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Review Article

Prevalence of Tuberculosis Among HIV Patients

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Abstract

Tuberculosis (TB) remains a leading cause of illness and death among people living with HIV, particularly in countries with high dual prevalence. HIV-induced immune suppression increases susceptibility to new TB infections, reactivates latent TB, and accelerates progression to active disease. This review provides a comprehensive overview of the microbiological, clinical, and epidemiological aspects of TB-HIV coinfection. Key contributing factors to TB in HIV-positive individuals include malnutrition, tobacco use, advanced age, exposure to infectious TB cases, and poor living conditions. Diagnostic limitations and atypical presentations complicate timely detection. Treatment is further challenged by drug interactions, adverse effects, immune reconstitution inflammatory syndrome (IRIS), and rising drug resistance. Integrated strategies—such as early initiation of antiretroviral therapy (ART), routine TB screening, TB preventive therapy (TPT), and coordinated healthcare services are essential to improving outcomes. Strengthening public health infrastructure and implementing targeted prevention and treatment interventions are critical for reducing the dual burden of TB and HIV.

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1. INTRODUCTION

In 2004, approximately 9 million people worldwide contracted tuberculosis (TB) for the first time, and the disease claimed the lives of almost 2 million. Currently, when AIDS and malaria are excluded, TB causes more years of lost healthy life globally (2.5% of all disability-adjusted life years, or DALYs) than any other infectious disease [1]. Over the centuries, tuberculosis (TB) has been a major health concern and has caused more human misery, suffering, and loss of income as well as failure of economic and social development than any other disease [2].

Annually, there are approximately eleven million active cases of the disease, and three million deaths are attributed to TB [3].

Microbiology

M. Tuberculosis, M. Bovis (and bacillus Calmette-Guérin), M. Canetti, M. Africanus, and M. Microti are the five types of species that make up the M. Tuberculosis complex. The species complex's variations are biochemically and culturally distinct from the type strain. These variations, however, are not known to affect prognosis or management. The main exception is M. Bovis, which causes a small percentage of human tuberculosis cases but is inherently resistant to the medication pyrazinamide, which is why it shouldn't be used for treatment. Atypical mycobacteria, or mycobacteria other than M. tuberculosis (MOTT), can also cause human disease. These organisms have been isolated and are found in large quantities in nature.[4]

Transmission

Transfer of Although live tubercle bacilli are carried by M. tuberculosis patients, the bacteria may also be present in trace amounts and latent, meaning that no disease symptoms may be visible. When bacteria proliferate, evade the immune system, and become numerous enough to harm tissues, disease results. The primary source of infection is patients with pulmonary tuberculosis (PTB). Inhaling droplet nuclei, which are infectious particles of respiratory secretions that typically measure less than 5 micrometres and contain tubercle bacilli, is how infection is contracted. They can linger in the air for extended periods of time and are dispersed by coughing, sneezing, talking, spitting, and singing. Tubercle bacilli are killed in minutes by direct sunlight. However, they can endure longer periods of time in dim, poorly ventilated environments. Due to their small size, droplet nuclei can bypass the bronchi's defenses and enter the lungs' terminal alveoli, where infection and multiplication start.[5]

Etiology of tuberculosis

Causes of Tuberculosis The acid-fast bacillus Mycobacterium tuberculosis (M. tuberculosis) is the cause of tuberculosis. It mainly affects the lungs but can spread to other organs such as the lymph nodes, bones, central nervous system, and gastrointestinal tract. The disease is spread by airborne particles released during a cough, sneeze, or speech by an infected person. The bacilli are taken up by macrophages in the alveoli after being inhaled. Even though immune containment keeps many infections dormant, a sizable portion can develop into active tuberculosis, particularly in immunocompromised hosts [6]. Both host-related and microbial factors impact the etiology. Different strains of M. tuberculosis have varying ranges of bitterness and hostility, especially those that belong to the M. tuberculosis complex, which includes M. africanus, M. bovis, and M. microti. These strains may all contribute to distinct clinical presentations.[7] Genetic predisposition, co-morbid conditions such as HIV, malnourishment, diabetes mellitus, smoking, and chronic kidney disease all contribute to host susceptibility by lowering the immune system's capacity to contain latent infection [8]. When host defenses weaken, latent TB can reactivate and cause extrapulmonary or pulmonary illness. Almost any organ can be affected by extrapulmonary tuberculosis (EPTB), which is more prevalent in immunocompromised individuals.[9] Notably, in certain areas, M. Bovis, which is frequently spread by unpasteurized dairy, is a contributing factor to zoonotic TB cases [10]. Environmental and socioeconomic factors that raise exposure risk and decrease early detection and treatment, such as inadequate ventilation, crowded living conditions, and restricted access to healthcare, are also taken into account in the etiopathogenesis.[11] According to recent molecular research, M. tuberculosis uses immune evasion techniques like blocking phagosome–lysosome fusion and causing granuloma formation to prolong its persistence [12]

