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Review Article

Post-tuberculosis pulmonary hypertension and related vascular sequelae

Hipertensión pulmonar postuberculosis y secuelas vasculares relacionadas

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ABSTRACT

Introduction: Tuberculosis (TB) remains a leading global infectious killer, and survivors frequently develop long-term vascular complications. Post-tuberculosis pulmonary hypertension, fibrosing mediastinitis, and systemic cardiovascular and cerebrovascular sequelae are increasingly recognized but remain underdiagnosed.

Objective: To summarize current evidence on post-TB vascular disease, including pathophysiology, clinical manifestations, diagnostic strategies, and gaps in management.

Methods: A targeted narrative literature review was conducted of peer-reviewed articles published between 2000 and 2025 using PubMed, Scopus, Google Scholar, and World Health Organization databases. Included studies were full-text English-language observational studies, case series, systematic reviews, meta-analyses, and guideline reports addressing post-tuberculosis vascular or cardiopulmonary outcomes. Articles unrelated to vascular sequelae or derived from non-scientific sources were excluded. No formal evaluation of the methodological quality of the included studies was performed.

Development: Post-TB vascular disease arises from parenchymal lung damage, fibrosis, vascular remodeling, and chronic inflammation. Pulmonary hypertension is commonly reported among TB survivors, particularly in individuals with recurrent disease, extensive fibrosis, or treatment default.


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Fibrosing mediastinitis, although rare, may result in significant vascular obstruction and superior vena cava syndrome. TB survivors may also face increased long-term risks of ischemic heart disease and stroke. Diagnosis typically requires a multimodal approach. However, standardized screening protocols and long-term epidemiological data remain limited.

Conclusions: Post-TB vascular disease remains largely overlooked and contributes substantially to long-term morbidity. Focused screening strategies, risk assessment, and ongoing follow-up are needed. Coordinated multidisciplinary research is essential to clarify disease burden, improve diagnostic approaches, and develop evidence-based interventions for TB survivors.

Keywords: cardiovascular diseases; fibrosing mediastinitis; hypertension; pulmonary vascular diseases; tuberculosis.

RESUMEN

Introducción: La tuberculosis (TB) es una de las principales causas de mortalidad infecciosa a nivel mundial y los supervivientes, con frecuencia desarrollan complicaciones vasculares a largo plazo. La hipertensión pulmonar postuberculosis, la mediastinitis fibrosante y las secuelas cardiovasculares y cerebrovasculares sistémicas se reconocen cada vez más, pero permanecen infradiagnosticadas.

Objetivo: Resumir la evidencia actual de la enfermedad vascular postuberculosis, incluyendo fisiopatología, manifestaciones clínicas, estrategias diagnósticas y deficiencias en el manejo**.**

Métodos: Se realizó una revisión narrativa dirigida de artículos revisados por pares, publicados entre 2000 y 2025, utilizando PubMed, Scopus, Google académico y bases de datos de la Organización Mundial de la Salud. Se incluyeron estudios completos en inglés: observacionales, series de casos, revisiones sistemáticas, metaanálisis e informes de guías que abordaran resultados vasculares o cardiopulmonares postuberculosis. Se excluyeron artículos no relacionados con secuelas vasculares o de fuentes no científicas. No hubo evaluación formal de la calidad metodológica de los estudios incluidos**.**

Desarrollo: La enfermedad vascular postuberculosis se asocia con daño pulmonar parenquimatoso, fibrosis, remodelación vascular e inflamación crónica. La hipertensión pulmonar


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se reporta con frecuencia entre los supervivientes de TB, especialmente en casos de enfermedad recurrente o fibrosis extensa. El diagnóstico requiere un enfoque multimodal, aunque los protocolos de cribado estandarizados siguen siendo limitados.

