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The intersection of HIV and tuberculosis: A comprehensive literature review on co-infection challenges and management

Nafidza Kumala Putri [1], Muhammad Vitanata Arfijanto [2,*] and Rebekah Juniati Setiabudi [3]

[1] *Medical Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.*
[2] *Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga; Dr. Soetomo General Academic Hospital Surabaya, Indonesia.*
[3] *Department of Medical Microbiology, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.*

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Abstract

HIV/AIDS is still a global health problem. HIV weakens the immune system, reducing the body's ability to effectively fight off infections. The risk of developing active tuberculosis (TB) is considerably increased in people living with HIV/AIDS (PLWH). This literature review examines the intersection of HIV and TB, focusing on the epidemiology, pathophysiology, clinical challenges, and management strategies associated with co-infection. With both diseases being significant global health issues, their synergistic impact creates a critical public health burden. The review aims to synthesize current evidence to understand co-infection dynamics and propose pathways for effective management.

Keywords: HIV; Tuberculosis; HIV-TB coinfection; Management

1. Introduction

HIV and TB are two of the world's deadliest infectious diseases, disproportionately affecting low- and middle-income countries. Their intersection, marked by a bi-directional relationship, amplifies the morbidity and mortality associated with each disease. This review explores the burden of HIV-TB co-infection, its underlying mechanisms, and the challenges it poses for healthcare systems.

2. Epidemiology

2.1. Global burden of HIV-TB Coinfection

HIV and tuberculosis (TB) co-infection remains a significant global health challenge, particularly in regions with high rates of both diseases. In 2022, approximately 10.6 million people worldwide were diagnosed with TB, with about 6.3% of these cases being co-infected with HIV, translating to roughly 667,800 individuals (1). The World Health Organization (WHO) reported that TB is the leading cause of death among people living with HIV/AIDS (PLWH), accounting for a substantial number of deaths attributed to HIV/TB co-infection (1,2). In 2022, there were 167,000 deaths due to this co-infection (1).

2.2. Regional variations

The prevalence of TB/HIV co-infection varies significantly by region, with the highest rates reported in Africa (up to 43% and 50-80% in sub-Saharan areas) (3), followed by Latin America (25.06%) (4), Europe (20.11%) (4), Asia (17.21%) (4), and the USA (14.84%) (4).

* Corresponding author: Muhammad Vitanata Arfijanto

Sub-Saharan Africa has the highest rates of HIV-TB co-infection, driven by a combination of interrelated factors. These include high HIV prevalence (approximately 75% of global HIV/AIDS cases) (5,6), widespread poverty (5,7), limited access to healthcare (5), risky behaviors such as multiple sexual partners, substance abuse (including alcohol), and the sharing of contaminated needles (5). Additionally, a lack of awareness and education, along with overcrowded living conditions, contributes to the persistence of this significant public health challenge in the region (7).

3. Risk Factors

3.1. Social Determinants

Social determinants such as poverty, malnutrition, and limited healthcare access significantly contribute to the risk of HIV-TB co-infection. These factors intersect and amplify each other, creating a complex environment that fosters the spread of both diseases.

3.1.1. Poverty

Economic Constraints

Poverty limits access to basic necessities like nutritious food, clean water, and proper housing, all of which are critical for maintaining good health. Malnutrition, a direct consequence of poverty, weakens the immune system, making individuals more vulnerable to infectious diseases like TB (7,8).

Healthcare Disparities

Financial constraints prevent many poor individuals from accessing quality healthcare services, including early detection and treatment of TB and HIV. Delayed diagnoses and incomplete treatments worsen outcomes and increase the risk of co-infection (7).

3.1.2. Malnutrition

Weakened Immunity

Nutritional deficiencies impair the functioning of the immune system, rendering individuals more susceptible to infections (9). Chronic malnutrition can lead to micronutrient deficiencies, such as vitamin D deficiency, which affects bone density but also plays a role in modulating immune responses.

Increased Morbidity

Malnourished individuals experience higher morbidity rates when they contract either HIV or TB (8). Their bodies struggle to fight off these infections efficiently, leading to prolonged illness durations and reduced chances of recovery.

