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Relationship Between Tuberculosis and Anemia in African Hospitals: Pathophysiology, Clinical Burden, and Management Implications

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ABSTRACT

Tuberculosis (TB) remains a leading cause of morbidity and mortality in Africa. Anemia is a frequent hematological abnormality among hospitalized TB patients and is increasingly recognized as both a consequence and prognostic marker of disease severity. In African hospitals where TB, HIV co-infection, malnutrition, and parasitic diseases coexist, the burden of TB-associated anemia is substantial. This review synthesizes current evidence on prevalence, mechanisms, risk factors, clinical consequences, diagnostic challenges, and management strategies of anemia among TB patients in African healthcare settings. The predominant mechanism is anemia of chronic inflammation mediated by hepcidin-driven iron sequestration. However, iron deficiency anemia (IDA), HIV-associated bone marrow suppression, helminth infections, micronutrient deficiencies, and anti-TB drug effects contribute significantly. TB-associated anemia correlates with delayed sputum conversion, severe disease, prolonged hospitalization, and increased mortality. Management remains challenging due to diagnostic overlap between IDA and anemia of inflammation (AI), and concerns regarding iron supplementation in active infection. Context-specific protocols integrating TB care with anemia screening, nutritional assessment, and HIV management are urgently needed. Addressing anemia may improve TB outcomes and reduce inpatient mortality in African hospitals.

Keywords: Tuberculosis, Anemia, Africa, Hepcidin, HIV co-infection, Inflammation, Iron metabolism, Hospital outcomes

INTRODUCTION

Tuberculosis (TB) remains one of the leading infectious causes of morbidity and mortality worldwide, with Sub-Saharan Africa (SSA) bearing a disproportionate burden of disease. Despite decades of global control efforts, SSA continues to record some of the highest TB incidence and mortality rates, largely driven by socio-economic vulnerabilities, health system limitations, and high HIV prevalence. In many African countries, TB remains endemic, with hospitals frequently managing patients presenting with advanced pulmonary and extrapulmonary disease [1]. Delayed diagnosis, limited access to early treatment, and coexisting conditions such as HIV infection, malnutrition, parasitic infections, and chronic inflammation contribute to complex clinical presentations. One of the most common yet underappreciated hematological abnormalities observed in TB patients is anemia. Studies conducted across African hospital settings report anemia prevalence ranging from 30% to 80%, depending on diagnostic thresholds, patient populations, and study design. Severe anemia is not uncommon, particularly among hospitalized individuals with advanced disease [2]. The burden appears even higher among patients co-infected with HIV, those with disseminated TB, and individuals from nutritionally vulnerable populations.

Anemia in TB is multifactorial. The predominant mechanism is anemia of chronic disease (ACD), driven by inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ), which disrupt iron metabolism and erythropoiesis. Hepcidin-mediated iron sequestration reduces iron availability for red blood cell production, while chronic immune activation suppresses erythropoietin responsiveness [3]. In addition to inflammation-mediated mechanisms, other contributors include iron deficiency, nutritional deficiencies (folate, vitamin B12), bone marrow suppression, hemolysis, HIV-associated marrow dysfunction, opportunistic infections, and drug-related hematotoxicity.

The clinical relevance of anemia in TB extends beyond being a mere laboratory abnormality. Increasing evidence demonstrates that anemia correlates strongly with TB disease severity, bacterial burden, and systemic inflammation. Several African cohort studies have shown that baseline hemoglobin levels independently predict treatment outcomes and mortality [4]. Severe anemia has been associated with delayed sputum conversion, prolonged hospitalization, and increased case fatality rates. Furthermore, anemia significantly contributes to fatigue, reduced functional capacity, impaired cognitive performance, and diminished quality of life, thereby compounding the socioeconomic burden of TB in already vulnerable populations.