Conclusiones: La enfermedad vascular postuberculosis sigue subestimada; contribuye de manera significativa a la morbilidad a largo plazo. Se requieren estrategias de cribado dirigidas, evaluación del riesgo, seguimiento continuo e investigación multidisciplinaria coordinada, para mejorar el diagnóstico y el manejo de los supervivientes de la TB.

Palabras clave: enfermedades cardiovasculares; enfermedades vasculares; hipertensión pulmonar; mediastinitis fibrosante; tuberculosis.

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INTRODUCTION

Tuberculosis (TB) continues to be a major global health challenge. In 2023, TB returned to being the world's leading infectious disease killer, surpassing COVID-19. It was also the leading cause of death among people living with HIV and a major contributor to deaths related to antimicrobial resistance. The global TB burden remains substantial with an estimated 10.8 million fell ill with TB in 2023, of whom 55% were men, 33% women, and 12% children or adolescents. People living with HIV accounted for 6.1% of all cases. The estimated number of deaths from TB was 1.25 million.[1]

While the acute infection is often treated successfully, TB survivors may experience long-term complications that extend beyond the lungs. Pulmonary and systemic vascular sequelae, including pulmonary hypertension, ischemic heart disease and cerebrovascular events are increasingly recognized as important contributors to morbidity and mortality in this population. However, these post-TB complications remain poorly characterized, and guidance on their screening, diagnosis,


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and management is limited. This mini-review aims to summarize current evidence on the vascular and cardiovascular complications following TB, highlighting key pathophysiological mechanisms, clinical spectrum, and diagnostic considerations, while identifying gaps for future research to improve the care of TB survivors.

METHODS

This narrative review was conducted using a targeted literature search of peer-reviewed articles published between 2000 and 2025. Searches were performed in PubMed, Scopus, Google Scholar, and the World Health Organization database. The following keywords and combinations were used: “post-tuberculosis,” “post-TB,” “pulmonary hypertension,” “pulmonary vascular disease,” “fibrosing mediastinitis,” “tuberculosis survivors,” “cardiovascular complications,” and “cerebrovascular complications.”

A narrative review approach was adopted to summarize the available evidence, given the heterogeneity of studies and the predominance of observational data, case series, and limited longitudinal research in this field. Only full-text articles in English were included. Eligible publications comprised observational studies, cohort studies, case series, systematic reviews, meta-analyses, and major guideline reports relevant to post-TB vascular or cardiopulmonary disease. Non-scientific sources and articles not addressing vascular sequelae were excluded. Reference lists of included studies were screened to identify additional relevant publications. No formal assessment of methodological quality or risk of bias was performed.

The final synthesis focused on the pathophysiology, clinical spectrum, and diagnostic approaches to post-tuberculosis pulmonary hypertension and other vascular complications.


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DEVELOPMENT

Pathophysiology and Clinical Spectrum

Tuberculosis (TB) can trigger persistent vascular injury that extends beyond the period of active infection. Even after microbiological cure, many patients exhibit structural and functional vascular changes collectively referred to as post-TB vascular disease.

Pulmonary Hypertension

Post-TB pulmonary hypertension arises from a combination of vascular, parenchymal, and hemodynamic changes. Central to its pathogenesis is pulmonary arteriopathy, including endothelial dysfunction, medial and intimal remodeling, and plexiform lesions, often driven by smooth muscle proliferation and resistance to apoptosis via TGF-β and BMP pathways.[2] Fibrotic and cavitary changes from post-TB lung disease (PTLD) further contribute to vascular remodeling.[3]

Clinically, repeated TB episodes have been associated with an increased risk of post-TB pulmonary hypertension, with data suggesting that each additional episode may approximately double the odds (OR = 2.13, 95% CI: 1.17–3.88; p = 0.013).[4] A small study reported that the presence of pulmonary hypertension in these patients correlates with adverse outcomes, including higher mortality and readmission rates.[5] In one case series of previously treated TB patients, 30 patients who were treated for pulmonary TB and presented with shortness of breath were screened for pulmonary hypertension. Of these patients, 14 were found to have pulmonary hypertension, with pulmonary artery systolic pressures ranging from 40 to over 80 mmHg—findings attributed to chronic hypoxia, parenchymal destruction and vascular remodeling.[6]