3.1.3. Limited Healthcare Access

Diagnostic Delays

Insufficient healthcare resources result in delays in diagnosing both HIV and TB (7). Timely diagnosis is crucial because prompt treatment reduces the risk of transitioning from latent to active TB and minimizes the progression of HIV to AIDS (10).

Adherence Issues

Patients with limited access to healthcare might find it challenging to adhere to long-term medication regimens required for treating both HIV and TB. Non-adherence leads to treatment failures, resistance, and ultimately worse health outcomes (11).

3.2. Immune Suppression

HIV infection significantly impacts the immune system, leading to an increased susceptibility to TB and a higher risk of reactivation of latent TB infections. This phenomenon is primarily due to the depletion of CD4+ T cells, which play a crucial role in orchestrating immune responses against *Mycobacterium tuberculosi*s (12).

4. Pathophysiology

4.1. Interaction Between HIV and TB

4.1.1. Depletion of CD4+ T Cells

HIV primarily targets CD4+ T cells, leading to their progressive loss. This depletion impairs the host's ability to mount effective immune responses against TB, as CD4+ T cells are essential for activating macrophages and other immune cells that contain *M. tb* (13). The loss of CD4+ T cells increases the risk of latent TB reactivation by approximately 20-fold, as these cells play a vital role in maintaining immune surveillance against opportunistic infections (12).

4.1.2. Altered T Cell Responses

HIV shifts the balance of T helper (Th) cell responses from Th1 (which is protective against intracellular pathogens like *M. tb*) towards Th2 responses, which are less effective in controlling such infections (12). This imbalance reduces the overall effectiveness of the immune response against TB.

CD8+ T cells are important for controlling viral infections and also play a role in controlling *M. tb*. HIV infection can impair the activation and function of CD8+ T cells, further weakening the immune defense against TB (12).

4.1.3. Impaired Macrophage function

HIV infection alters the function of macrophages, which are essential for engulfing and destroying pathogens like *M. tb*. HIV can manipulate macrophage bactericidal pathways, reducing their ability to kill intracellular bacteria and allowing *M. tb* to survive and replicate (14).

HIV infection leads to dysregulated production of pro-inflammatory cytokines such as tumor necrosis factor (TNF)-α, which is necessary for effective macrophage activation against TB. This dysregulation can enhance HIV replication while impairing the macrophage's ability to control TB (14,15).

4.2. Impacts on Disease Progression

4.2.1. Chronic Immune Activation

The inflammatory response triggered by *M. tb* can inadvertently promote HIV replication through elevated levels of cytokines that stimulate viral activity, creating a vicious cycle that exacerbates both infections (12,15). Both HIV and *M. tb* induce chronic immune activation, leading to a state of persistent inflammation that can exhaust immune resources over time. This chronic activation contributes to immunosenescence, characterized by a decline in immune function due to prolonged stimulation (15).

4.2.2. Increased Susceptibility to Opportunistic Infections

The presence of TB can accelerate the progression of HIV disease due to increased immune activation and inflammation, which can lead to a more rapid decline in CD4+ T cell counts (16). This cycle increases susceptibility to additional opportunistic infections. The immunocompromised state resulting from HIV infection increases susceptibility not only to TB but also to other opportunistic infections that can complicate treatment and recovery from either disease.

5. Clinical Challenges

5.1. Atypical Clinical Presentations

In HIV-positive patients, TB often presents in extrapulmonary forms (e.g., pleural, pericardial, or lymphatic TB) rather than the typical pulmonary manifestations. This can lead to misdiagnosis or delayed diagnosis as these forms may mimic other diseases and present with non-specific symptoms such as fever, weight loss, and cough (17).

A high frequency of smear-negative TB is observed in HIV co-infected individuals. Traditional diagnostic methods, such as sputum smear microscopy, may yield negative results despite active infection, leading to underdiagnosis and inappropriate treatment (18,19).

