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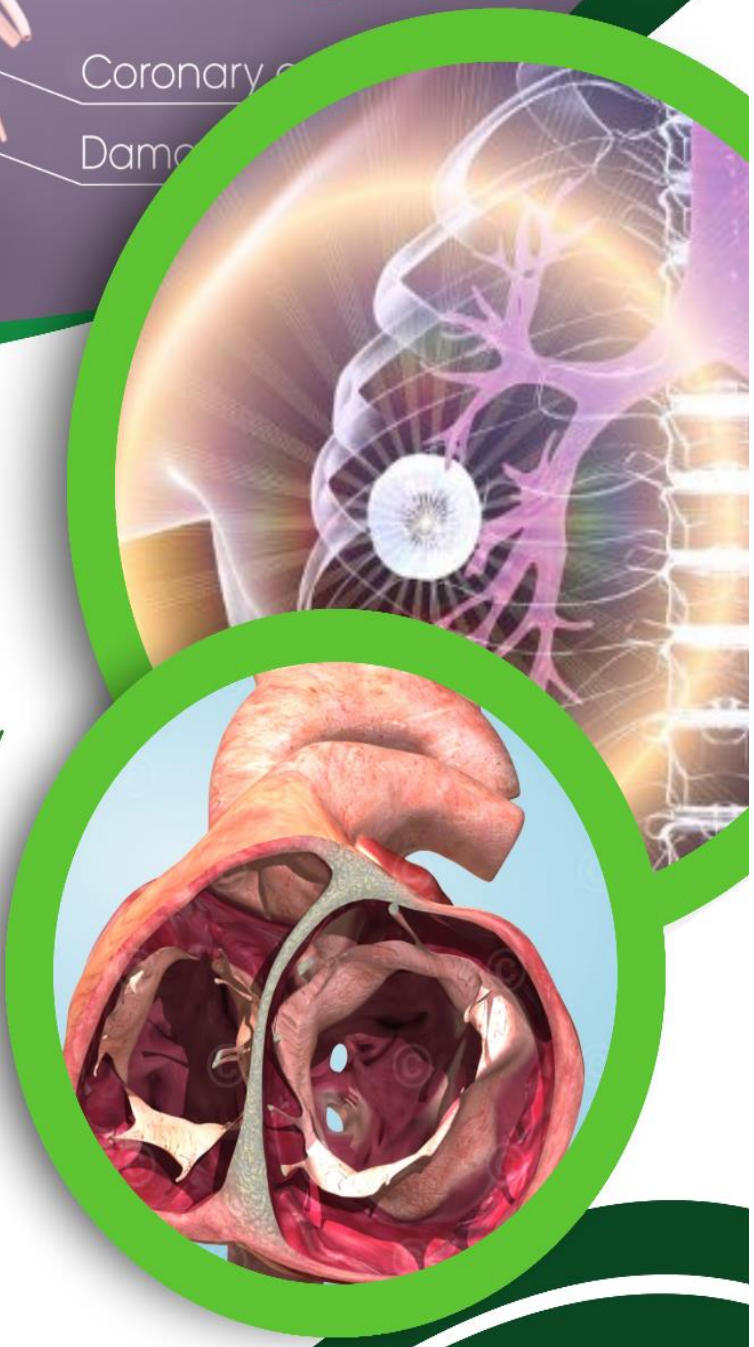

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"UNRAVELING THE TRIPLE THREAT: TUBERCULOSIS, MYOCARDIAL INFARCTION, AND HEPATITIS LINKED BY IMMUNITY AND MEDICATION"

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ABSTRACT

Tuberculosis (TB) is still a serious global health concern, aggravated by its complex connections with myocardial infarction (MI) and hepatitis. This review investigates the interconnectivity of various disorders via immunological, inflammatory, and pharmacological pathways. TB, caused by *Mycobacterium tuberculosis*, is a major infectious illness that can be latent (LTBI) or active (ATBI), with pulmonary and extrapulmonary symptoms. Chronic inflammation in LTBI is associated with an increased risk of cardiovascular disorders, including acute MI, due to immunological activation, endothelial dysfunction, plaque destabilization, and atherosclerosis. Furthermore, molecular mimicry between *M. Tuberculosis* heat shock proteins(mHSP65) and human heat shock protein (HSP) antigens may cause immunological responses, exacerbating cardiovascular disease. Hepatitis, particularly from viral infections such as Hepatitis B and C, frequently coexists with tuberculosis, which is often exacerbated by the hepatotoxic effects of antiTB medications. Particularly in genetically susceptible individuals, the typical TB treatment regimen (RIPE) clearly increases the risk of anti-tuberculosis drug-induced hepatotoxicity.

Given these intricate relationships, early detection, preventive efforts, and personalised therapy interventions are critical. Screening for LTBI in high-risk populations, optimising cardiovascular risk management, and monitoring liver function throughout tuberculosis treatment is vital to improving patient outcomes. The review emphasises the intricate interplay of tuberculosis, myocardial infarction, and hepatitis, highlighting the importance of integrated molecular diagnostic and immunomodulatory medicational approaches to mitigate their combined impact on disease progression and clinical consequences.

Keywords: *Mycobacterium tuberculosis*, LTBI, Antituberculosis drug induced hepatotoxicity, RIPE, Immune modulation, Autoimmune response

Tuberculosis (TB), a disease that has long been acknowledged as a major cause of morbidity and mortality worldwide, has unfortunately received insufficient attention in both developed and developing countries over the past several decades. *Mycobacterium tuberculosis* (Mtb) causes TB, which is still a leading cause of infectious deaths globally despite the availability of feasible and effective treatments[1]. In clinical terms, there are two main types of TB: latent TB (LTBI) and active TB infection (ATBI)[2]. There are several types of TB, such as pulmonary and extrapulmonary TB [3].Diagnosing tuberculosis involves an extensive medical history, clinical examination, and advanced diagnostic tools. The main treatment for TB is still the RIPE regimen[2].

