



<https://doi.org/10.47430/ujmr.25102.003>

Received: 14/08/2025

Accepted: 03/11/2025



Evaluation of Haematological Parameters in Tuberculosis Patients Attending Infectious Diseases Hospital, Kano, Nigeria

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Abstract

Mycobacterium tuberculosis (MTB) infection is a global health concern, with Nigeria being among the countries with a high burden of the disease. This study aimed to evaluate haematologic and haemostatic parameters in subjects with MTB attending the Infectious Diseases Hospital, Kano, Nigeria. The method employed was a cross-sectional design with consecutive sampling. Significant differences in haematological and coagulation profiles between MTB subjects and non-MTB controls were found. TB subjects showed a statistically significant ($p < 0.05$) reduction in haemoglobin, haematocrit, and RBC count, coupled with increased mean corpuscular volume (MCV) and Mean Corpuscular Haemoglobin Concentration (MCHC), compared with the control group, indicating anaemia and altered red blood cell indices. The WBC count variation was insignificant, but differential leucocyte parameters (lymphocytes, monocytes, and granulocytes) varied significantly. Additionally, subjects with MTB infection also displayed prolonged Prothrombin Time (PT) and Activated Partial Thromboplastin Time with Kaolin (aPTTK), suggesting perturbed coagulation pathways. These findings accentuate the impact of MTB infection on haematologic and haemostatic systems, highlighting potential diagnostic and prognostic markers. The study recommends further longitudinal studies to explore the dynamic changes in haematological and coagulation parameters throughout the course of TB treatment and recovery.

Keywords: Biomarkers, Haematology, Haemostasis, Infectious diseases, *Mycobacterium tuberculosis*

INTRODUCTION

Tuberculosis (TB) is a widespread chronic infectious disease caused by the bacterium *Mycobacterium tuberculosis* (Mina *et al.*, 2019). TB is a major global health issue and one of the most critical contagious killer diseases (Delia *et al.*, 2025). One-third of the world's population is affected by *M. tuberculosis* infection (WHO, 2024). The primary route of infection is by inhalation of air droplets or dust particles containing *M. tuberculosis*. It occurs as pulmonary tuberculosis (PTB), extrapulmonary tuberculosis, or disseminated tuberculosis (Atiegha *et al.*, 2020; Prasiska *et al.*, 2024). PTB is an infection of the lower respiratory tract caused by the deposition of *Mycobacterium tuberculosis* (MTB) in aerosol droplets onto the lung alveolar surfaces. The active disease is marked by persistent cough, exhaustion, weight

loss, loss of appetite, and night sweats. PTB consequences include haemoptysis, subsequent fungal infection, and chronic lung injury (Akpan *et al.*, 2018; Mekenye, 2024). Haemoptysis is one of the most common symptoms of vascular wall diseases (Saidu *et al.*, 2020). Coughing, exhaustion, weight loss and appetite loss, and night sweats are symptoms of active disease. Nigeria is the top in Africa and fourth among the world's 22 high-TB burden countries (Mustapha *et al.*, 2017). In Nigeria, 460,000 cases of TB are reported each year (Oyeniyi *et al.*, 2025). The coinfection of PTB with human immunodeficiency virus has, in recent years, compounded the epidemiology, clinical outcomes, diagnosis, and treatment of the disease worldwide. This has led to different reports on this condition in Nigeria (Mwatenga *et al.*, 2024; Ochonye *et al.*, 2024).

Some haematological and biochemical abnormalities may occur in PTB and can be helpful for presumptive diagnosis and for monitoring disease or treatment outcomes (Mwatenga *et al.*, 2024). TB affects the bone marrow in addition to the lungs (Gabriel *et al.*, 2025). Haematological measures such as haemoglobin, packed cell volume (PCV), red blood cell (RBC) count, red blood cell indices, platelet count, white blood cell (WBC) count, and erythrocyte sedimentation rate (ESR) can be utilized as markers to help with the diagnosis of patients, disease prognosis and follow-up for treatment response (Shah *et al.*, 2022).

Studies have suggested that the most prevalent haematological signs of PTB include anaemia, elevated ESR, increased peripheral leucocyte count, and neutrophilia (Rabita, 2012; Raychaudhuri *et al.*, 2022; Ifeyinwa *et al.*, 2023). Additionally, Okafor *et al.* (2013) reported mild anaemia, elevated ESR, leucocytosis, neutrophilia with toxic granulation, thrombocytosis, platelet stickiness, and monocytosis in their study conducted in Kano, northwestern Nigeria. Thus, increased haemoglobin and haematocrit values are used as indicators of therapeutic response in patients with PTB (Al-muhammadi and Al-Shammery, 2011; Kumaravel *et al.*, 2024).