Pathogenesis of tuberculosis

The pathogenesis. The risk of progression from infection to disease depends on the host's immune status and the amount of tubercle bacilli that are used to infect the host. A pathological lesion, which is typically localized and frequently accompanied by extensive tissue destruction and cavitation, is the result of the patient's immune response. Post-primary TB can emerge in any organ system where tubercle bacilli spread during the initial infection, typically due to the reactivation of those dormant bacteria. A person who already has a latent infection may develop active disease as a result of secondary or exogenous reinfection.[13]

Signs and symptoms of tuberculosis

Typical signs of tuberculosis include: Prolonged coughing, chest pain, weakness, exhaustion, fever, and night sweats. These symptoms are frequently mild for several months, which causes people to put off getting help and raises the possibility of infecting others. A patient will be sent for testing if the medical professional believes they may have tuberculosis. Patients who are suspected of having lung tuberculosis will be asked to provide a sample of their sputum for TB bacterial testing. Samples of afflicted bodily fluids and tissue can be tested for non-lung tuberculosis. Rapid molecular diagnostic tests are advised by the WHO as the first tests for individuals exhibiting TB symptoms. Sputum smear microscopy and chest X-rays. Individuals infected with TB do not exhibit any TB symptoms or indicators. Healthcare professionals may use a skin or blood test to check for TB infection, and they will screen at-risk patients to rule out active TB.[14]

Treatment for Tuberculosis

Therapy for tuberculosis (TB) First-line anti-TB medications (for TB that is susceptible to drugs) A typical 6-month regimen consists of: Intensive Phase (two months): Ethambutol €, Pyrazinamide (Z), Isoniazid (H), and Rifampicin ® Phase of continuation (4 months): Isoniazid (H), Rifampicin ® Guidelines for Dosage: 10 mg/kg of rifampicin (maximum 600 mg) 5 mg/kg of isoniazid (maximum 300 mg) 25 mg/kg of pyrazinamide 15–20 mg/kg of ethambutol [15] Therapy for

Particular TB Cases MDR/XDR drug-resistant tuberculosis: MDR-TB: Isoniazid and rifampicin-resistant 18–24 months of second-line medication treatment (e.g., Bedaquiline, Linezolid, Levofloxacin)[16].TB in Patients with HIV: Same 6-month Treatment. If CD4 is less than 50 cells/mm³, begin ART within two weeks. Keep an eye out for medication interactions, particularly with rifampicin.[17] Latent TB Infection (LTBI): Rifampicin + Isoniazid for 3 months OR Rifampicin for 4 months OR Isoniazid for 6–9 months. Monitoring and Sputum Test Follow-Up at 2,5, and 6 Months. Track adherence, liver function, and vision (ethambutol) [18]

Prevention of tuberculosis:

TB prevention Vaccination against Bacillus Calmette-Guérin (BCG). What it is: a vaccine that is mostly administered to infants and kids in nations where tuberculosis is highly prevalent. Drawbacks: Limited ability to prevent adult pulmonary tuberculosis[19]. Strategies for Infection Control in Healthcare and Community Settings: Mask use and adequate ventilation, coughing manners, Quick detection and isolation of cases of infectious tuberculosis[20].Latent TB Infection Treatment (LTBI) The objective is to stop the latent TB infection from becoming an active Illness. Typical routines: 6–9 months of isoniazid, four months of rifampin, Weekly Isoniazid + Rifapentine for 3 months [21]. Screen people who have had close contact with infectious TB patients using the active case finding and contact tracing approach. Importance: Reduces community transmission by facilitating early diagnosis and treatment[22]. Taking Social Determinants into Account: Cutting down on crowding, enhancing dietary intake dealing with HIV coinfection [23].TB Preventive Therapy in High-Risk Populations: Healthcare Workers, HIV-positive Individuals, and Household Contacts of TB Patients WHO Suggestion: In high-burden environments, administer TB preventive treatment (TPT).[24]

Human immunodeficiency virus:

An Overview of HIV The retrovirus known as HIV targets the body's immune system, particularly the CD4+ T cells, which are essential for immune defense, and causes a chronic, progressive illness. Untreated HIV infection results in acquired immunodeficiency syndrome (AIDS), a disorder in which the body's defenses against opportunistic infections are severely weakened[25]