Additionally, LTBI is correlated to a higher risk of cardiovascular disease, particularly acute myocardial infarction (AMI) [4].CVD and atherosclerosis are associated with chronic immunological activation. According to recent research, TB raises the risk of peripheral artery disease, heart attack, and stroke[5].AMI is a severe coronary artery disease that is classified by ECG findings and into six types according to its cause: atherothrombosis (type 1), supply-demand mismatch (type 2), sudden death (type 3), PCI-related (type 4a), stent thrombosis (type 4b), and CABG-related (type 5) [6].An immunological response comprising T cells, macrophages, and cytokines (TNF-α, IL-6, and IFN-γ) is triggered by an infection with *M. tuberculosis*. In order to improve TB outcomes, effective treatment must target the pathogen while controlling inflammation to prevent tissue damage and autoimmune consequences.

Patients with active TB frequently have Hepatitis C, which is associated with drug use and incarceration. Hepatitis, or inflammation of liver cells, is caused by viruses such as hepatitis A, B, C, D, and E. Hepatitis B and C are more common in TB patients[7],[8],and [9].The standard treatment for TB involves a combination of anti-tuberculosis drugs (ATDs), for a duration of 6 to 9 months[10]. During the treatment of TB, it is not uncommon for patients to experience adverse drug reactions (ADRs), one of which is anti-tuberculosis drug-induced hepatotoxicity (ATDH)[11]. ATDH risk is affected by immunological responses, oxidative stress, and genetic/non-genetic variables. Key mechanisms include antioxidant defenses, liver transport (Phase III), detoxification enzymes (Phase II), and cytochrome enzymes (Phase I) [12].This review presents an updated perspective on the connection between TB, MI, and hepatitis. It explores the immunological, inflammatory, and molecular mechanisms that underlie their association. Furthermore, we discuss the clinical implications, the challenges faced, and recent advancements in managing the combined effects of TB, MI, and hepatitis.

HISTORY OF TUBERCULOSIS

The oldest spinal TB fossils date to 8000 BC, and a Neolithic bone from 5000 BC also exhibits signs of infection. TB likely existed in animals before spreading to humans and affects all primates. Evidence of TB's ancient origins can be found in bone fragments that demonstrate Pott's disease [13],[14],and[15].

PATHOGENESIS

M. tuberculosis is an obligate-aerobic, non-spore-forming, non-motile bacillus characterized by a waxy coat that allows it to retain the red dye during acid-fast staining, often referred to as the “red snapper” effect (Fig.1). The pathogenicity of *M. tuberculosis* is linked to its ability to evade destruction by macrophages and to induce delayed-type hypersensitivity. This characteristic is attributed to several components found within the cell wall of *M. tuberculosis* that is cord factor, Lipoarabi nomannan (LAM), Complement and a highly immunogenic 65-kD *M. tuberculosis* heat shock protein [3].

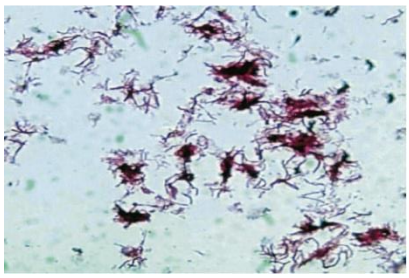


Fig1: Acid fast staining of *M. tuberculosis* [16]

TYPES OF TUBERCULOSIS

TB usually affects the lungs, though it can also involve other body parts. When it involves the lungs, it is termed pulmonary TB, whereas TB occurring outside the lungs is referred to as extrapulmonary TB. The manifestation of extrapulmonary TB depends on the site of infection, leading to several distinct types (Fig :2)[17].

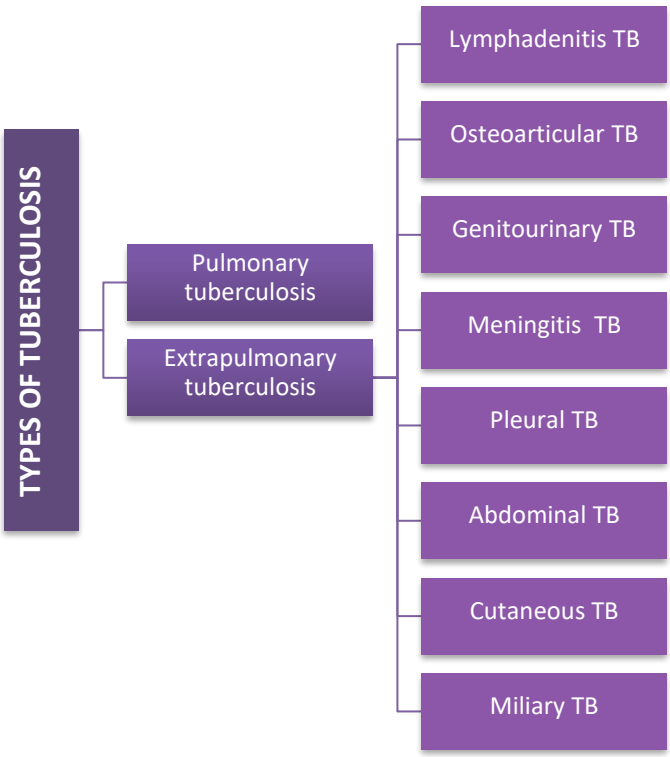


Fig 2: Overview of types of TB: It comprises pulmonary TB, which impacts the lungs, and extra pulmonary TB, which affects other parts of the body[17].

