DESCRIPTION OF HEMOGLOBIN LEVELS IN PULMONARY TUBERCULOSIS PATIENTS AT THE TOTO KABILA REGION AL GENERAL HOSPITAL (RSUD), BONE BOLANGO DISTRICT

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ABSTRACT

Pulmonary tuberculosis (TB) is a major infectious disease and remains one of the leading causes of morbidity and mortality worldwide. Anemia is a common hematological complication of TB resulting from chronic inflammation, impaired iron metabolism, and reduced erythropoiesis, which may adversely affect treatment outcomes. This study aimed to describe the hemoglobin levels of pulmonary tuberculosis patients treated at Toto Kabila Regional General Hospital, Bone Bolango Regency. A quantitative descriptive study with a cross-sectional design was conducted in May 2025. Thirty pulmonary tuberculosis patients receiving anti-tuberculosis treatment were included using total sampling. Hemoglobin concentrations were measured using an automated hematology analyzer. Data were analyzed descriptively and presented as frequencies, percentages, means, and standard deviations. Among the 30 participants, 56.7% were male and 43.3% were female, with a mean age of 47.17 ± 18.82 years. Moderate anemia was the most prevalent condition (63.3%), while severe anemia and mild anemia each accounted for 6.7%. Only 23.3% of patients had normal hemoglobin levels. The mean hemoglobin concentration was 10.09 ± 1.96 g/dL (range: 7.3–14.2 g/dL). All patients were receiving anti-tuberculosis therapy, whereas only 16.7% received iron supplementation. Anemia, predominantly moderate anemia, was highly prevalent among pulmonary tuberculosis patients. Routine hemoglobin assessment should be integrated into tuberculosis management to facilitate early detection and appropriate management of hematological abnormalities.

Keywords: *pulmonary tuberculosis; hemoglobin; anemia; iron metabolism; hematological abnormalities.*

INTRODUCTION

Pulmonary tuberculosis (TB) remains one of the most important infectious diseases and continues to represent a substantial global public health burden. Despite decades of control efforts, TB remains among the leading causes of infectious disease-related morbidity and mortality worldwide. Earlier epidemiological reports estimated 9.4 million incident cases, 14 million prevalent cases, and approximately 1.3 million deaths among HIV-negative individuals, demonstrating the persistent burden of TB on global health systems [1]. More recent global analyses further indicate that tuberculosis continues to disproportionately affect low- and middle-income countries, with the highest disease burden concentrated in Asia and Africa (Lu et al., 2025; Fu et al., 2026). Furthermore, epidemiological projections suggest that TB will remain a major public health concern over the coming decades despite improvements in disease control strategies [2].

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The global distribution of tuberculosis is uneven, with developing countries contributing the majority of reported cases. Epidemiological evidence indicates that countries such as Indonesia, China, India, and several African nations continue to experience high TB incidence due to population density, socioeconomic disparities, limited healthcare access, and delayed diagnosis [3][2]. Consequently, tuberculosis remains a major challenge for healthcare systems because of its high transmission rate, prolonged treatment duration, and substantial socioeconomic impact. Understanding the clinical manifestations and complications of pulmonary TB is therefore essential for improving patient management and reducing disease burden.

Beyond pulmonary manifestations, tuberculosis is frequently associated with hematological abnormalities, particularly anemia characterized by reduced hemoglobin (Hb) concentrations. Anemia in pulmonary TB patients is a multifactorial condition resulting from chronic inflammation, impaired erythropoiesis, nutritional deficiencies, and disturbances in iron metabolism [4]. During active tuberculosis infection, inflammatory cytokines stimulate hepatic production of hepcidin, a key regulator of iron homeostasis. Elevated hepcidin limits intestinal iron absorption and prevents iron release from macrophages, resulting in functional iron deficiency despite adequate body iron stores [5][6]. Simultaneously, Mycobacterium tuberculosis competes with the host for iron, further disrupting erythrocyte production and contributing to decreased hemoglobin concentrations [4][7].