Biological Mechanism

HIV is a member of the Retroviridae family's Lentivirus genus. After entering the body, it uses reverse transcriptase to incorporate its RNA into the host DNA, enabling viral replication. HIV can spread via breast milk, semen, vaginal and rectal fluids, and blood. Typical means of transmission consist of transfusions of contaminated blood products, sharing of needles, unprotected intercourse, and transmission from mother to child during childbirth or breastfeeding. Infection Stages: 1. Acute HIV Infection: Symptoms include flu-like symptoms and appear 2–4 weeks after exposure. 2. Clinical Latency (Chronic HIV): The virus is still active but reproduces at low levels; symptoms may not appear for ten years or longer. 3. AIDS: The last and most severe stage, in which the CD4 count drops below 200 cells/mm³, making the patient more vulnerable to infections.[26]

HIV Origination

HIV originated from a particular kind of chimpanzee that lived in Central Africa, according to the Centers for Disease Control and Prevention (CDC)Trusted Source. Reasons and dissemination of HIV can be acquired by coming into contact with bodily fluids. Most people contract HIV from: Anal intercourse, Vaginal intercourse, sharing syringes and needles. HIV can also be acquired during pregnancy, childbirth, and breastfeeding. The risk of being exposed to HIV can be increased by several factors. The following are some typical risk factors for HIV exposure: Reliable Source: Vaginal or anal intercourse without the use of a barrier, sharing needles the existence of an additional STI, Unintentional needlestick injuries, especially among medical personnel, Transfusions or injections that are not sterile[27].

Signs and symptoms

The signs and symptoms of HIV and AIDS can vary significantly depending on the individual and how far along the infection has progressed. The initial stage of HIV infection, often called acute HIV or primary infection, can cause flu-like symptoms in some people. These symptoms typically appear within two to four weeks of becoming infected with HIV. Fever is one of the possible symptoms. A headache. Joint and muscle pain. Rash. Mouth sores and a sore throat. Nodes, or enlarged lymph glands, are mostly found on the neck. Diarrhea. Reduction of weight. Cough. Sweats at night. You may not even notice these symptoms because they can be so subtle. Subsequently, the infection spreads to others more quickly during the initial infection than in the continuous phase. Chronic HIV is another name for clinical latent infection. However, many people do not exhibit HIV-related symptoms or infections during this time. For those who are not receiving antiretroviral therapy, or ART, this stage may persist for many years. More severe disease strikes some people much earlier. HIV infection symptoms. You may experience minor infections or chronic symptoms like fever as the virus keeps growing and destroying immune cells. Exhaustion. One of the earliest signs of HIV infection is frequently enlarged lymph nodes. Diarrhea. Reduction of weight. Herpes zoster is another name for shingles. Pneumonia. Development of AIDS Globally, AIDS-related deaths have significantly decreased due to improved antiviral therapies. The majority of HIV-positive individuals in the United States today do not develop AIDS because of these life-saving therapies. HIV develops into AIDS in 8–10 years if left untreated. Your immune system is severely compromised if you have AIDS. Diseases that people with AIDS wouldn't contract if their immune systems were healthy are more likely to strike them. Persistent fever. Persistent diarrhea. Enlarged lymph nodes. lesions or white spots on the tongue or within the

oral cavity. Constant exhaustion. Weakness. Quick weight loss. Skin lumps or rashes.[28]

Pathophysiology of HIV

HIV, a member of the Reoviridae family, stands apart from other viruses due to its unique mechanism: its RNA genetic material is converted into DNA through a process called reverse transcription. This newly formed viral DNA then becomes a permanent part of the infected host cell's own genetic code, establishing a persistent, lifelong infection. This mechanism is best characterized in HIV-1, although recent research has also highlighted key differences in HIV-2 and HTLV replication and biology. The primary cellular receptor for both HIV-1 and HIV-2 is the CD4+ antigen, making CD4+ T cells and macrophages the main targets for infection. The virus uses envelope glycoproteins to bind to CD4+ receptors and engage co-receptors CCR5 or CXCR4, triggering structural changes that facilitate fusion of the viral envelope with the host cell membrane.

Contrary to earlier understanding, reverse transcription is now believed to occur within the nucleus or during capsid transport to the nucleus, rather than solely in the cytoplasm. The intact or nearly intact capsid travels to the nuclear pore complex, where it either begins uncoating or partially uncast to initiate viral DNA synthesis near the nuclear envelope. Some studies suggest that both uncoating and reverse transcription occur within the nucleus, though this process is not yet fully understood. The reverse transcriptase enzyme, utilizing host tRNA primers, initiates the synthesis of negative-sense single-stranded DNA from the viral RNA in a 5' to 3' direction. This is followed by the synthesis of the complementary strand to form double-stranded DNA, which is then integrated into the host genome by the viral integrase enzyme, often at random locations. Once integration occurs, the host cell machinery can produce new viral particles, enabling the spread of infection to other cells.