TRANSMISSION OF TUBERCULOSIS

TB is mostly transmitted through airborne routes, making it one of the most contagious infectious illnesses worldwide. *M. tuberculosis* spreads when a person infected with active pulmonary or laryngeal TB generate droplet nuclei that contain the bacterium and through coughs, sneezes, speaks, or any other forceful expiratory maneuver that shears respiratory secretions from the airways (Fig:3).As a result, people with close and extended contact with an infected person are more likely to contract the infection [18].

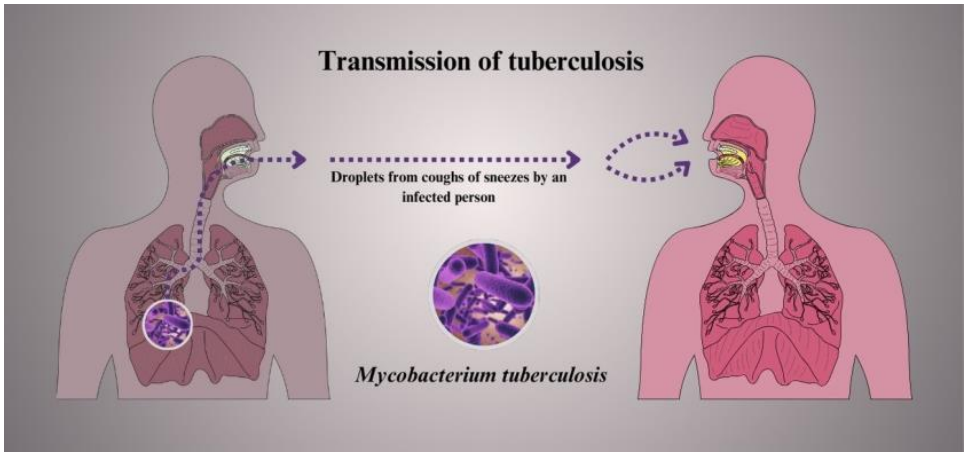


Fig 3: Diagrammatic illustration of TB transmission: *M. tuberculosis* enters other people's lungs through airborne droplets released by an infected person when they cough, sneeze, or communicate [19].

There are two types of TB: latent and active. A primary TB or the reactivation latent TB, which occurs in 90% of cases, can cause active TB, which damages several organs [20].

When *M. tuberculosis* is present but inactive, resulting in no symptoms, it is known as LTBI. Particularly in high-risk people, it can become active (highly infectious). The risk of reactivation is highest within two years but remains forever (Fig: 4)[21].

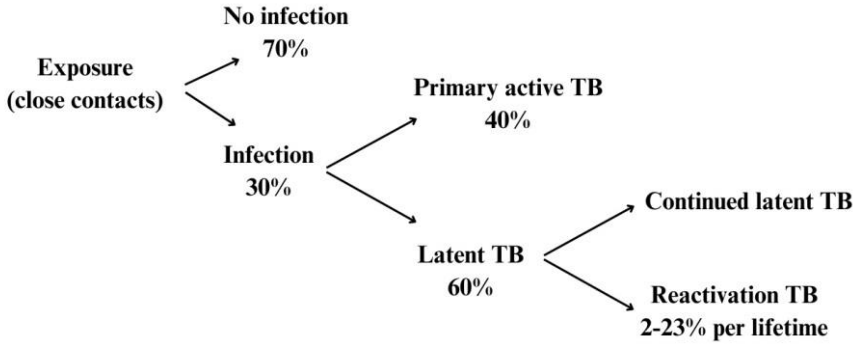


Fig 4: Following intimate contact, 30% individuals become infected, with approximately 40% acquiring primary active TB and 60% developing latent infection. 2-23% of immunocompetent individuals with latent TB will reactivate at a later date [22].

MECHANISM OF TUBERCULOSIS

M. tuberculosis is a classic intracellular pathogen that initially infiltrates the alveolar epithelial cells (pneumocytes I and II) following the host's intake of bacterial droplets by airborne transmission [23] (Fig: 5). They also have immunomodulatory roles through the production of cytokines and chemokines [24]. One of the first lines of defense against *M. tuberculosis* in the host is macrophages. Certain reactive proteins of *M. tuberculosis* can inhibit the well-equipped pattern recognition receptors (PRRs) of resident alveolar macrophages, which are responsible for recognizing and phagocytizing inhaled *M. tuberculosis* into phagosomes for clearance [25].Moreover, alveolar macrophages release a series of pro-inflammatory cytokines and chemokines in response to *M. tuberculosis* infection, including granulocyte macrophage colony-stimulating factor (GM-CSF), interleukin-1β (IL-1β), IL-6, IL-23, and tumor necrosis factor-α (TNF-α) [23].

Following the phagocytosis of *M. tuberculosis*, DCs migrate to the surrounding lymph nodes and present the antigens of *M. tuberculosis* to trigger CD4+ T lymphocytes [23].Unlike macrophages and DCs, neutrophils do not identify and phagocytose mycobacteria at the primary infection sites. Until recently, neutrophils were overlooked while being crucial responders in host defense against *M. tuberculosis*. Early on in the development of granulomas, neutrophil recruitment takes place.When *M. tuberculosis*-infected granuloma macrophages die, they emit signals that attract neutrophils to phagocytose both the macrophages and *M. tuberculosis* [26]. Between growth and decline, tuberculous granuloma formation is a dynamic process, and they are the primary battlegrounds for *M. tuberculosis* and host immune cells.