Reduced hemoglobin levels have important clinical consequences for pulmonary tuberculosis patients. Anemia decreases oxygen-carrying capacity, exacerbates fatigue, weakness, reduced physical performance, and impaired immune function, potentially delaying recovery and compromising treatment outcomes. Persistent anemia has also been associated with sustained systemic inflammation and poorer clinical prognosis, particularly among patients with complicated tuberculosis [8]. Consequently, assessment of hemoglobin status represents an important component of comprehensive tuberculosis management because it provides valuable information regarding disease severity, nutritional status, and patient prognosis [6].

Previous studies have demonstrated considerable variation in hemoglobin profiles among pulmonary TB patients. Sulochana et al. [9], reported significant hematological alterations during active pulmonary tuberculosis, whereas Reddy [10], observed variations in clinical and hematological parameters among TB patients depending on disease characteristics. Likewise, Bashir et al. [7], highlighted the important role of iron indices in understanding anemia among pulmonary tuberculosis patients, while Amila and Sembiring [11], demonstrated that improving nutritional intake may contribute to increased hemoglobin concentrations during TB treatment. Collectively, these findings suggest that hemoglobin status varies considerably among pulmonary TB patients and is influenced by multiple physiological and clinical factors.

Although the association between pulmonary tuberculosis and anemia has been widely recognized, several important research gaps remain. Most published studies have focused primarily on the mechanisms of anemia, iron metabolism, or therapeutic interventions rather than

providing descriptive evidence regarding hemoglobin distribution among pulmonary tuberculosis patients in hospital settings [4][6]. Furthermore, studies describing hemoglobin levels in different geographical and clinical populations remain limited, making it difficult to compare patient characteristics across healthcare institutions [9][10]. Hospital-based descriptive studies are therefore needed to provide baseline clinical information that may support early identification and management of anemia among pulmonary TB patients.

Understanding hemoglobin levels in pulmonary tuberculosis patients has important clinical implications. Early detection of reduced hemoglobin concentrations may facilitate appropriate nutritional assessment, improve supportive management, and optimize patient monitoring throughout anti-tuberculosis therapy. In addition, characterizing hemoglobin status may contribute to better understanding of disease severity and support individualized clinical decision-making, particularly in resource-limited healthcare settings [7][6].

Therefore, this study aims to describe the hemoglobin levels of pulmonary tuberculosis patients treated at the hospital. The findings are expected to provide baseline evidence regarding the distribution of hemoglobin concentrations among pulmonary TB patients and contribute to the understanding of the clinical implications of anemia in this population.

Literature Review

Pulmonary Tuberculosis

Pulmonary tuberculosis (PTB) is a chronic infectious disease caused by Mycobacterium tuberculosis that primarily affects the lungs and remains one of the leading causes of morbidity and mortality from infectious diseases worldwide. Transmission occurs through airborne droplets released by infected individuals during coughing, sneezing, or speaking. Although effective anti-tuberculosis treatment is available, delayed diagnosis, prolonged treatment duration, poor treatment adherence, and socioeconomic inequalities continue to impede disease control efforts [12][13]. Developing countries continue to experience the highest TB burden because of population density, inadequate healthcare access, and limited preventive measures [14][15]. Consequently, pulmonary tuberculosis remains a major public health concern requiring comprehensive clinical management and continuous epidemiological surveillance.

Pulmonary tuberculosis also produces systemic manifestations beyond pulmonary tissue damage. Persistent infection triggers chronic inflammatory responses that affect nutritional status, hematological profiles, and immune function. These systemic effects contribute substantially to disease severity and clinical prognosis, making laboratory evaluation an essential component of patient assessment [16][17].

Hemoglobin Physiology

Hemoglobin is the principal oxygen-carrying metalloprotein contained within erythrocytes and plays a fundamental role in maintaining tissue oxygenation. Structurally, hemoglobin consists of four globin polypeptide chains, typically two α-globin and two β-globin chains, each containing a heme group capable of reversibly binding oxygen molecules. Besides transporting oxygen from the lungs to peripheral tissues, hemoglobin also facilitates carbon dioxide transport and contributes to acid-base

balance, thereby supporting normal cellular metabolism [18].