Genetic diversification is a critical aspect of HIV pathogenesis, influencing disease progression and resistance to antiretroviral therapy (ART). A key factor in this variability is the high error rate of HIV-1 reverse transcriptase, particularly in group M, subtype B, which is estimated to be 100–1000 times higher than that of normal cellular DNA polymerases. Additionally, recombination events, host restriction factors, and limited availability of deoxynucleotide triphosphates (dNTPs) further contribute to viral mutation and diversity, often resulting in ART resistance and treatment failure.[29]

Because there is no host immune response and a large number of susceptible CD4+ T cells, viral replication is rife during the early stages of infection, leading to an exponential rise in the plasma HIV RNA level. Following this decline, the initial strong immune response and related symptoms go away, but HIV replication settles when infection and replication continue. We still don't fully understand the precise mechanisms underlying humoral immunity failure. Even with antiretroviral therapy (ART), individuals living with HIV often experience compromised humoral immunity and persistent viral reservoirs. Researchers believe this is linked to the activity of T cells found within B-cell follicles, specifically follicular T-helper cells and follicular regulatory T cells. Furthermore, follicular CD8+ cytotoxic T cells are less common in these patients compared to their counterparts outside the follicles. This imbalance is also thought to play a role in disrupted immune responses in HIV-positive individuals.[30,31]

Life Cycle of HIV

Further, HIV binds to and enters host T cells, it produces HIV RNA and enzymes into the host cell. HIV reverse transcriptase converts viral RNA into proviral DNA. HIV integrase helps the proviral DNA integrate into the host's DNA after it enters the nucleus of the host cell. HIV RNA and HIV proteins are subsequently produced by the host cell. HIV protease transforms the immature virion into a mature, contagious virus by cleaving viral proteins.[32]

HIV PREVENTION:

There are certain actions one can take to lessen the risk of contracting HIV from another person. The following are some suggestions to avoid infection: Using male or female condoms when having penetrative sex, getting tested for additional STIs, never sharing needles with others taking PrEP, and postexposure prophylaxis if someone is at risk of infection. To help prevent HIV transmission to the fetus, pregnant individuals with HIV should take HIV medications as directed by their doctor. After birth, a doctor will administer 4–6 weeks Trusted Source of HIV medications to a baby who has a biological parent with HIV. This reduces the risk of transmitting HIV to the baby to 1% or less.[33]

TREATMENT

Antiretroviral Therapy (ART):

Antiretroviral therapy (ART) is the use of medications to treat HIV infection. It involves taking a combination of drugs daily and is recommended for all individuals diagnosed with HIV. While ART does not cure HIV, it significantly improves quality of life by helping people with HIV live longer, healthier lives. Additionally, ART lowers the risk of transmitting the virus to others.

HIV medications work by reducing the concentration of virus (viral load) in the body. This helps in two important ways:

Strengthening the immune system: By lowering the viral load, the immune system has a chance to recover and become strong enough to fight infections and HIV-related cancers. Reducing transmission risk: Reducing the concentration. of the virus in the body makes it much less likely to spread HIV.

Classes of HIV Medicines

HIV drugs are grouped into different classes based on how they act against the virus. Many of them target and block enzymes that HIV uses to replicate itself, thereby reducing viral load. These include:

Nucleoside Reverse Transcriptase Inhibitors (NRTIs) – inhibit the reverse transcriptase enzyme.

Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs) – attach to and alter reverse transcriptase.

Integrase Strand Transfer Inhibitors (INSTIs) – prevent the action of the integrase enzyme.

Protease Inhibitors (PIs) – block the protease enzyme.
Other HIV treatments prevent the virus from entering CD4 immune cells:

CCR5 Antagonists and Post-Attachment Inhibitors – CCR5 antagonists and post-attachment inhibitors work by blocking specific molecules on CD4 cells that HIV typically uses to enter the cell. Separately, attachment inhibitors function by binding to certain proteins on HIV's outer surface, thereby preventing the virus from attaching to and infecting target cells. Together, these different classes of medications are crucial for controlling HIV and helping to maintain the body's immune function the cells.[34]

Entry into CD4 T Cells
Some HIV medications prevent the virus from entering CD4 T cells, which are pivotal to the immune system. Examples of these entry inhibitors include:
Enfuvirtide (Fuzeon), Maraviroc (Selzentry), Newer medications: Ibalizumab-uiyk (Trogarzo) and Fostemsavir (Rukobia)

Starting and Adhering to Treatment
Antiretroviral therapy (ART) is recommended for everyone diagnosed with HIV, regardless of CD4 T cell count or the presence of symptoms. Beginning treatment early and maintaining consistent therapy is the most effective way to stay healthy and control the virus.
Adhering strictly to your ART regimen is crucial. Missing doses or skipping medication can reduce the effectiveness of the treatment. Keeping an undetectable viral load by taking ART consistently provides several key benefits:
Helps maintain a strong immune system, reduces the risk of infections, and prevents the development of drug-resistant HIV strains.