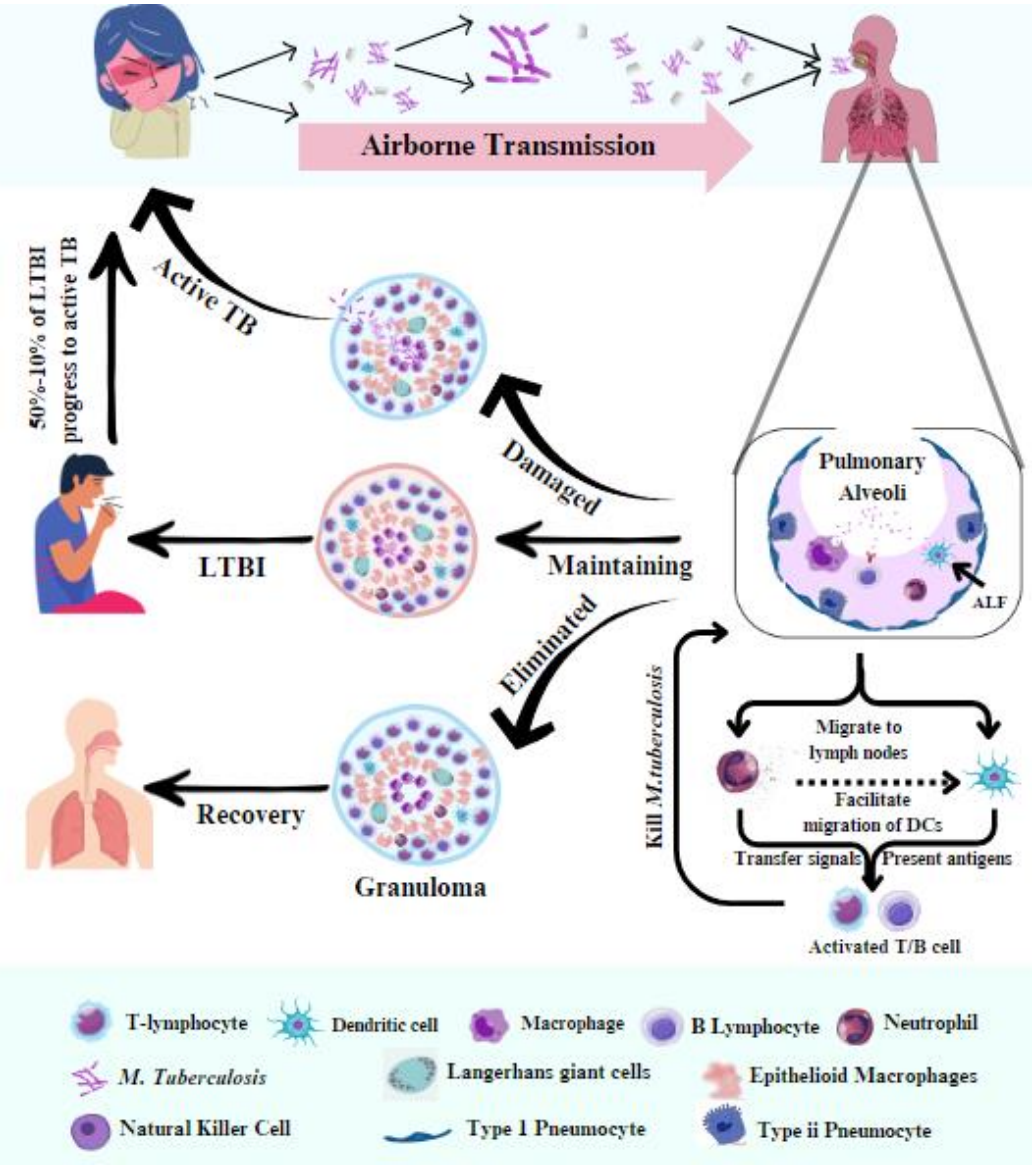


Fig 5: Schematic representation of the mechanism of LTBI. The immune system balances *M. tuberculosis* invasion by activating T cells producing granulomas, which confine and prevent active TB [27].

DIAGNOSTIC TOOLS

In order to prevent the spread of *M. tuberculosis*, prompt diagnosis and treatment are essential (Fig. 6). Accuracy is increased by innovations like interferon-γ release tests, fast liquid culture, nucleic acid amplification, and LED fluorescence microscopy, particularly when it comes to detecting latent TB [28],[29], and [30].