Normal hemoglobin concentrations are essential for adequate tissue oxygen delivery. Reduced hemoglobin levels impair oxygen transport, resulting in tissue hypoxia, fatigue, weakness, reduced physical capacity, and diminished physiological function. Therefore, hemoglobin concentration is widely used as a primary laboratory indicator for diagnosing anemia and evaluating the overall hematological status of patients with chronic infectious diseases, including pulmonary tuberculosis [18].

Anemia in Pulmonary Tuberculosis

Anemia is among the most frequently reported hematological complications in pulmonary tuberculosis patients. The condition may present as normocytic normochromic anemia, microcytic hypochromic anemia, iron deficiency anemia, or, less commonly, autoimmune hemolytic anemia. The pathogenesis is multifactorial, involving chronic inflammation, nutritional deficiencies, impaired erythropoiesis, and altered iron metabolism [12][19].

Recent evidence has demonstrated a significant relationship between anemia and pulmonary tuberculosis. Liu et al. (2026), through a bidirectional Mendelian randomization study, suggested a causal association between several types of anemia and pulmonary tuberculosis, indicating that hematological abnormalities are not merely secondary manifestations but may also influence disease progression. Clinically, anemia has been associated with prolonged recovery, increased disease severity, and poorer treatment outcomes, emphasizing the importance of routine hemoglobin assessment in TB management.

Iron Metabolism in Pulmonary Tuberculosis

Iron is an essential micronutrient required for erythropoiesis, oxygen transport, cellular respiration, and immune function. However, iron is also indispensable for the survival and replication of Mycobacterium tuberculosis. Consequently, pulmonary tuberculosis disrupts iron homeostasis through complex interactions between host defense mechanisms and bacterial iron acquisition strategies [7].

Alterations in iron metabolism among TB patients are reflected by changes in several laboratory parameters, including hemoglobin concentration, serum iron, ferritin, transferrin saturation, and total iron-binding capacity. Bashir et al. [7], demonstrated that these iron indices provide valuable information for differentiating iron deficiency anemia from anemia of inflammation in pulmonary tuberculosis patients. Likewise, Tupý and Solovič [20], reported that pre-existing iron deficiency further aggravates hematological abnormalities during active tuberculosis, highlighting the importance of evaluating iron status in conjunction with hemoglobin levels.

Inflammation and the Role of Hepcidin

Chronic inflammation is the principal mechanism underlying anemia in pulmonary tuberculosis. During active infection, inflammatory cytokines stimulate hepatic synthesis of hepcidin, the master regulator of systemic iron homeostasis. Increased hepcidin inhibits ferroportin-mediated iron export from enterocytes and macrophages, thereby reducing intestinal iron absorption and limiting circulating iron availability despite adequate body iron stores [21][5].

The resulting functional iron deficiency suppresses erythropoiesis and

contributes to anemia of chronic inflammation. Hella et al. [5], demonstrated that elevated hepcidin concentrations were closely associated with anemia severity and inflammatory activity among tuberculosis patients. Furthermore, Kaushik et al. [22], reported that reduced circulating iron concentrations accompanied by increased inflammatory cytokine ratios represent a host defense mechanism known as nutritional immunity, whereby iron availability is intentionally restricted to inhibit bacterial proliferation. While this response limits microbial growth, it also compromises hemoglobin synthesis and erythrocyte production.

Previous Studies on Hemoglobin Levels in Pulmonary Tuberculosis Patients

Numerous studies have documented reduced hemoglobin concentrations among patients with active pulmonary tuberculosis. Sulochana et al. (reported in related hematological investigations) and Bashir et al. [7], consistently observed that pulmonary TB patients frequently exhibit lower hemoglobin concentrations accompanied by abnormalities in iron indices. These hematological disturbances become more pronounced in patients with severe disease or prolonged inflammatory responses.

Similarly, Tupý and Solovič [20], found that anemia associated with pulmonary tuberculosis presents heterogeneous characteristics depending on baseline iron status, whereas Hella et al. [5], emphasized the role of inflammation-mediated hepcidin regulation in determining anemia severity. More recently, Liu et al. [23], provided evidence supporting a causal relationship between anemia and pulmonary tuberculosis, suggesting that hematological abnormalities may influence disease susceptibility and progression. Collectively, these findings indicate that hemoglobin concentration is not only a routine laboratory parameter but also an important clinical indicator reflecting inflammatory activity, nutritional status, iron metabolism, and disease severity in pulmonary tuberculosis patients.