Managing Challenges with ART
Staying on HIV treatment can sometimes be difficult. If you're experiencing side effects, difficulty remembering doses, or mental health or substance use issues, it's important to speak openly with your healthcare provider. Together, you can find strategies or alternative treatments to help you stay on track.
Regular medical follow-ups are essential to monitor your progress and manage any complications. Notify your healthcare team promptly if you encounter problems with your therapy.

Common Side Effects of HIV Treatment
Some people on ART may experience side effects, including:
Nausea, vomiting, or diarrhea; Heart complications; Kidney or liver damage; Bone weakening or loss; Abnormal cholesterol levels; Increased blood sugar; Cognitive, emotional, or sleep-related issues
While these effects can occur, many can be managed with the help of your healthcare team. Prompt communication and proper support can make ART safer and more effective in the long term.[35]

Prevalence of Tuberculosis (TB) in HIV-Infected Individuals
The emergence of HIV/AIDS has significantly impacted the epidemiology of tuberculosis (TB). HIV increases the likelihood of reactivating latent TB, heightens the risk of TB infection upon exposure to Mycobacterium tuberculosis (MTB), and accelerates progression from latent infection to active disease. In individuals without HIV, the lifetime risk of developing active TB after infection is about 5–10%, with the greatest risk occurring within the first five years. However, among those co-infected with HIV, this risk rises dramatically, with annual rates of active TB development exceeding 30%.[36]
Between 2000 and 2010, the probability of rapid progression to active TB after infection increased from below 10% to approximately 25% due to HIV coinfection.[37] Age also influences TB risk. Children aged five to fifteen are relatively resistant, but the risk rises again during adolescence and remains steady in adulthood, increasing once more in the elderly. Other factors contributing to TB progression include undernutrition, exposure to harmful substances (such as tobacco, alcohol, and immunosuppressive drugs), and comorbid conditions like diabetes, silicosis, leukemia, measles, and whooping cough in children. Despite these, HIV remains the strongest risk factor for the development of active TB disease following MTB infection.[38]

Risk Factors That Increase the Susceptibility of HIV Patients to Tuberculosis (TB)
Multiple studies have identified key risk factors that increase the vulnerability of HIV-positive individuals to tuberculosis. Understanding these factors is crucial for effective allocation of healthcare resources and prioritizing TB control efforts. Among all, HIV infection remains the strongest independent risk factor associated with both pulmonary and extrapulmonary TB, contributing significantly to the ongoing transmission of TB in communities.[39]

- Gender: Research conducted in Eastern Uganda (Cahn, 2019) revealed that gender may influence TB incidence in HIV-positive individuals. Women having HIV were found to be more susceptible to TB than men, due to hormonal influences of TB cases in men.[40]
- Age: A cross-sectional survey in East Africa identified older age as a contributing factor, accounting for 23,000 TB cases, largely due to weakened immune systems among the elderly. It was concluded that HIV-positive individuals who become infected with TB have up to a

50% chance of developing active TB within just two months.[41]

- Consumption of Animal Products: In Botswana's dairy-producing regions, a study found a strong association between TB (caused by Mycobacterium bovis) and consumption of unpasteurized or half-cooked milk. Around 70% of HIV-positive individuals who consumed such milk developed a form of TB over 10 years. Additionally, TB was found to be linked with body wasting, which is common in co-infected patients.[42]
- Exposure to TB Patients: A study in Pakistan found that HIV-positive individuals living with TB-infected persons (e.g., friends, family members, cellmates) were 3.5 times more likely to contract TB than those living with healthy individuals.
- Malnutrition: Malnutrition weakens the immune system and significantly increases the risk of TB in HIV patients. TB itself can also lead to malnutrition, creating a vicious cycle. Poor nutrition, especially diets low in calories, reduces survival rates in co-infected individuals. Supplementation with vitamins and zinc has shown potential in reducing mortality. Furthermore, intestinal parasites and malnutrition may suppress the immune system to the extent that they cause false-negative results on tuberculin skin tests (TST), complicating TB diagnosis.[43]
- Smoking: Around 30% of HIV-positive individuals exposed to either active or passive tobacco smoke are at increased risk of TB infection and progression to active TB disease. Smoking weakens the immune system and impairs the cilia in the airways, reducing the body's ability to clear pathogens.[44]

HIV-positive smokers are:

25% more likely to experience respiratory symptoms (cough, dyspnea)

More prone to severe lung involvement (e.g., upper zone opacities, cavities, miliary TB)

More likely to have positive sputum cultures

At greater risk of TB relapse and mortality

A meta-analysis reported that HIV-positive smokers had a 73% higher risk of TB infection and were more than twice as likely to develop active TB compared to non-smokers. Overall, they were 40–60% more likely to develop active TB after TB infection than HIV-negative smokers.[45]

Complications of Tuberculosis in People Living with HIV (PLHIV)

The coexistence of tuberculosis (TB) and HIV presents a complex and challenging health scenario, with far-reaching clinical, public health, and socioeconomic consequences. The relationship between the two infections is synergistic, where each disease worsens the progression and outcomes of the other. The high prevalence of TB among PLHIV contributes to a vicious cycle of illness and burden that requires urgent, integrated healthcare strategies.