In a cross-sectional study of 46 patients with post-TB pulmonary sequelae, 45.7% developed pulmonary hypertension, most commonly among underweight individuals, treatment defaulters, and those with extensive or bilateral fibrotic lesions.[7] Additional contributing mechanisms include obliterative bronchiolitis leading to hypoxia-induced vasoconstriction and vascular obliteration.[8]

Fibrosing Mediastinitis (FM)

FM, a rare but severe complication involving fibrotic thickening of the mediastinum leading to extrinsic compression of the mediastinal broncho-vascular structures. The dense fibrosis encases


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and compresses the pulmonary arteries, pulmonary veins and superior vena cava.[9] Although *Histoplasma capsulatum* infection has been reported in literature as the most common cause worldwide, TB remains a well-recognized etiology.[10]

The vascular compression associated with FM potentially causes secondary pulmonary hypertension due to increased pulmonary vascular resistance and impaired venous drainage. Furthermore, superior vena cava syndrome may develop from obstruction of venous return, manifesting as facial swelling, venous distention and upper extremity edema.[11] Pulmonary hypertension secondary to FM is clinically classified into three types, type 1 referring to FM causing stenosis of the pulmonary arteries, type 2 referring to causing stenosis of the pulmonary vein, and type 3 resulting in stenosis of pulmonary artery, vein and bronchus.[12] A retrospective study analyzed contrast-enhanced computed tomography (CT) scans of chest of 100 patients with previous history of treated pulmonary TB and showed parenchymal fibrosis and volume loss in up to 90% of cases and fibrosing mediastinitis accounting for approximately 1%.[13]

Cardiovascular and Cerebrovascular Complications

It has been found that TB survivors have a higher risk of ischemic heart disease.[14] Tuberculosis may contribute to an increased risk of cardiovascular disease through sustained systemic inflammation, immune activation, hypercoagulability and lipid metabolism disturbances—all of which foster atherogenesis.[15] In addition, TB exerts direct pathogenic effects on the myocardium and pericardium, leading to structural and functional cardiac involvement.[16]

A population-based analysis of 69,331 participants from the Korea National Health and Nutrition Survey found that tuberculosis survivors had a higher 10-year atherosclerotic cardiovascular disease (ASCVD) risk compared to controls. Post-TB survivors were more likely to fall into intermediate and high-risk ASCVD categories, underscoring the elevated long-term cardiovascular risk in this population.[17]

Cerebrovascular complications are another important, yet often under recognized component of the post-tuberculosis vascular spectrum. Population-based studies have reported a higher incidence of ischemic stroke among tuberculosis survivors, with a nationwide Korean cohort study including 72,863 tuberculosis survivors, and age- and sex-matched controls found that post-TB patients


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showed a 22% increased risk of ischemic stroke.[18] These findings highlight that systemic vascular effects of TB, rather than rare CNS-specific TB manifestations, contribute to the elevated cerebrovascular risk.

Screening and Diagnosis

Early recognition of pulmonary hypertension is vital, as it contributes to reduced exercise tolerance, chronic respiratory disability and increased mortality among TB survivors. Because no single diagnostic test can reliably predict pulmonary hypertension in this population, a multimodal approach combining clinical assessment, imaging, functional testing and biomarkers is essential.