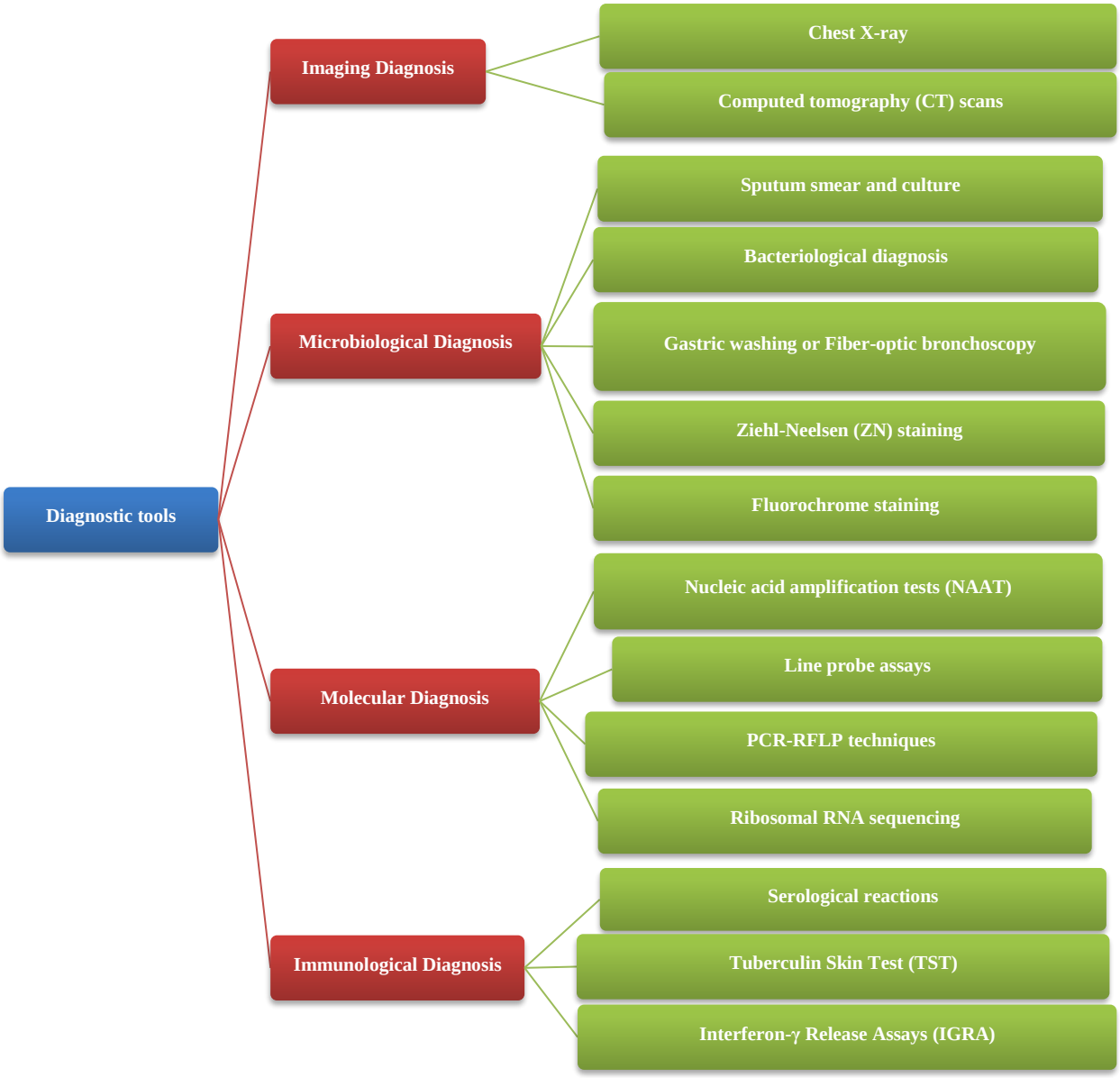


Fig 6: An Overview of TB Diagnostic Tools: A systematic categorization of diagnostic approaches highlights the comprehensive techniques employed to accurately detect and diagnoseTB.

Imaging Diagnosis

The primary technique for screening, diagnosing, and monitoring pulmonary TB is a chest X-ray. Detecting radiographic indicators of active TB is aided by CT scans. According to recent research, [(18)F]-2-fluoro-deoxy-D-glucose positron emission tomography scans are a non-invasive way to monitor the course of a disease and evaluation [31].

Molecular Diagnosis

The *M. tuberculosis* - specific nucleic acid amplification tests(NAAT) conducted on bronchopulmonary specimens is the most commonly used molecular tests for laboratory diagnosis. Line probe assays, which are a type of NAAT, are used to detect common genomic mutations responsible for antibiotic resistance. These assays work by analyzing a biological probe or culture through DNA hybridization[31].

Immunological Diagnosis

The Tuberculin Skin Test(TST) and Interferon-γ Release Assays(IGRA) evaluate the presence of persistent mycobacteria-specific T-cell responses in vivo(TST) and ex vivo(IGRA). IGRA is based on the discovery of the antigens ESAT-6 and CFP-10, which are relatively specific to *M. tuberculosis*, as well as the advancement of simplified technologies for measuring interferon-γ[31].

MEDICATION

A thorough understanding of anti-TB medications, as well as the pharmacokinetics and pharmacodynamics (PK/PD) involved, is essential. Additionally, it is important to consider variations in patient absorption and distribution, along with potential side effects and drug interactions, to develop effective anti-TB regimens[32] (Table :1).

Table 1: Details of medications used for the treatment of TB, including the drug name, manufacturing company, market name, recommended dosage, and their application in mild and severe TB cases[8],[33],[34],[35],[37], [38],and [39].

Drug Name	Company Name	Market Name	Dosage	Mild/Severe TB	References
Isoniazid	Multiple	Isonex, Laniazid	5 mg/kg/day (max 300 mg/day)	Mild: Nausea, rash, fatigue. Severe: Hepatotoxicity, peripheral neuropathy (can be prevented by pyridoxine).	WHO, CDC, Indian Pharmacopeia
Rifampicin	Multiple	Rifadin, Rimactane	10 mg/kg/day (max 600 mg/day)	Mild: Nausea, orange discoloration of urine. Severe: Hepatotoxicity, hypersensitivity reactions.	WHO, CDC
Pyrazinamide	Multiple	Tebrazid, Pyrafat	15–30 mg/kg/day	Mild: Joint pain, nausea. Severe: Hepatotoxicity, hyperuricemia leading to gout.	WHO, CDC
Ethambutol	Multiple	Myambutol	15–25 mg/kg/day	Mild/Severe	WHO, CDC

Rifabutin	Pfizer	Mycobutin	300 mg/day	Severe (MDR-TB)	WHO, Global Drug Facility
Rifapentine	Sanofi	Priftin	10 mg/kg once weekly	Mild	WHO, CDC
Fluoroquinolones	Multiple	Ciprofloxacin, Ofloxacin	Varies (depending on specific drug)	Severe (MDR-TB)	WHO, CDC
Moxifloxacin	Bayer	Avelox	400 mg/day	Severe (MDR/XDR-TB)	WHO, CDC, Bayer
Levofloxacin	Multiple	Levaquin, Tavanic	500–750 mg/day	Severe (MDR-TB)	WHO, CDC

Rifamycin

Rifamycin antibiotics (rifampicin, rifabutin, and rifapentine) are efficient against TB and chronic staph infections. Food reduces the absorption of rifampicin, and lowering the dosage from 600 mg to 450 mg reduces its effectiveness.Predicting their impact on dormant intracellular infections is difficult because of these considerations [40].