RESEARCH METHODS

Study Design

This study employed a quantitative descriptive research design using a cross-sectional approach. The study was designed to describe the hemoglobin levels of patients diagnosed with pulmonary tuberculosis at a single point in time without examining causal relationships between variables.

Study Setting and Period

The study was conducted in May 2025 at Toto Kabila Regional General Hospital (RSUD Toto Kabila), Bone Bolango Regency, Gorontalo Province, Indonesia. Laboratory examinations were performed in the Clinical Laboratory of the hospital.

Population and Sample

The study population consisted of pulmonary tuberculosis patients who underwent treatment at RSUD Toto Kabila during the study period. A total of 30 patients who met the inclusion criteria were recruited as the study sample.

Inclusion Criteria

1. Patients diagnosed with pulmonary tuberculosis.
2. Patients receiving anti-tuberculosis treatment (OAT).
3. Patients willing to participate and provide informed consent.

Exclusion Criteria

1. Patients with incomplete clinical or laboratory data.

2. Patients with hematological disorders unrelated to pulmonary tuberculosis that could affect hemoglobin levels.

Data Collection

Primary data were obtained through venous blood collection from eligible participants. Hemoglobin concentration was measured using an automated hematology analyzer according to the standard operating procedures of the Clinical Laboratory at RSUD Toto Kabila. Demographic characteristics, including age and sex, together with information regarding anti-tuberculosis treatment and iron supplementation, were collected from medical records and patient interviews.

Study Variables

The primary variable investigated in this study was the hemoglobin level of pulmonary tuberculosis patients. Supporting variables included age, sex, anti-tuberculosis treatment status, and iron supplementation.

Data Analysis

Data were analyzed descriptively using statistical software. Categorical variables were presented as frequencies and percentages, whereas continuous variables were summarized using the minimum value, maximum value, mean, and standard deviation. The results were presented in tables to describe the distribution of hemoglobin levels among pulmonary tuberculosis patients.

RESEARCH RESULT

Respondent Characteristics

A total of 30 patients with pulmonary tuberculosis participated in this study. The distribution of respondents by sex is presented in Table 1.

Table 1. Distribution of Respondents by Sex

Sex	Frequency (n)	Percentage (%)
Male	17	56.7
Female	13	43.3
Total	30	100.0

Source: Primary Data, 2025.

As shown in Table 1, the majority of respondents were male (56.7%), while 43.3% were female.

Distribution of Hemoglobin Levels

The distribution of hemoglobin levels among pulmonary tuberculosis patients is presented in Table 2.

Table 2. Distribution of Hemoglobin Levels

Hemoglobin Category	Frequency (n)	Percentage (%)
Severe anemia	2	6.7
Moderate anemia	19	63.3
Mild anemia	2	6.7
Normal	7	23.3
Total	30	100.0

Source: Primary Data, 2025.

Table 2 demonstrates that moderate anemia was the most common hemoglobin abnormality, affecting 19 patients (63.3%). Only 7 patients (23.3%) had normal hemoglobin levels. Severe anemia and mild anemia were each observed in 2 patients (6.7%), indicating that anemia was prevalent among pulmonary tuberculosis patients included in this study.

Distribution of Hemoglobin Categories According to OAT Treatment and Iron Supplementation

Table 3. Distribution of Hemoglobin Categories by Anti-Tuberculosis Treatment (OAT)

Hemoglobin Category	Receiving OAT	Not Receiving OAT	Total
Severe anemia	2	0	2
Moderate anemia	19	0	19
Mild anemia	2	0	2
Normal	7	0	7
Total	30	0	30

Table 4. Distribution of Hemoglobin Categories by Iron Supplementation

Hemoglobin Category	Iron Supplement	No Iron Supplement	Total
Severe anemia	0	2	2
Moderate anemia	0	19	19
Mild anemia	0	2	2
Normal	5	2	7
Total	5	25	30

Source: Primary Data, 2025.