Transthoracic echocardiography (TTE) remains the most widely used screening tool for pulmonary hypertension. However, sensitivity and specificity of TTE declines in patients with underlying lung disease (81% and 61%, respectively) due to poor acoustic windows and Doppler limitations.[19]

The maximal tricuspid regurgitation velocity serves as the principal echocardiographic parameter for estimating pulmonary artery systolic pressure, with a threshold of 2.9 m/s considered indicative of elevated pulmonary pressures. TRV-based assessment, supplemented by indirect signs (right ventricular/atrial enlargement, septal flattening, RV outflow tract abnormalities) improves diagnostic accuracy of pulmonary hypertension.[20]

CT provides valuable structural insight. In a systematic review and meta-analysis of 20 studies including 2,134 subjects, they evaluated the diagnostic performance of CT-based pulmonary artery (PA) diameter and pulmonary artery-to-aorta (PA:A) ratio in detecting pulmonary hypertension. Pooled estimates showed that PA diameter had a sensitivity of 79% and specificity of 83%, while the PA:A ratio demonstrated a sensitivity of 74% and specificity of 81%. The study concluded that both CT-based measures are useful noninvasive tools for identifying pulmonary hypertension, though results should be interpreted alongside echocardiographic findings.[21]

Beyond imaging and echocardiographic evaluation, there are functional and biomarker-based assessments validated as reliable indicators of the severity and prognosis in pulmonary hypertension. Noninvasive clinical markers such as the 6-minute walk distance (6MWD), functional class (FC) and brain-natriuretic peptide (BNP)/NT-proBNP levels correlate with the severity and prognosis of PH. A systematic review which included 25 studies demonstrated that


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reduced 6MWD, worsening FC (III/IV) or elevated BNP/NT-proBNP predicted higher mortality, hospitalization and lung transplantation rates. These measures, individually or combined, provide cost-effective and reliable tools for risk stratification and long-term outcome prediction in pulmonary hypertension.[22]

Research Gaps and Future Directions

Despite declining global TB incidence, increasing attention is being directed toward the long-term health consequences experienced by tuberculosis survivors. Among these, post-TB pulmonary hypertension remains one of the least well-characterized complications. Current evidence regarding its prevalence, natural history, and long-term clinical impact remains limited, highlighting the need for large, collaborative studies to better define disease burden following completion of tuberculosis treatment.

Diagnostic limitations represent a major challenge, particularly in high TB-burden settings where access to right heart catheterization is limited. Future research should therefore prioritize the development and validation of pragmatic diagnostic pathways feasible in resource-limited environments.[23] Universal screening of all TB survivors is unlikely to be practical, emphasizing the potential value of risk-stratified screening strategies. However, reliable predictors for identifying high-risk populations have not yet been clearly established.

A recent scoping review of national tuberculosis guidelines from 34 high-burden countries found that only 7 (21%) included specific references to PTLD, with substantial variability in scope and detail. While Kenya and Malawi incorporated structured screening and management protocols, other countries, including Peru, South Africa, and Uganda, provided limited or nonspecific recommendations.[24] These findings underscore the absence of standardized global guidance addressing PTLD.

Uncertainty also persists regarding the timing of onset and prognostic implications of post-TB pulmonary hypertension. Available data on mortality and long-term outcomes remain limited, and it is unclear whether pulmonary hypertension independently contributes to reduced survival or reflects disease severity in advanced post-tuberculosis disease. Further studies integrating clinical,

imaging, and pathological data are required to clarify disease mechanisms and inform the development of targeted interventions aimed at mitigating progressive vascular damage.

Post-TB vascular disease represents an important spectrum of long-term complications affecting both the pulmonary and systemic circulation. Post-TB pulmonary hypertension emerges as a particularly clinically relevant manifestation, arising from a combination of residual parenchymal lung damage, vascular remodeling, and altered pulmonary hemodynamics. Available evidence indicates that diagnosis is often challenging, especially in resource-limited settings, and typically requires a multimodal approach integrating clinical assessment, imaging, and functional evaluation.

Despite increasing recognition, substantial gaps remain regarding disease prevalence, timing of onset, prognostic implications, and optimal screening strategies. Most current evidence is derived from observational studies, underscoring the need for larger, prospective investigations. Focused research efforts, development of practical risk-based screening approaches and structured long-term follow-up may contribute to earlier detection and improved outcomes among TB survivors.

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There is no data associated with this article.