5.2. Limitations of Diagnostic Tools

Conventional diagnostic techniques often lack sensitivity in HIV-positive patients. For example, cultures may take weeks to yield results, delaying treatment initiation (19). Molecular diagnostic methods, though faster and more precise, remain inaccessible in many areas due to high costs and limited resources. As a result, many HIV co-infected individuals lack access to advanced tools like GeneXpert, which offer quicker and more reliable diagnoses. This gap in access contributes to the reliance on less effective traditional methods (19).

5.3. Challenges in Radiologic Diagnosis

The radiographic presentation of TB in individuals with HIV often appears atypical and may lack the classic features, such as cavitary lesions. This can result in clinicians overlooking TB as a possible diagnosis when assessing respiratory symptoms. Furthermore, the overlap between symptoms of HIV-related illnesses and TB, along with similar presentations of conditions like pneumonia or lung cancer, adds to diagnostic challenges and confusion (18).

5.4. Increased Risk of Drug Resistance

HIV infection raises the risk of MDR-TB (20). HIV-TB co-infections significantly contribute to the development and spread of drug-resistant tuberculosis (MDR-TB) through a combination of immunological, treatment-related, behavioral, and pharmacokinetic factors. The immune suppression caused by HIV, particularly CD4+ T cell depletion (21,22) and chronic immune activation (23), undermines the body's ability to control TB infections, fostering an environment conducive to resistance. Treatment complexities, including interruptions, non-adherence, and adverse drug interactions, further exacerbate the issue, allowing TB bacteria to survive and evolve resistance.

5.5. Drug Interactions between Antiretroviral Therapy (ART) and TB Medications

Rifampin, a first-line TB medication, is a potent inducer of cytochrome P450 (CYP) enzymes, particularly CYP3A4. This induction can significantly reduce the plasma concentrations of several antiretroviral drugs, including protease inhibitors (PIs) and non-nucleoside reverse transcriptase inhibitors (NNRTIs), potentially leading to loss of antiviral efficacy and the development of viral resistance (24,25). Due to these interactions, rifabutin is often recommended as an alternative to rifampin for patients on ART because it has fewer interactions with antiretrovirals (25). However, rifabutin is not always available or affordable in resource-limited settings, which can limit treatment options.

Antiretroviral drugs and TB medications often have overlapping side effects, including hepatotoxicity, gastrointestinal disturbances, and skin rashes, which can amplify toxicity and may require discontinuation of one or both treatments (25). Additionally, starting ART in patients with active TB can trigger immune reconstitution inflammatory syndrome (IRIS), where the recovering immune system launches an inflammatory response to previously unrecognized opportunistic infections. This condition adds complexity to the clinical management of co-infected patients (24).

6. Management Strategies

6.1. Diagnosis and Screening

Integrated testing strategies for the simultaneous detection of HIV and tuberculosis (TB) play a crucial role in improving health outcomes for individuals at risk of co-infection. These approaches enable early diagnosis, promote treatment adherence, and optimize healthcare resources. Community-based interventions, such as household contact management, have proven effective. For instance, trained community health workers can visit TB patients' households to offer both TB screening and voluntary HIV testing. Studies in Cameroon and Uganda have shown high acceptance rates of HIV testing among household contacts, particularly among those unaware of their HIV status (26).

Involving community organizations in planning and implementing integrated services increases awareness and demand for both HIV and TB testing. Advocacy efforts aimed at reducing stigma associated with both diseases can also improve participation in testing programs (27).

Screening efforts should prioritize high-risk groups who are more vulnerable to HIV-TB co-infection (28). These include individuals with a history of TB exposure or previous TB disease, as they are at greater risk of reactivation. People living with HIV, especially those with low CD4 counts, are also a key focus due to their compromised immune systems. Additionally, homeless individuals or those residing in congregate settings, such as shelters, face heightened transmission risks, making targeted screening essential in these populations.

The use of rapid diagnostic tests is another critical component. The GeneXpert MTB/RIF, a rapid molecular test, is recommended for individuals suspected of having TB, especially those living with HIV. This test provides quick results and detects both TB and rifampicin resistance, allowing for timely initiation of treatment (27).