In humans, haemostasis is an efficient system that uses enzymatic processes to control bleeding at sites of vascular injury (Gomez and McVey, 2015; Malik *et al.*, 2021). It explains a delicate equilibrium between procoagulant and anticoagulant mechanisms that requires a complex sequence of events (Abraham and Rachel, 2024). When a microbial infection injures tissue, the body responds with inflammation. This defense mechanism seeks to eliminate or at least limit the spread of the invading pathogen while also clearing necrotic cells and tissues from the damaged area (Mohan, 2010; Kumar, 2020). TB has also been described as a persistent granulomatous disease caused by infection with *Mycobacterium tuberculosis* complex (Iseman, 2000; Charles and Geraint, 2021). It was also claimed that inflammation activates the haemostatic system, with the latter influencing the former's activity (Hazrati *et al.*, 2022; Wang *et al.*, 2022). Although haemostatic function is intended to maintain the integrity of the circulatory system, it can become unbalanced, with morbidity and mortality as possible outcomes. Poorly managed haemostatic activity as a result of inflammation may be a substantial factor in the genesis,

course, and progression of illness. This is seen in the systemic inflammatory response to infection, as in TB (Akpan *et al.*, 2018).

Haemoptysis is one of the major presentations of vascular wall disorders. Several studies have reported thrombotic complications, particularly disseminated intravascular coagulation (DIC) and deep vein thrombosis (DVT), in TB patients (Kumar *et al.*, 2025; Mitroi *et al.*, 2025). The aberrant expression of tissue factor, the primary activator of the coagulation cascade, is known to be responsible for thrombotic disorders in many diseases, including bacterial infections (Colling *et al.*, 2021). However, severe infection and inflammation almost invariably lead to haemostatic abnormalities, ranging from insignificant laboratory changes to severe DIC (Saidu *et al.*, 2020). This study aimed to elucidate the relationships between TB and alterations in these haematologic and haemostatic parameters.

MATERIALS AND METHODS

Study Area

The study was carried out at the Infectious Diseases Hospital, Kano, within the Kano metropolis. The state lies on latitude 12° 0' 0.0000" N, 8° 31' 0.0012" E. It is bordered by the states of Jigawa to the north and east, Bauchi to the southeast, and Kaduna and Katsina to the northwest (Abaje *et al.*, 2014; Salisu *et al.*, 2025). According to the national census done in 2006, Kano (with a population of 9,401,288) is the most populous state in Nigeria (Bello *et al.*, 2024).

Study Design

This was a cross-sectional study design, and consecutive sampling was used to recruit all participants.

Sampling Technique

The sampling technique employed was random sampling.

Study Population

The study population comprised subjects with *Mycobacterium tuberculosis* infection attending the Infectious Diseases Hospital, Kano, and apparently healthy individuals as controls.

Ethical Approval

Ethical clearance to conduct this study was obtained from the Research Ethics Committee of the Kano State Ministry of Health before the commencement of the study. Approval number: NHREC/17/03/2018, dated 22nd November, 2023. Each study participant provided consent before participation and was assured of anonymity and confidentiality.

Inclusion Criteria:

All TB-positive patients who attended the health facility during the study period were included. Furthermore, only patients on anti-tuberculosis therapy were included in this study. Additionally, only male and female adults who consented to participate were included in this study.

Exclusion Criteria:

Pregnant female patients were excluded from this study. Moreover, all patients who are co-infected with human immunodeficiency virus (HIV), with active or recent malaria infection, or on intestinal antihelminth drugs to guide against the cofounder effect were also excluded from this study. Furthermore, all patients below the age of eighteen (18) years were excluded from the study.

Samples Collection and Processing

Venous blood samples for haematological profiles were analyzed using an automated full blood count haematology analyzer (Orphee Mythic 18, Switzerland). The autoanalyzer's detection concept consists of two principles: electrical and optical. Electrical concepts include resistance and RF conductivity, while optical principles include laser scattering and spectrophotometry.

Haemostatic profiles' tests for prothrombin time (PT) and activated partial thromboplastin time (aPTT), respectively.