- **Increased Morbidity and Mortality**

In 2023, approximately 161,000 deaths were attributed to HIV-associated TB. Faster HIV Progression: Active TB triggers chronic immune activation, which accelerates HIV replication, speeds up CD4+ T-cell depletion, and hastens the onset of AIDS, increasing the risk of death.[46]

- **Unusual and Difficult-to-Diagnose TB Presentations**

Extrapulmonary and Disseminated TB: Immunosuppression in PLHIV increases susceptibility to TB outside the lungs, such as in the lymph nodes, brain, or bones. Over 50% of TB cases in PLHIV are extrapulmonary or disseminated, making them harder to detect and treat. Low Bacillary Load in Sputum: In advanced HIV, the sputum often contains fewer TB bacteria, resulting in pauci-bacillary samples that are harder to detect using standard diagnostic tools like smear microscopy. False-Negative Test Results: Immunosuppression can lead to false negatives in diagnostic tests such as the Tuberculin Skin Test (TST) and Interferon-Gamma Release Assays (IGRA), complicating latent TB diagnosis.[47]

- **Challenges in Treatment and Management**

Drug-Drug Interactions: Co-treatment with ART and TB drugs (e.g., rifampicin) is complicated by interactions that affect drug effectiveness, requiring careful medication selection and dosage adjustments. Increased Side Effects: PLHIV are more prone to experiencing adverse drug reactions from both ART and TB medications, which can hinder adherence and treatment success. Immune Reconstitution Inflammatory Syndrome (IRIS): Starting ART in TB-infected patients can cause TB-IRIS, a paradoxical inflammatory reaction due to immune recovery, which may worsen TB symptoms and can be severe or life-threatening. Higher Risk of Treatment Failure and Relapse: Co-infection is linked to lower success rates, and even after successful treatment, PLHIV face a greater risk of TB relapse compared to HIV-negative individuals [48]

- **Emergence of Drug-Resistant TB**

Multidrug-Resistant (MDR-TB) and Extensively Drug-Resistant TB (XDR-TB): The complexity of treating HIV and TB together, especially when adherence is poor, increases the emergence of resistant TB strains. These forms are harder to treat, require longer and more expensive regimens, and are associated with poorer health outcomes.[49]

- **Public Health and Socioeconomic Burdens**

Ongoing Community Transmission: PLHIV with active TB are a major source of continued TB spread, particularly in overcrowded or resource-limited settings. Pressure on Healthcare Systems: Managing dual epidemics of TB and HIV places a significant strain on healthcare infrastructure, including diagnostics, specialized care, and long-term monitoring.

Financial Hardship: The burden of co-infection results in substantial economic challenges for individuals, families, and health systems, due to long illness duration, reduced productivity, and high treatment costs.

Social Stigma and Discrimination: The stigma surrounding both HIV and TB can lead to isolation, discrimination, and reluctance to seek medical care, hindering early diagnosis and adherence to treatment.
Reduced Quality of Life: The chronic nature of both diseases, along with their complications and side effects, severely impacts the physical, emotional, and social well-being of those affected.[50]

Prevention of Tuberculosis (TB) Among People Living with HIV (PLHIV)

Preventing the spread and impact of tuberculosis (TB) among people living with HIV (PLHIV) is a critical global health priority. Since HIV is the strongest known risk factor for progression from latent TB infection to active disease, a comprehensive and integrated approach is essential. Effective prevention requires combining HIV and TB prevention, early diagnosis, and appropriate treatment strategies.

- **Early HIV Diagnosis and Treatment**

HIV Testing and Counselling: Routine HIV testing, especially for high-risk groups, along with counseling to promote safer behaviors, is vital. Early diagnosis allows for timely initiation of care. Antiretroviral Therapy (ART): Starting ART early is key to preventing TB. ART strengthens the immune system, dramatically reducing the risk of TB development by as much as 65%. For those with low CD4 counts, starting ART promptly is especially important.[51]

- **Active TB Case Detection**

Routine TB Screening: All patients visiting HIV clinics, ART centres, or healthcare facilities should be screened for TB symptoms. Adults and adolescents should be evaluated for cough, fever, weight loss, and night sweats. In children, symptoms include cough, fever, poor weight gain, and known contact with a TB patient.
Rapid molecular tests like Nucleic Acid Amplification Tests (NAAT) are recommended.
HIV Testing for TB Patients: All individuals diagnosed with TB should also be tested for HIV to ensure early detection and integrated care.[52]