6.2. Therapeutic Approaches

The standard treatment for active TB in HIV-positive individuals typically involves the RIPE regimen, which includes Rifampin (RIF), Isoniazid (INH), Pyrazinamide (PZA), and Ethambutol (EMB) during an initial two-month phase. This is followed by a continuation phase using RIF and INH for an additional four to seven months, depending on the patient's clinical response and culture results (29). For individuals with multidrug-resistant TB (MDR-TB), treatment requires the use of second-line drugs and specialized management to address the added complexities of HIV co-infection (30).

ART should ideally be commenced within the first two weeks of starting TB treatment for patients with CD4 counts <50 cells/mm^3. For those with CD4 counts ≥50 cells/mm^3, ART should be initiated within 8 to 12 weeks of starting TB therapy (29,31). In cases of HIV-associated TB meningitis, ART should be delayed and not initiated during the first 8 weeks of anti-TB treatment to minimize the risk of immune reconstitution inflammatory syndrome (IRIS) (29).

Fixed-dose combinations (FDCs) offer significant benefits in managing HIV-TB co-infection by simplifying treatment regimens. By combining multiple medications into a single pill, FDCs reduce the daily pill burden, which is especially advantageous for patients dealing with complex HIV and TB treatments. Simplified regimens help improve adherence and decrease the likelihood of treatment failure due to poor compliance, drug resistance, or overlapping toxicities (32,33). Studies have shown that FDCs, such as Atripla (emtricitabine, tenofovir, and efavirenz), are associated with higher adherence rates and better virological suppression, along with improved immunological recovery in co-infected patients (32).

The clinical efficacy and safety of FDCs have also been demonstrated in trials involving HIV-TB co-infected individuals. For example, the combination of efavirenz with rifampicin has been extensively studied, showing no significant virological failures despite a reduction in efavirenz plasma levels caused by rifampicin's enzyme-inducing effects (32). Additionally, FDCs may reduce the risk of adverse effects linked to the use of multiple medications. However, careful monitoring is essential to address potential drug interactions between antiretroviral and anti-TB drugs, particularly concerning liver toxicity and overlapping side effects (33).

6.3. Preventive Strategies

For individuals living with HIV who have a positive tuberculin skin test (TST) or are at high risk for tuberculosis (TB), isoniazid preventive therapy (IPT) is recommended to reduce the likelihood of developing active TB. This can involve a regimen of 6 to 9 months of daily isoniazid (INH), which has been shown to decrease the risk of progression to active TB by approximately 33% (34). Alternatively, a 3-month short-course regimen of once-weekly isoniazid plus rifapentine (3HP) has proven to be both effective and well-tolerated (29,34). Another option is 4 months of daily rifampin (4R); however, this regimen may not be suitable for all patients due to potential drug interactions with antiretroviral therapy (ART) (29).

7. Conclusion

The intersection of HIV and tuberculosis (TB) represents a critical public health challenge due to the synergistic impact of these two diseases, particularly in low- and middle-income countries. The bi-directional relationship between HIV and TB exacerbates disease progression, increases morbidity and mortality, and presents significant diagnostic, therapeutic, and management challenges. Epidemiological trends highlight the disproportionate burden of co-infection in regions with limited healthcare access, while pathophysiological insights reveal the complex interactions between the pathogens, underscoring the need for integrated management strategies.

Effective management of HIV-TB co-infection requires a multidisciplinary approach, including timely diagnosis, tailored therapeutic interventions, and preventive measures such as isoniazid preventive therapy. The integration of HIV and TB healthcare services, community-based interventions, and robust public health policies are pivotal in addressing this dual burden. Advancements in diagnostics, such as GeneXpert, and simplified treatment regimens, including fixed-dose combinations, offer promise in improving outcomes for co-infected individuals.

Moving forward, increased research efforts, targeted global funding, and strengthened healthcare infrastructure are essential to reduce the burden of HIV-TB co-infection. Collaborative initiatives and community engagement are also critical to overcoming barriers such as stigma and treatment adherence issues. By prioritizing comprehensive and

integrated approaches, significant progress can be made toward mitigating the impact of this co-infection and improving the health and well-being of affected populations worldwide

Compliance with ethical standards

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No conflict of interest to be disclosed.

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