Despite its high prevalence and prognostic implications, anemia in TB patients is often under-investigated in African clinical practice. In many hospital settings, anemia is treated empirically—frequently with iron supplementation, without adequate differentiation between anemia of chronic disease and true iron deficiency anemia. Such non-targeted management may be ineffective or potentially harmful, particularly in inflammatory states where iron supplementation could exacerbate infection or oxidative stress [5]. Moreover, laboratory resources required for detailed anemia workup such as ferritin, transferrin saturation, reticulocyte counts, and inflammatory markers are often limited in low-resource settings. Given the high TB burden in SSA and the frequent coexistence of anemia, a comprehensive evaluation of the epidemiology, mechanisms, clinical implications, and management strategies of TB-associated anemia is urgently needed. Understanding this relationship is critical for optimizing patient outcomes in African hospitals [6].

Anemia is highly prevalent among hospitalized tuberculosis (TB) patients in Sub-Saharan Africa (SSA) but remains poorly characterized and inadequately managed in routine hospital care. Most TB guidelines prioritize antimicrobial therapy, with limited emphasis on systematic hematologic evaluation, so anemia is often recorded without investigation of its etiology or prognostic significance [7]. This creates major clinical and public health risks: failure to differentiate anemia of chronic disease from iron deficiency anemia can lead to inappropriate therapy; uncorrected or severe anemia may independently worsen TB outcomes through delayed recovery, greater morbidity, and increased mortality; and weak integration of hemoglobin assessment into TB care pathways limits risk stratification and individualized management. Although individual studies report anemia prevalence in TB cohorts, consolidated evidence focused on African hospital settings is limited, despite these contexts having higher disease severity and frequent comorbidities such as HIV, malnutrition, and other infections [8]. Mechanistic synthesis is also insufficient, particularly regarding inflammation-driven disruptions of erythropoiesis and iron metabolism. This review therefore aims to synthesize evidence on the epidemiology and severity of TB-associated anemia in SSA hospitals, clarify key mechanisms (inflammatory pathways, iron dysregulation, nutritional deficits, HIV co-infection, bone marrow suppression), and evaluate clinical implications for disease severity, treatment response, functional outcomes, and survival. It will also appraise current diagnostic and therapeutic practices, identify gaps in care, and propose research and policy priorities to strengthen anemia evaluation and management within TB frameworks. The findings may improve clinical decision-making, inform integrated public health interventions, guide guideline updates, and support investment in diagnostic capacity and targeted supportive therapies.

Epidemiology of TB-Associated Anemia in African Hospitals

TB-associated anemia is highly prevalent in African hospital settings, with hospital-based studies reporting anemia in ~50–80% of admitted pulmonary TB patients. The burden is consistently higher among TB/HIV co-infected patients and tends to be more severe in disseminated or extrapulmonary TB, where inflammatory load and systemic involvement are greater. Moderate-to-severe anemia (Hb <10 g/dL) is frequently observed in hospitalized cohorts, often reflecting late presentation and advanced disease [9]. In African contexts, anemia risk is intensified by overlapping "risk modifiers," including high HIV co-infection rates, chronic malnutrition, helminthic infections, malaria endemicity, limited diagnostic capacity, and delayed health-seeking behavior, making TB-associated anemia rarely mono-causal. Pathophysiologically, the dominant mechanism is anemia of chronic inflammation (AI): persistent immune activation driven by Mycobacterium tuberculosis elevates IL-6, TNF-α, and IFN-γ, with IL-6 inducing hepatic hepcidin. Hepcidin degrades ferroportin, reduces intestinal iron absorption, and traps iron in macrophages, producing functional iron deficiency despite preserved or increased iron stores [10]. Typical laboratory features include low serum iron and transferrin saturation with normal/elevated ferritin, and a normocytic or mildly microcytic picture. True iron deficiency anemia is also common due to poor dietary intake and chronic blood loss (e.g., hookworm, schistosomiasis), and may coexist with AI, complicating ferritin interpretation. In TB/HIV, additional contributors include bone marrow suppression (direct HIV effects, opportunistic infections, ART such as zidovudine) and, in disseminated disease, marrow infiltration causing reduced erythropoiesis and pancytopenia. Nutritional deficits (iron, folate, B12, protein) are frequent in hospitalized patients [11]. Anti-TB drug–related anemia (rifampicin hemolysis, rare isoniazid sideroblastic anemia) occurs but is uncommon relative to inflammatory and comorbidity-driven mechanisms.