The principle of PT - is when tissue thromboplastin and calcium ions are added to plasma at 37°C, extrinsic clotting factors are activated, resulting in the generation of thrombin and the formation of a fibrin clot, and the time taken for the clot to form is recorded in seconds. While for the aPTT, the principle is that kaolin (surface activator) and platelet factor 3 substitute (phospholipid) are incubated with citrated plasma at 37°C for the time specified in the test method. Calcium chloride is added, and the time to clot is recorded in seconds (Chesbrough, 2009).

Data Analysis

Data was summarized using the appropriate measures of central tendency and dispersion. The data were subjected to normality testing, after which an appropriate test of significance for two independent groups was used at a 95% confidence interval, and p-value ≤ 0.05 was considered statistically significant. Data analysis was performed using GraphPad Prism version 10. The results were presented in tables and expressed as mean $\pm$ standard deviation (M $\pm$ SD) and percentages.

RESULTS**Socio-anthropometric**

Table 1 shows the comparison of socio-anthropometric attributes between the two groups: Subjects (n=42) and Controls (n=42). Based on gender distribution, subjects comprised 17 males (40%) and 25 females (60%), whereas controls comprised 28 males (67%) and 14 females (33%). The difference in gender distribution was statistically significant ($p = 0.010$), indicating a notable difference between the two groups. And it indicates that female subjects are more affected than male subjects. Distribution based on age showed that subjects had a mean (M) $\pm$ standard deviation (SD) of 31 ± 10.05 years, whereas the controls had a mean (M) $\pm$ standard deviation (SD) of 26 ± 2.9 years. This age difference was statistically significant ($p = 0.036$), suggesting that the participants in the subjects' group were, on average, older than those in the control group. The Body Mass Index (BMI) also showed a significant contrast between the two groups. The subject's group had a BMI of 16.25 ± 1.26 , indicating a lower BMI than the control group, which had a BMI of 20.84 ± 1.16 . This difference in BMI was statistically significant ($p \leq 0.001$), highlighting a distinct anthropometric profile between the subject and control groups, with the subjects group exhibiting lower BMI values on average.

Table 1: Distribution of Socio-anthropometric Attributes of Study Participants

Parameter	Subject (n=42)	Control (n=42)	p - value
Gender n (%)			
Male	17 (40%)	28 (67%)	0.010*
Female	25 (60%)	14 (33%)	
Age (M $\pm$ SD)	31 ± 10	26 ± 2.9	0.036*
BMI (M $\pm$ SD)	16.25 ± 1.26	20.84 ± 1.16	0.001*

Keys: * = statistically significant, n = number, SD = Standard Deviation, BMI = Body mass index, % = percentage, p = probability value

The $M \pm SD$ WBC count and differential parameters among the study participants in the subject and control groups, along with the corresponding p-values for comparison, are presented in Table 2.

For the WBC count ($\times 10^9/L$) of the subject's group, $M \pm SD$ is 4.7 ± 2.9 , while the control group has $M \pm SD$ of 5.36 ± 1.15 . When compared, a statistically significant difference (0.006) was observed.

The differential counts for granulocytes, lymphocytes, and mid cells were $43.55 \pm 17.73\%$,

$44.47 \pm 18.47\%$, and $12.37 \pm 7.8\%$ for subjects, while the control group showed $48.82 \pm 1.2\%$, $43.70 \pm 9.13\%$, and $7.54 \pm 2.44\%$ for controls, respectively. The p-values for these comparisons showed statistically significant differences.

These results indicate significant differences in WBCs and their differential counts between the subject and control groups, with the subject group generally showing lower values than the control group across almost all parameters.

Table 2: Distribution of White Blood Cells (WBCs) and Differential WBC Counts of the study participants

Parameter	Subjects (n=42)	Control (n=42)	p - value
WBC ($10^9/L$)	4.7 ± 2.9	5.36 ± 1.15	0.006*
LYM (%)	44.47 ± 18.47	43.70 ± 9.13	0.006*
MID (%)	12.37 ± 7.8	7.54 ± 2.44	0.001*
GRAN (%)	43.55 ± 17.73	48.82 ± 10.02	0.001*

Keys: * = statistically significant, WBCs: White Blood Cells, LYM: Lymphocytes, MID: mid cells (basophils, monocytes), GRAN: Granulocytes, % = percentage

The $M \pm SD$ of red blood cells (RBCs) and their indices from study participants are presented, along with the corresponding p-values for comparisons, in Table 3.