All respondents (100%) were receiving anti-tuberculosis drug (OAT) therapy during the study period. Regarding iron supplementation, only 5 respondents (16.7%) reported taking iron supplements, whereas 25 respondents (83.3%) did not receive iron supplementation. None of the patients with severe or moderate anemia reported consuming iron supplements. Iron supplementation was observed only among patients with normal hemoglobin levels.

Descriptive Statistics

The descriptive statistics of the study variables are presented in Table 5.

Table 5. Descriptive Statistics

Variable	N	Minimum	Maximum	Mean	Standard Deviation
Sex	30	1	2	1.43	0.504
Age (years)	30	20	72	47.17	18.82
Hemoglobin (g/dL)	30	7.3	14.2	10.09	1.96

Source: Primary Data, 2025.

The mean age of the respondents was 47.17 ± 18.82 years, with ages ranging from 20 to 72 years, indicating that most participants were adults to older adults. The mean hemoglobin concentration was 10.09 ± 1.96 g/dL, with values ranging from 7.3 g/dL to 14.2 g/dL. The average hemoglobin level was below the normal reference value for adults, indicating that anemia was common among pulmonary tuberculosis patients treated at RSUD Toto Kabila.

DISCUSSION

Characteristics of Pulmonary Tuberculosis Patients

This study found that the majority of pulmonary tuberculosis (TB) patients were male (56.7%), while females accounted for 43.3% of the respondents. This finding is consistent with global epidemiological evidence indicating that pulmonary TB is generally more prevalent among men than women. The higher incidence among males has been associated with greater exposure to environmental and occupational risk factors, smoking habits, delayed healthcare-seeking behavior, and biological differences influencing immune responses to *Mycobacterium tuberculosis* [1][3][2]. The predominance of male patients observed in this study therefore reflects the demographic pattern commonly reported in countries with a high TB burden, including Indonesia.

The mean age of respondents was 47.17 ± 18.82 years, ranging from 20 to 72 years, indicating that pulmonary TB predominantly affected adults and older adults. Individuals within this age group are generally more vulnerable to chronic infectious diseases because aging is associated with gradual deterioration of immune function, increased prevalence of comorbidities, and nutritional deficiencies. These conditions may contribute not only to TB susceptibility but also to hematological abnormalities, including decreased hemoglobin levels.

Hemoglobin Levels Among Pulmonary Tuberculosis Patients

The principal finding of this study was the high prevalence of anemia among pulmonary TB patients. Moderate anemia was the most frequent condition (63.3%), while only 23.3% of patients had normal hemoglobin concentrations. Furthermore, the mean hemoglobin level was 10.09 ±

1.96 g/dL, which is below the normal reference range for healthy adults. These findings indicate that anemia remains one of the most common hematological manifestations in pulmonary tuberculosis.

The occurrence of anemia in pulmonary TB is biologically plausible because chronic infection induces a sustained inflammatory response that interferes with erythropoiesis and iron metabolism. During TB infection, inflammatory cytokines stimulate hepatic production of hepcidin, the principal regulator of systemic iron homeostasis. Elevated hepcidin suppresses intestinal iron absorption and inhibits iron release from macrophages, resulting in reduced iron availability for erythropoiesis despite adequate iron stores. Consequently, hemoglobin synthesis decreases, leading to anemia of inflammation [4][6][5].

In addition to inflammation, *Mycobacterium tuberculosis* competes with the host for iron, an essential micronutrient required for bacterial survival and proliferation. This competition further disrupts iron metabolism and contributes to functional iron deficiency. Bashir et al. [7], reported that pulmonary TB patients frequently exhibit abnormalities in serum iron indices, including reduced serum iron concentrations and altered ferritin levels, supporting the role of disturbed iron homeostasis in the development of anemia. Therefore, the predominance of moderate anemia observed in the present study likely reflects the combined effects of chronic inflammation, impaired erythropoiesis, and dysregulated iron metabolism.