Clinical Implications of Anemia in TB Patients

Anemia in tuberculosis (TB) patients has major clinical implications across disease severity, treatment response, survival, and daily functioning. Lower hemoglobin often indicates more advanced TB, correlating with high

mycobacterial burden, extensive lung involvement, elevated inflammatory markers, and disseminated disease. During therapy, several African cohort studies associate anemia with delayed sputum smear conversion, slower clinical recovery, and prolonged hospitalization [12]. Hepcidin-mediated iron sequestration may further influence host immunity and immune–mycobacterial dynamics. Importantly, anemia is an independent predictor of in-hospital mortality and early mortality during TB treatment, including in TB/HIV co-infection; severe anemia (Hb <8 g/dL) markedly increases risk. Beyond survival, anemia contributes to fatigue, reduced exercise tolerance, impaired immune function, and lower work productivity, amplifying household costs and the broader socioeconomic burden of TB [13]. These links support routine hemoglobin screening at diagnosis and follow-up, risk stratification, and earlier supportive care for severely anemic patients. Evaluation should consider nutritional deficiency, chronic inflammation, HIV, helminths, and drug-related marrow suppression. Because inflammation can trap iron, empiric iron therapy should be individualized and guided by iron indices, while transfusion is reserved for symptomatic or life-threatening anemia.

Diagnostic Challenges in African Hospitals

Diagnosing anemia in many African hospitals is challenging because differentiating anemia of inflammation (AI) from iron deficiency anemia (IDA) is difficult in low-resource settings. Key confirmatory tests are often unavailable, including soluble transferrin receptor testing, hepcidin assays, and bone marrow evaluation. As a result, clinicians frequently rely on ferritin; however, ferritin becomes unreliable in the presence of infection or chronic inflammation, which are common in hospitalized patients [14]. A practical approach in resource-limited contexts prioritizes accessible investigations: a complete blood count and peripheral smear as first-line tests, followed by ferritin when available. Because inflammation can distort iron markers, CRP or ESR should be used alongside ferritin to help interpret results. Clinical risk assessment remains essential, considering dietary iron intake, helminth exposure, and comorbidities such as HIV. When anemia is severe, atypical, or unexplained, further evaluation is warranted for prevalent and treatable causes, including malaria, helminth infection, gastrointestinal bleeding, and HIV progression. This stepwise strategy balances diagnostic accuracy with feasibility, supporting timely management despite laboratory constraints [15].

Management Considerations

Effective anti-TB therapy often leads to anemia improvement within 2–3 months as systemic inflammation declines and hepcidin levels fall, allowing better iron mobilization; a rising hemoglobin during treatment is therefore a favorable prognostic sign. Iron supplementation in active tuberculosis remains controversial because iron can potentially enhance mycobacterial growth, excess iron may worsen infection, and iron overload increases oxidative stress. However, true iron deficiency still requires correction, since untreated severe iron deficiency anemia (IDA) increases morbidity and mortality risk [16]. For this reason, iron should be prescribed only when IDA is strongly suspected clinically or confirmed by appropriate testing, rather than routinely for all anemic TB patients. Nutritional support is also essential: integrated interventions can improve weight gain, accelerate hemoglobin recovery, and strengthen immune function, with particular attention to correcting protein–energy malnutrition. In TB/HIV co-infection, early initiation of antiretroviral therapy reduces inflammatory burden, supports bone marrow recovery, and improves survival; zidovudine should be avoided in patients with severe anemia due to its myelosuppressive effects. Blood transfusion is reserved for critical cases, typically hemoglobin <6–7 g/dL, hemodynamic instability, or severe symptomatic anemia, though access to safe transfusion services remains limited in many African health facilities [17].