RBC count of subjects was $M \pm SD = 3.57 \pm 0.68 \times 10^{12}/L$, while the control group had $M \pm SD = 4.67 \pm 0.46 \times 10^{12}/L$. The p-value of 0.0013 indicates a statistically significant difference between the two groups, suggesting that the control group has a significantly higher RBC count than the subjects.

The haemoglobin (Hb) concentration for the subjects' group was 9.40 ± 1.2 g/dl, whereas for the control group it was 12.09 ± 1.3 g/dl. The p-value is <0.0001 , indicating a statistically significant difference in Hb concentration between the subject and control groups, with the control group having significantly higher Hb concentrations.

Regarding PCV (Packed Cell Volume), the subject's group has a mean PCV of $29.82 \pm 5.38\%$, while the control group has a mean PCV of $36.84 \pm 4.09\%$. The p-value is <0.0001 , indicating a statistically significant difference in PCV

between the subject and control groups, with the control group having a significantly higher PCV.

Moreover, on MCV (Mean Corpuscular Volume), the Test group has a mean MCV of 81.47 ± 11.74 fl, and the Control group has a slightly lower mean MCV of 79.11 ± 6.13 fl. The p-value is 0.2383, indicating no statistically significant difference in MCV between the subject and control groups. Furthermore, regarding the MCH (Mean Corpuscular Haemoglobin), the subject's group has a mean MCH of 27.17 ± 5.77 pg, while the control group has a slightly lower mean MCH of 25.95 ± 1.95 pg. The p-value was 0.5564, indicating no statistically significant difference in MCH between the subject and control groups. Finally, when looking at the MCHC (Mean Corpuscular Haemoglobin Concentration), the subject group has a mean MCHC of $32.20 \pm 6.90\%$, and the control group has a slightly higher mean MCHC of $32.83 \pm 1.29\%$. The p-value was 0.1917, indicating no statistically significant difference in MCHC between the subject and control groups.

Table 3: Distribution of RBCs Parameter and its Indices of Study Participants

Parameter	Subject	Control	p - value
RBC ($10^{12}/L$)	3.57 ± 0.68	4.67 ± 0.46	0.0013*
HGB (g/dl)	9.40 ± 1.2	12.09 ± 1.3	0.0001*
PCV (%)	29.82 ± 5.38	36.84 ± 4.09	0.0001*
MCV (fl)	81.47 ± 11.74	79.11 ± 6.13	0.2383
MCH (pg/cell)	27.17 ± 5.77	25.95 ± 1.95	0.5564
MCHC	32.20 ± 6.90	32.83 ± 1.29	0.1917

Keys: * = statistically significant, RBC: Red Blood Cell, HGB: Haemoglobin, PCV: Pack Cell Volume, MCV: Mean Cell Volume, MCH: Mean Cell Haemoglobin, MCHC: Mean Cell Haemoglobin Concentration = percentage, fl = femtoliter, pg/cell= picogram per cell

Table 3 shows the platelet count (Plt), wherein the subject's group had a mean platelet count of $243.8 \pm 115.3 \times 10^9/L$, while the control group had a slightly higher mean of $250.8 \pm 68.9 \times 10^9/L$. The p-value is 0.036, indicating a statistically significant difference in platelet counts between the subject and control groups, with the control group having a slightly higher platelet count. Regarding the Prothrombin Time (PT), the subject's group had a mean PT of 19.98 ± 3.16 seconds, whereas the control group had a significantly lower mean PT of 13.98 ± 1.5 seconds. The p-value was 0.006, indicating a

statistically significant difference in PT between the subjects and control groups, with the control group showing reduced PT.

Moreover, regarding the Activated Partial Thromboplastin Time (aPTT), the subject group had a mean aPTT of 62.43 ± 19.34 seconds, while the control group had a significantly reduced mean aPTT of 35.24 ± 4.87 seconds. The p-value is 0.003, indicating a statistically significant difference in aPTTK between the subjects and control groups, with the control group showing a reduced aPTTK.