1. TB Preventive Treatment (TPT)

Preventive Therapy Regimens: Preventive TB treatment helps stop latent TB from becoming active disease in HIV-positive individuals. Common regimens include:
Isoniazid alone (INH): Daily for 6–9 months.
3HP (Isoniazid + Rifapentine): Once weekly for 3 months.
1HP (Isoniazid + Rifapentine): Daily for 1 month.
3HR (Isoniazid + Rifampicin): Daily for 3 months.
4R (Rifampicin only): Daily for 4 months.
Eligibility and Initiation: TPT is started after ruling out active TB. In many high TB-burden areas, testing like TST or IGRA is not required; a clinical assessment is sufficient. Combining with ART: TPT used alongside ART further reduces the risk of TB and lowers mortality rates.[53]

2. TB Infection Control in Healthcare and Communities

Administrative Measures: Early identification and isolation of suspected TB cases, fast-tracking coughing patients, promoting cough etiquette, and minimizing hospital stays for infectious patients.
Environmental Measures: Improving natural or mechanical ventilation in healthcare and congregate settings to reduce airborne TB spread. UV lights and proper facility layout may be used.
Personal Protection: Healthcare workers should wear respirators such as N95 masks when treating suspected or confirmed TB cases.
Healthcare Worker Screening: Routine screening for TB among healthcare staff helps detect and control infections early.[54]

3. Integrated TB/HIV Services and Support

Service Integration: TB and HIV prevention, diagnosis, and treatment services should be co-located or closely coordinated to improve access and treatment adherence.
Managing Drug Interactions: Anti-TB and ART regimens can interact. Careful drug selection and monitoring are necessary, especially when using medications like rifampicin with ART (e.g., dolutegravir-based regimens).
Addressing Co-existing Conditions: Managing other HIV-related conditions (e.g., malnutrition, hepatitis, and other infections) enhances resilience against TB and supports overall health.
Community Involvement and Awareness: Educating communities, involving local stakeholders, and tackling stigma are vital for successful prevention and treatment efforts.[55]

Treatment for the prevalence of TB in HIV

Managing TB in PLHIV is both complex and vital. While standard anti-TB regimens apply, specific considerations must be addressed due to overlapping drug toxicities, potential drug-drug interactions, and the risk of immune reconstitution inflammatory syndrome (IRIS).

1. Standard Treatment for Drug-Susceptible TB

PLHIV with drug-susceptible TB are typically treated with a combination of first-line anti-TB medications over 6 to 9 months, divided into two distinct phases:
Intensive Phase (first 2 months):
Isoniazid (H)
Rifampicin (R)
Pyrazinamide (Z)
Ethambutol (E)
Continuation Phase (next 4–7 months):
Isoniazid (H)
Rifampicin (R)
Key Considerations for PLHIV:
Daily Dosing: Daily administration under Directly Observed Therapy (DOT) is preferred, especially for those with advanced HIV or pediatric patients.

Duration Adjustments: While 6 months is the standard, a 9-month regimen may be recommended for extrapulmonary TB or delayed treatment response in PLHIV.[56]

2. Initiating Antiretroviral Therapy (ART)

The timing of ART initiation in PLHIV with active TB is critical:

CD4 < 50 cells/mm³: ART should be started within 2 weeks of TB treatment to reduce mortality.

CD4 ≥ 50 cells/mm³: ART is recommended within 2–8 weeks of starting TB therapy.

TB Meningitis: Antiretroviral Therapy may be delayed up to 8 weeks due to enhanced risk of CNS-IRIS. Clinical judgment and specialist input are essential.[57]

3. Managing Drug Interactions Between TB and HIV Medications

Rifampicin, a core anti-TB drug, significantly induces liver enzymes (e.g., CYP3A4), which can lower ART drug levels. Appropriate ART selection is crucial:

Preferred ART Regimens:

Efavirenz (EFV)-based: Less affected by rifampicin; standard dosing usually maintained.

Dolutegravir (DTG)-based: Requires dose adjustment (e.g., 50 mg twice daily) with rifampicin.

Raltegravir (RAL)-based: May be used at higher doses (800 mg twice daily).

Regimens to Avoid: Protease inhibitors (PIs) and NNRTIs like nevirapine due to high interaction risk.

Alternative: Rifabutin may be used in place of rifampicin when PI-based ART is necessary, though it requires expert dose management.[58]

4. Management of Drug-Resistant TB (DR-TB) in PLHIV

Treating DR-TB is more complex and requires second-line drugs that often carry greater toxicity and interaction potential.

Diagnosis: Early detection using molecular tests (e.g., GeneXpert MTB/RIF, Truenat) is essential.