The present findings are also consistent with previous studies. Sulochana et al. [9], demonstrated that hematological abnormalities, particularly decreased hemoglobin levels, are common among pulmonary TB patients. Similarly, Reddy [10], reported that anemia frequently accompanies pulmonary tuberculosis and is associated with the overall clinical condition of affected patients. Moreover, Hella et al. [5], found that inflammatory activity and increased hepcidin concentrations were strongly associated with anemia among individuals with tuberculosis. Collectively, these studies support the present finding that reduced hemoglobin concentration is a common clinical manifestation of pulmonary TB.

Hemoglobin Levels in Relation to Anti-Tuberculosis Therapy and Iron Supplementation

All respondents in this study were receiving anti-tuberculosis drug (OAT) therapy, indicating that anemia persisted despite ongoing pharmacological treatment. This finding suggests that successful control of *Mycobacterium tuberculosis* infection alone may not immediately normalize hemoglobin levels because inflammatory pathways and impaired iron metabolism may continue during treatment. Cercamondi et al. [4], demonstrated that disturbances in iron homeostasis often persist throughout the inflammatory phase of tuberculosis and gradually improve as inflammation resolves.

Another important finding was that only 16.7% of respondents reported taking iron supplements, whereas the majority of patients (83.3%) did not receive iron supplementation. Interestingly, none of the patients with severe or moderate anemia consumed iron supplements. This observation may reflect cautious clinical practice because routine iron supplementation during active tuberculosis remains controversial. Excessive iron supplementation may increase iron availability for *Mycobacterium tuberculosis*, potentially promoting bacterial survival if provided without careful clinical evaluation [6].

Current evidence indicates that iron supplementation in TB patients should be individualized according to iron status rather than administered routinely. Functional iron deficiency caused by inflammation differs from absolute iron

deficiency; therefore, indiscriminate iron supplementation may not effectively correct anemia and may even increase infection-related risks. Bashir et al. [7], emphasized that comprehensive assessment of iron indices is essential before initiating iron therapy in patients with pulmonary tuberculosis.

Clinical Implications

The high prevalence of anemia observed in this study highlights the importance of routine hematological assessment among pulmonary tuberculosis patients. Hemoglobin measurement provides valuable clinical information regarding disease severity, nutritional status, inflammatory burden, and treatment monitoring. Persistent anemia has been associated with delayed recovery, impaired physical function, reduced quality of life, and unfavorable treatment outcomes [8].

Considering that the average hemoglobin concentration in this study remained below the normal reference value, comprehensive management of pulmonary TB should extend beyond antimicrobial therapy. Nutritional assessment, evaluation of iron metabolism, and periodic monitoring of hemoglobin concentrations should be integrated into routine TB care to facilitate early identification and management of anemia. Such an approach may improve clinical recovery, enhance treatment response, and reduce the risk of complications among pulmonary tuberculosis patients.

Overall, the present findings strengthen previous evidence that anemia is a common hematological complication of pulmonary tuberculosis and is closely associated with chronic inflammation and disrupted iron metabolism. The predominance of moderate anemia among patients treated at RSUD Toto Kabila emphasizes the need for comprehensive clinical evaluation and appropriate management of hematological abnormalities as part of integrated tuberculosis care.

CONCLUSION

This study demonstrated that anemia is a common hematological abnormality among pulmonary tuberculosis patients treated at Toto Kabila Regional General Hospital. Moderate anemia was the predominant finding, affecting 63.3% of patients, while the mean hemoglobin concentration (10.09 ± 1.96 g/dL) was below the normal reference range, indicating that most patients experienced reduced hemoglobin levels despite receiving anti-tuberculosis therapy.

These findings support previous evidence that chronic inflammation and disrupted iron metabolism during tuberculosis contribute substantially to decreased hemoglobin concentrations. The high prevalence of anemia highlights the importance of incorporating routine hemoglobin assessment into tuberculosis management to enable early detection, appropriate nutritional evaluation, and individualized supportive care.

Future studies with larger sample sizes and analytical study designs are recommended to investigate the determinants of anemia among pulmonary tuberculosis patients, including inflammatory biomarkers, iron status, nutritional factors, disease severity, and treatment outcomes.

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