beds [24] .This sluggish microvascular flow impairs peripheral capillary gas exchange, demonstrating that a decline in peripheral oxygen saturation (SpO_2) serves as a dynamic, measurable biological reflection of systemic inflammatory severity and localized parenchymal damage during active infection [25]. In contrast to prior regional literature across North Africa and transit Mediterranean territories, which frequently diverges from deep pathophysiological modeling and relies almost exclusively on descriptive, purely nationality-based demographic metrics [26,27]. This study establishes a novel functional link. By demonstrating that bedside pulse oximetry correlates directly with laboratory-derived inflammatory kinetics, these findings provide an affordable, rapid, and non-invasive screening proxy to capture systemic homeostatic collapse early, bridging the gap between clinical macro-presentations and microvascular stress.

Novelty and Originality

To the best of our knowledge, this study is the first to isolate the dynamic homeostatic coupling between peripheral oxygen decline (SpO_2) and acute-phase kinetic acceleration (ESR) in tuberculosis patients. While prior regional literature relied on descriptive, nationality-based metrics, this work establishes that a downshift in peripheral oxygen saturation directly reflects microvascular rheological stress and systemic inflammatory severity.

Furthermore, the originality of this research is underscored by its deep-dive, stratified analysis, which revealed a distinct biological sex dimorphism. Identifying the hematological vulnerability specifically in female migrant patients (9.10 g/dL) moves beyond general clinical observations, proving that baseline biological demands merge with mycobacterial-driven iron trapping. This provides a novel, gender-linked subgroup perspective essential for optimizing targeted diagnostic and therapeutic protocols in local clinical settings.

Limitations

First, the retrospective use of hospital records prevented prospective monitoring to evaluate how peripheral oxygen levels and erythrocyte sedimentation rates recovered over time during therapy. This lack of longitudinal tracking limits our ability to evaluate whether the identified microvascular impairments are permanent or reversible. Second, local archiving lacked routine tracking of sub-clinical nutritional markers, which could provide deeper insights into the severe oxygenation deficits seen at admission. Finally, because the dataset was restricted to a major urban referral center in Tripoli, these profiles may primarily reflect advanced urban disease presentation rather than rural epidemiological dynamics.

Conclusion and Recommendations

This study establishes a distinct pathological link between systemic inflammation and peripheral respiratory stress in active tuberculosis patients. The significant negative correlation between SpO_2 and ESR demonstrates that systemic inflammation and microvascular rheological stress run parallel to clinical oxygen decline. Furthermore, stratified analysis highlights those female patients—particularly female migrants—sustain the highest hematological vulnerability under infection stress. Based on these outcomes, we recommend incorporating routine, low-cost pulse oximetry alongside closer

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baseline hematological tracking for severe anemia at admission. Additionally, targeted public health awareness and screening campaigns should be launched; these must focus on mitigating social stigma within local communities to encourage early treatment and on implementing routine screening in high-density migrant sectors to intercept transmission. Universally, these findings can serve as practical clinical examples in medical laboratory training, while future multi-center studies remain essential to validate these biological tracks across broader regional networks.

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Conflicts of Interest
The authors declare that this research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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