Treatment Regimens: WHO recommends newer all-oral, shorter regimens like BPaLM (Bedaquiline, Pretomanid, Linezolid, Moxifloxacin), which reduce treatment duration to 6–9 months.

Individualized Treatment: Should be based on drug susceptibility testing (DST) and handled by experienced clinicians.

New and Repurposed Drugs: Use of bedaquiline, delamanid, and linezolid is encouraged to avoid injectable agents.[59]

5. Supportive and Adjunctive Therapies

Pyridoxine (Vitamin B6): Prevents isoniazid-induced neuropathy, especially important in PLHIV.

Cotrimoxazole Preventive Therapy (CPT): Protects against other opportunistic infections like PCP, regardless of CD4 count.[60]

Nutritional Support: Vital for recovery; includes assessment, counselling, and supplementation if needed.

Directly Observed Therapy (DOT): Enhances adherence and reduces treatment failure; video DOT (vDOT) is a modern alternative.

Managing TB-IRIS: May require corticosteroids and symptomatic treatment.

Integrated Care: Coordination between HIV and TB services is essential for comprehensive care and follow-up.[61]

DISCUSSION

A retrospective hospital-based study was carried out by Mitku et al. at five referral hospitals in Ethiopia's Amhara Region. To assess the prevalence and contributing factors of TB/HIV co-infection among 571 adult HIV-positive patients. They sought to ascertain the prevalence and risk factors for tuberculosis in HIV-positive people receiving antiretroviral therapy. They selected participants using stratified random sampling, and they used chi-square tests and logistic regression analysis to find related factors. A structured questionnaire was used to gather data from hospital records, and SPSS and STATA software were used for statistical analysis. They discovered that 158 out of 571 HIV patients, or 27.7%, also had TB. The study found that being ambulatory at the start of ART, drinking alcohol, having a lower baseline CD4 count (<200 cells/ul), and being single all significantly increased the risk of co-infection with TB. Conversely, the likelihood of co-infection was lower among non-smokers, patients in WHO clinical stages I or II, and homeowners ($p < 0.05$). The authors concluded that poor socioeconomic circumstances, substance use, and advanced clinical status (e.g., WHO stage III/IV, low CD4) were all significant predictors of tuberculosis among PLWHIV. To effectively manage at-risk patients, they recommended the establishment of TB/HIV co-infection units in hospitals and emphasized the necessity of focused screening and intervention programs.[62]

In order to assess the prevalence of TB-HIV co-infection, Tewelde Medhin et al. carried out a systematic review and meta-analysis study with 26,746 participants at the national level in Ethiopia. After retrieving 30 studies through electronic searches (PubMed, EMBASE, HINARI, Cochrane Library, and Google Scholar), they performed statistical analysis using MetaXL and R (version 3.2.3). With a pooled TB-HIV co-infection prevalence of 22% (95% CI: 19-24%) and significant heterogeneity ($I2 = 95.84\%$), they concluded that TB-HIV co-infection is still very common with considerable regional variation, necessitating integrated prevention, early detection, and management strategies[63]

In order to determine the prevalence and TB incidence rate per 1000 people annually, M Maji Goat carried out a retrospective coherent data analysis. To assess the prevalence and incidence rate of tuberculosis among HIV-detected patients, the centroid of HIV-K treatment, a multivariable Cox proportional hazards regression model was employed to estimate ratios in the Tanzania National Aids Control Program database between January 2011 and December 2014 among 1000 individuals annually on all HIV clients enrolled in HIV Kare patients. In this Status statistical software release by College Station, TX

State Corporation Limited, they have employed a descriptive analytical study of retrospective, coherent data and discovered data analysed from the records of 527249 individuals, totaling 1 crore 1539844. Clinical interaction: Meet someone who is HIV-enrolled. Assume that between 2011 and 2014, we traded services, averaging six encounters annually, and that they conducted research showing a general decline in TB incidence in PL HIV and a greater number among those on ART. Adult females who have advanced past the clinical stages of HIV reiterate the need for TB prevention therapy, HIV testing, and treatment for severe TB case findings. For people with HIV who do not have active TB.[64]

CONCLUSION

Among individuals with HIV, tuberculosis (TB) continues to be the most common and fatal opportunistic infection, especially in areas where both infections are endemic. Immunosuppression brought on by HIV significantly raises the risk of contracting TB and the disease's progression from latent to active. According to epidemiological data, low- and middle-income nations, particularly those in sub-Saharan Africa and Southeast Asia, have a disproportionately high burden of TB-HIV co-infection. This co-epidemic emphasizes the necessity of integrated healthcare approaches, such as timely initiation of antiretroviral and anti-TB treatments, preventive therapy, and routine TB screening in HIV-positive individuals. To lower incidence, stop transmission, and enhance outcomes in co-infected populations, surveillance systems must be strengthened and interventions must be customized based on epidemiological trends.

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