Rifampin

Rifampin (RIF) is a key TB drug that hinders mycobacterial RNA polymerase, stopping transcription. Rapid genotypic assays can identify resistance caused by mutations in the rpoB gene. Food has little effect on RIF's bioavailability,which is around 70%.Absorption includes the SLCO1B1 transporter and increases nonlinearly up to 40 mg/kg.

Isoniazid

The pro-drug isoniazid (INH), which inhibits the formation of mycolic acid and weakens the bacterial cell wall, is activated by the KatG enzyme in *M. tuberculosis*.Mutations in the katG or inhA genes cause resistance. Peripheral neuropathy, central nervous system toxicity, and drug-induced liver injury(DILI), which affects 1-2 percent of patients, are the primary components of INH toxicity.

Pyrazinamide

A pro-drug called pyrazinamide (PZA) is converted to pyrazinoic acid (POA) by amidases. Although 25 mg is the usual dosage, in certain circumstances, 50–70 mg may be used three times a week.Although alveolar macrophage absorption is restricted,PZA levels in cerebrospinal fluid are comparable to those in plasma, accumulate well in epithelial lining fluid, and penetrate lung lesions efficiently.

Ethambutol

Ethambutol (EMB) targets arabinose transferases to prevent the formation of mycobacterial cell walls. It has little effect on meals or antacids and an oral bioavailability of 80%. Depending on kidney function, about 70% is eliminated unaltered in urine.15–25 mg/kg per day or 50 mg/kg three times per week is the suggested dosage.

Other medications

Fluoroquinolones**,** Moxifloxacin, Levofloxacin, Bedaquiline, Pretomanid, Linezolid, Clofazimine Delamanid, Capreomycin, Para-Aminosalicylic Acid, Thioamides (Ethionamide, Prothionamide), Cycloserine, Terizidone, Rifabutin, and Rifapentine [2].

CORRELATIONS WITH TUBERCULOSIS

Inflammatory and autoimmune responses associated with LTBI

Chronic inflammation caused by TB may cause elevated levels of immune activation markers and proinflammatory cytokines in the blood circulation. C-reactive protein(CRP), interleukin-6(IL-6), tumor necrosis factor-alpha (TNF-α), and other cytokines contribute to systemic, which leads to atherosclerosis and cardiovascular disease[41] .TB-induced immunological activation and enhanced systemic inflammation promote endothelial dysfunction and atherogenesis. Chronic inflammation promotes atherosclerosis by recruiting immune cells (monocytes, macrophages) and inflammatory mediators (CD14, CXCL3, CCL2, CCL8) to arterial walls. These cells release cytokines that destabilize plaques, increasing the risk of rupture and triggering thrombus formation [42]. Proinflammatory cytokines impair endothelial function by lowering nitric oxide and increasing oxidative stress, resulting in a prothrombotic state.

Another potential contributory mechanism linking LTBI and AMI is molecular mimicry of mycobacterial heat shock protein 65 (mHSP65) and human heat shock protein(HSP) antigens[43].This condition can trigger the development of autoimmune responses, which play an important role in promoting atherogenesis and, eventually, cardiovascular disease. mHSP65, which is expressed by *M. tuberculosis*, is structurally identical to human HSPs found in blood vessel endothelial cells and smooth muscle cells. The immune system may misidentify human HSPs for foreign during LTBI or TB, leading to an autoimmune blood vessel attack. This results in inflammation, endothelial damage, and the development of atherosclerotic plaque.AMI and thrombosis may result from the rupture of these plaques[44].

Anti-tuberculosis drug-induced hepatotoxicity

The most effective treatment for TB comprises a combination of isoniazid, rifampicin, and pyrazinamide. It is important to note that these medications have the potential to induce hepatotoxicity, which may result in drug-associated hepatitis[45].

A meta-analysis examining multiple anti-TB drug regimens, primarily within adult populations, revealed that the incidence rate of liver toxicity associated with the co-administration of isoniazid and rifampicin was 2.6%. In contrast, the incidence rate was significantly lower at 1.1% when rifampicin was administered alone and 1.6% with isoniazid alone[46].Pyrazinamide is associated with potential hepatotoxicity, which is a significant factor to consider within the components of a short-course anti-TB drug regimen[47].

Pyrazinamide produced more significant adverse events(1.48 per 100 person-months) than isoniazid (0.49) and rifampicin(0.43), particularly in people over 60 and those who were born in Asia. Hepatotoxicity caused by pyrazinamide was more severe than previously thought. Health officials advise a three-weekly regimen or lower dosages to minimize dangers. Of these medications, rifampicin has the lowest risk of hepatotoxicity[48], [49]and [50].

Mechanism and immunogenetics of anti-tuberculosis drug-induced hepatotoxicity

As previously indicated, the hepatotoxicity interactions of isoniazid and rifampicin may arise from either additive or synergistic effects. A potential mechanism for this phenomenon involves the heightened liver toxicity resulting from monoacetyl hydrazine, hydrazine, and related compounds, which are generated through hepatic metabolism as a consequence of enzyme induction. This process operates via the hydrolase system, commonly referred to as the direct pathway (Fig:7)[51].The significance of this pathway is especially pronounced in individuals exhibiting the slow acetylator phenotype.

After extensive investigation, genotyping has confirmed that the slow acetylator phenotype is characterized by a combination of mutant alleles of the N-acetyl transferase 2 (NAT2) gene. This contrasts with the rapid acetylator phenotype, defined by homozygous NAT2*4 alleles, and the intermediate acetylator phenotype, which includes heterozygous NAT2*4 alleles alongside mutant alleles [52].