Special Populations in African Hospitals

Special populations in African hospitals experience distinct tuberculosis (TB)–anemia challenges that demand tailored care. Among pregnant women, TB-associated anemia is linked to higher maternal morbidity and adverse birth outcomes, including preterm delivery and low birth weight, making management particularly sensitive. Clinicians must carefully balance iron supplementation needed to correct anemia against the risk of worsening infection, while ensuring effective TB treatment and close antenatal monitoring. In pediatric TB, children often present late with severe nutritional anemia, frequently compounded by helminth infections and other comorbidities that intensify inflammation and iron dysregulation. Diagnosis is especially challenging because clinical features overlap with malnutrition, parasitic disease, and other causes of childhood anemia, increasing the risk of under- or mis-treatment [18]. TB/HIV co-infected patients carry the greatest burden, with the highest anemia prevalence and mortality due to multifactorial mechanisms such as chronic inflammation, marrow suppression, opportunistic infections, and antiretroviral- or TB drug–related toxicity. Integrated TB-HIV service models, including combined clinics and coordinated follow-up, improve outcomes by streamlining diagnosis, optimizing therapy, and strengthening adherence and supportive care.

Public Health and Health Systems Implications

In African hospital settings, anemia among tuberculosis (TB) patients has major public health and health-systems implications. It is associated with prolonged hospital stays, increased demand for blood transfusions, higher overall healthcare costs, and a greater likelihood of early readmission, placing added pressure on already resource-

constrained services. Because of these downstream burdens, routine anemia screening at the point of TB diagnosis should be implemented as standard care to enable early risk stratification and targeted management [19]. A practical clinical pathway begins with measuring hemoglobin (Hb) at TB diagnosis, followed by classification of anemia severity to guide urgency and intensity of intervention. Given the high burden of comorbidity, HIV status should be assessed, alongside evaluation of nutritional status to identify deficiencies contributing to low Hb and impaired recovery. Clinicians should also investigate for co-infections that may worsen anemia or complicate TB treatment. Finally, Hb should be reassessed at two months to monitor response, detect persistent or worsening anemia, and adjust care accordingly. This algorithm supports timely clinical decision-making while strengthening hospital efficiency and patient outcomes.

Research Gaps

Significant research gaps persist regarding tuberculosis (TB)–associated anemia, particularly in African settings. A major unresolved issue is the identification of optimal, affordable biomarkers capable of reliably differentiating anemia of inflammation (AI) from iron deficiency anemia (IDA) in low-resource environments, where advanced assays are often unavailable [20]. Clarifying the safety and timing of iron supplementation during early anti-TB therapy also remains critical, given concerns that iron may exacerbate infection while untreated anemia worsens patient outcomes. The prognostic utility of hepcidin requires further investigation, especially its potential role as a biomarker for disease severity, treatment response, and anemia stratification. Additionally, the extent to which effective anemia correction improves TB-related morbidity and mortality has not been conclusively determined. Patterns and severity of TB-associated anemia may also differ between rural and urban African hospitals due to variations in nutrition, comorbidities, healthcare access, and delayed presentation, yet comparative data are limited [21]. Addressing these gaps requires large, well-designed prospective cohort studies across diverse African populations to generate context-specific evidence that can inform diagnostic algorithms, therapeutic guidelines, and public health policy.

CONCLUSION

In conclusion, TB-associated anemia remains a common, clinically consequential problem in African settings, yet management is constrained by major evidence gaps. Developing low-cost, point-of-care biomarker strategies to distinguish anemia of inflammation from iron deficiency is central to rational treatment decisions where laboratory capacity is limited. Robust data are also needed on the safety, timing, and dosing of iron supplementation during early anti-TB therapy, balancing potential pathogen support against the harms of persistent anemia. Hepcidin should be validated as a prognostic and stratification marker, alongside other accessible indices, to improve risk prediction and monitoring. Critically, interventional studies must clarify whether correcting anemia translates into lower TB mortality and better functional recovery. Comparative rural–urban cohorts can further define context-specific patterns driven by nutrition, comorbidities, and access to care. Large, well-designed prospective African studies—linked to implementation pathways—are essential to refine diagnostic algorithms, standardize supportive care, and accelerate progress toward equity in TB outcomes. Such evidence will support guideline updates, protect vulnerable patients, and optimize resources across TB programs.

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