Table 4: Distribution of Coagulation Parameters of Study Participants

Parameter	Subjects	Control	p - value
PLT ($\times 10^9/L$)	243.8 (± 115.3)	250.8 (± 68.9)	0.036*
PT(sec)	19.98 (± 3.16)	13.98 (± 1.5)	0.006*
APTTK (sec)	62.43 (± 19.34)	35.24 (± 4.87)	0.006*

Keys: * = statistically significant, PLT: Platelet, PT: Prothrombin Time, APTTK: Activated Partial Thromboplastin Time, Sec: second

DISCUSSION

Pulmonary tuberculosis (PTB) is a major infectious disease with a very high incidence in developing countries (Mariena and Rita, 2024). Based on the results of this study, the mean haemoglobin concentration and packed cell volume of subjects with PTB were significantly lower than those of control individuals. The mean cell volume and mean cell haemoglobin concentrations were significantly higher than those of controls. Previously, studies by Erster *et al.* (1980), Morris *et al.* (1989), Ebrahim *et al.* (1995), Ajayi *et al.* (2005), Lee *et al.* (2006), Miah *et al.* (2007), and Mekonnen *et al.* (2023) have reported a significant decrease in these haematological parameters of PTB subjects and agreed with the results of this study. Anaemia, defined as a reduction in haemoglobin concentration with a corresponding decrease in PCV, alongside variation in RBC indices (MCV, MCH and MCHC), has been reported in some studies (Robson, 1996; Turken *et al.*, 2002; Adewole *et al.*, 2024). Sometimes this may manifest as anaemia, coupled with other anaemia-related characteristics, in chronic disorders (Nwankwo *et al.*, 2005). PTB, being a chronic ailment, has a long-term effect on the haemopoietic system through bone marrow depression, leading to a decrease in erythropoiesis and consequently resulting in reduced values in the test subjects. In this study, the mean white blood cell count in PTB subjects is statistically significantly lower than in controls. This result agrees with the mean white blood cell count reported in some studies by Olaniyi and Aken'ova (2003) and Ibeneme *et al.* (2009), but differs from the leucocytosis previously reported by Robson *et al.*

(1996). It is also in accord with another report of decreased white cell count in PTB subjects (Awodu *et al.*, 2007). Although previous studies did not state whether their subjects were on treatment or naïve, these could have accounted for the differences in results. However, in this study majority of PTB subjects enrolled were undergoing anti-tuberculosis therapy. The results also showed a statistically significant difference in lymphocyte count between PTB subjects and control subjects. Meanwhile, the mean percentage of monocytes in the PTB subjects was significantly higher than that in the control subjects, whereas the mean percentage of granulocytes in the PTB subjects was significantly lower than that in the control subjects.

This study also shows no significant difference in mean platelet count between PTB subjects and controls. This is consistent with previous work by Martens *et al.* (1995).

Furthermore, this study has shown that the PTB subjects had both perturbations in the extrinsic and intrinsic coagulation systems, as indicated by prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT) compared with control individuals. The result is consistent with the work of Aditya *et al.* (2017), who demonstrated that TB subjects have prolonged PT and aPTTK compared with normal control subjects. A similar report was also documented by Kartaloglu *et al.* (2001). These authors opined that various cytokines, including tumor necrosis factor-alpha and interleukin 6 (IL-6), emerging from TB granulomatous lesions were thought to influence prolonged procoagulant biomarkers.

The prolonged aPTT was also believed to be due to a phospholipid-dependent coagulation marker, known to be prolonged by antiphospholipid antibodies such as the lupus anticoagulant (Osita et al., 2015). Several researchers had documented increased presence of lupus anticoagulant in TB subjects as well (Klein et al., 2015).

CONCLUSION

This study reveals significant haematological and coagulation disturbances in subjects with TB, signifying negative impact of the disease beyond pulmonary manifestations. The observed anaemia, leucopaenia, and perturbed coagulation parameters highlight the need for comprehensive work-up and monitoring strategies tailored to PTB progression and to respond to treatments, particularly with respect to haematological and haemostatic values.

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Recommendations

1. Efficient interdisciplinary collaboration among pulmonologists, haematologists, and infectious disease specialists should be encouraged to provide holistic care for TB patients. Thereby, addressing both respiratory and systemic manifestations.
2. Conducting a longitudinal study to explore the dynamic changes in haematological and coagulation parameters throughout the course of TB treatment and recovery, shedding more light on the pathophysiology and management of TB-related haematological complications.

Acknowledgements

We appreciate the support from everyone involved in ensuring the success of this study, particularly participants from the Infectious Diseases Hospital, Kano, and the Kano State Ministry of Health.

Conflict of Interest

The authors declare no conflict of interest.

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