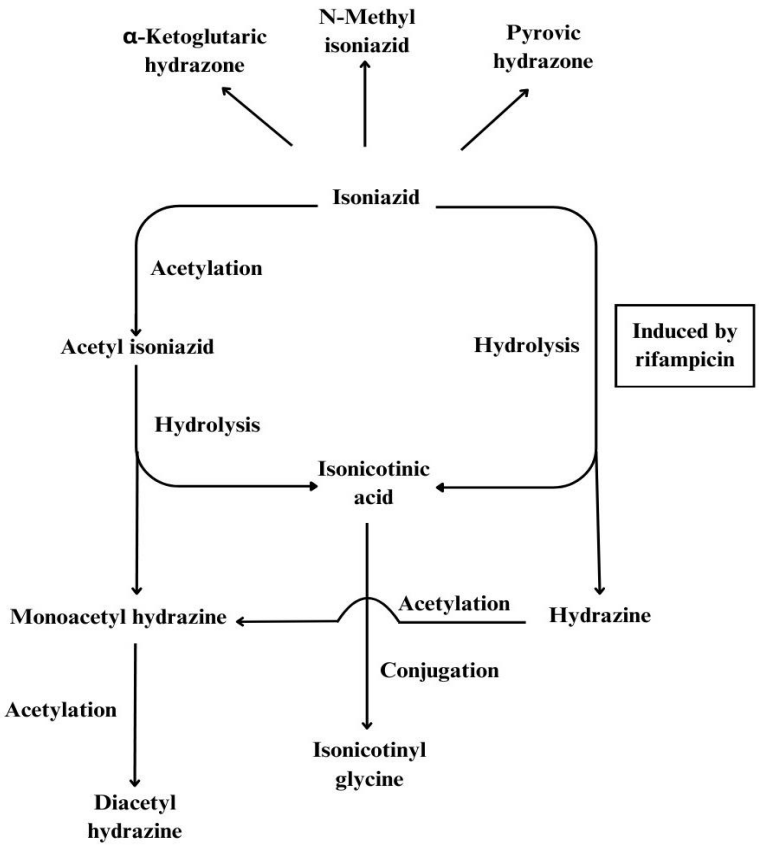
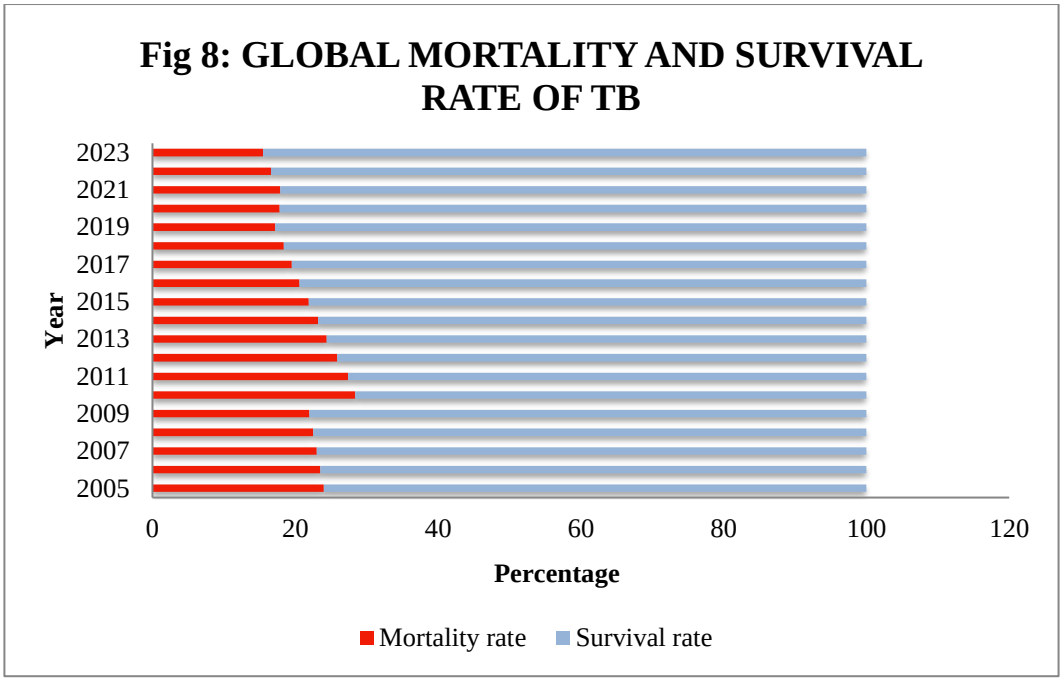


Fig 7: Rifampicin induction of the hydrolysis pathway of isoniazid metabolism into the hepatotoxic metabolite hydrazine [51].

Recent research has also identified additional genetic polymorphisms associated with anti-TB drug-induced hepatitis. These include variations in genes related to cytochrome P450 2E1 [53], glutathione S-transferase M1 [54], and specific MHC Class II-associated HLA-DQ alleles [55].

OVERVIEW OF TUBERCULOSIS MORTALITY AND SURVIVAL RATES

Drug resistance, early diagnosis, and treatment all have an impact on the mortality and survival rates of TB, which continues to be a serious worldwide health concern. Millions of people still die from TB each year, particularly in low-income nations, despite improvements in detection and treatment between 2005 and 2023 (Fig: 8). Although 86% of patients received treatment successfully by 2010, the number of drug-resistant TB infections was increasing, and the mortality rate was higher for older patients[56],[57], and [58].



The increase in TB cases worldwide has decreased after the COVID-19 pandemic, with 10.8 million cases in 2023—a delicate increase from 10.7 million in 2022. The number of TB-related deaths decreased by 23% since 2015, from 1.32 million in 2022 to 1.25 million in 2023. With interim goals of 75% and 50% reductions in 2025, respectively, the End TB Strategy seeks to reduce TB mortality by 90% and incidence by 80% by 2030 [59].

REMEDIAL STRATEGIES

Early Detection and Screening of LTBI

Treatment, prevention, and adherence techniques are all part of TB management. BCG vaccination and chemoprophylaxis help prevent TB, especially in high-risk groups, and novel therapies like high-dose vitamin C may improve results [60], [61].ECG, cardiac biomarkers (troponin), echocardiography,and angiography are all used in early MI detection. Liver function tests evaluate liver damage, while serological testing identifies viral infections[62], [63].

Integrative Strategies for Preventing MI in LTBI

For LTBI patients, a multimodal strategy is required to avoid inflammation and immunological reactions that can result in MI. Before TB becomes active, at-risk patients can be identified through early detection with TST, IGRAs, or chest X-rays. Prophylactic treatment with rifampin or isoniazid (6–9 months) lowers the risk of TB and its associated cardiac problems. Heart disease can also be avoided by controlling cardiovascular risk factors, such as blood sugar, cholesterol, and hypertension, as well as by leading a healthy lifestyle, getting regular exercise, and giving up smoking.

Controlling Chronic Inflammation and Immune Modulation

The management of chronic inflammation has greatly enhanced from the use of more recent TNF blockers (Biologics), such as etanercept, infliximab, and adalimumab. Biologics assist lower systemic inflammation by blocking these cytokines, which can otherwise lead to endothelial dysfunction, atherosclerosis, and an elevated risk of heart disease. However, a number of the deleterious inflammatory processes they suppress play a crucial role in maintaining TB in its latent phase; thus, cautious handling is required [64].Anti-inflammatory medications, such as non-steroidal anti-inflammatory medicines (NSAIDs) or corticosteroids, can help lower the

early inflammatory response after LTBI, hindering tissue damage that might spread to the cardiovascular system. However, additional specialized treatments can be needed for chronic or severe inflammation [65].

Preventing autoimmune reactions in LTBI that may result in MI requires an understanding of TB infection and inflammation. The goals of prevention are to control the risk of heart disease, reduce inflammation, and regulate immunity[66]. By ensuring that patients take their TB medication as prescribed, Directly Observed Therapy (DOT) improves adherence and prevents LTBI from developing into active TB. Adhering to the LTBI treatment plan lowers chronic inflammation and preserves immunological balance [67].

Mitigating hepatotoxicity in tuberculosis treatment

Drug-induced hepatotoxicity represents a notable health concern in the treatment of TB, especially in relation to the use of first-line anti-TB medications, including isoniazid, rifampicin, and pyrazinamide. While these agents are effective against *M. tuberculosis*, they are also associated with potential toxicity to liver cells. The underlying mechanism primarily involves the generation of reactive metabolites during the metabolism of these drugs, which can damage hepatocytes and incite inflammatory responses.

Hepatoprotective agents such as N-acetylcysteine, silymarin, and ursodeoxycholic acid support liver health and help prevent oxidative stress and inflammation; regular liver function tests(LFTs) allow for early detection of damage, allowing for timely treatment adjustments; customized dosing reduces liver strain, particularly in high-risk individuals; and in severe cases, alternative drugs with lower toxicity may be required.

Pharmacological interventions

Embelin: A natural compound with hepatoprotective influence against isoniazid- and rifampicin-induced liver damage in rat models. It helps restore liver enzyme levels and reduces oxidative stress markers, implying potential therapeutic applications [68].

Curcumin: Exhibits preventative and therapeutic properties in albino rabbits. Co-administration with anti-TB drugs reduces liver enzyme levels and improves liver morphology[69].

Management of Drug-Induced Hepatotoxicity in Tuberculosis Treatment

1. Dose Adjustment: In order to avoid hepatotoxicity, 80% of patients needed lower doses of rifampicin and isoniazid [70].

2. Drug Discontinuation: 20% of patients had to quit taking isoniazid because of severe liver damage, which emphasizes the necessity for personalized treatment.

3. Liver Function Monitoring: Frequent LFTs aid in the early detection and treatment of liver problems, particularly during the first two months [71].

4. Ongoing Research: Improving patient safety requires ongoing assessment of hepatoprotective measures and alternative therapy.

Challenges in Global Tuberculosis (TB) Management

1. Drug Resistance: The emergence of drug-resistant TB strains necessitates complex, expensive treatments, burdening healthcare systems.

2. Political Commitment: Adequate resources, infrastructure, and policies are vital for effective TB prevention and treatment.

3. Resource Allocation: Limited access to diagnostics, treatment, and healthcare staff in low-income regions hinders TB control.

4. Global Collaboration: International cooperation, investment in novel therapies, and efficient resource distribution are essential for sustainable TB eradication.

Implementing these strategies will help balance treatment efficacy and patient safety in TB management.

CONCLUSION

The interaction of hepatitis, myocardial infarction, and TB highlights the significance of early identification, suitable treatment plans, and careful patient monitoring, particularly for patients with comorbidities. These hazards can be reduced and patient outcomes can be enhanced by preventive strategies such as immunization, chemoprophylaxis for high-risk groups, and routine monitoring of liver and cardiovascular function throughout TB treatment. It is thought that an untreated or inadequately managed TB infection may serve as a prelude to cardiovascular and hepatic problems, including myocardial infarction (MI) and hepatitis, because of the systemic character of TB and its correlation with chronic inflammation. While the toxic effects of TB treatment medications, especially in susceptible persons, may result in liver damage and hepatitis, the prolonged inflammatory response associated with TB may lead to atherosclerosis and an increased risk of MI. The occurrence of these serious problems could be reduced by managing TB effectively through early detection, effective anti-TB treatment, and thorough surveillance for systemic effects. To further comprehend the mechanisms behind the connection between TB, MI, and hepatitis and to create specialized regimens for impacted patients, additional investigation is required.

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