AANHS Journal
Journal homepage: http://aanhsj.usu.ac.id

Spinal Tuberculosis: A Comprehensive Literature Review

Yohana Novelia*[1], Rully Hanafi Dahlan[2]
[1]Faculty of Medicine, Universitas Abdurrab, Pekanbaru, 28281, Indonesia
[2]Department of Neurosurgery, Faculty of Medicine, Universitas Padjadjaran, Sumedang, 45363, Indonesia
*Corresponding Author: yohananoveliaa@gmail.com

ARTICLE INFO

Article history:
Received 15 Apr 2026
Revised 24 Apr 2026
Accepted 29 Aug 2026
Available online 31 Oct 2026

E-ISSN: 2686-0848

How to cite:
Novelia Y, Dahlan RH. Spinal Tuberculosis: A Comprehensive Literature Review. Asian Australasian Neuro and Health Science Journal. 2026 Oct 08(02):93-101

This work is licensed under a Creative Commons Attribution-ShareAlike 4.0 International.

DOI: 10.32734/aanhs-j.v8i02.25254

ABSTRACT

Introduction: Spinal tuberculosis (STB) is the most destructive form of musculoskeletal TB, causing severe biomechanical instability and neurological morbidity, particularly in endemic regions due to delayed diagnosis.

Methods: This review synthesizes current evidence on the epidemiology, pathogenesis, diagnosis, and neurosurgical management of STB. Its insidious disease course often leads to advanced vertebral destruction, cold abscesses, and progressive kyphosis before clinical detection.

Results: MRI remains the radiological gold standard. For definitive diagnosis, integrating GeneXpert MTB/RIF and histopathological biopsy is crucial given the paucibacillary nature of the infection and the rising threat of multidrug-resistant strains (MDR-STB). Management follows the "Middle Path" philosophy, balancing long-term antitubercular therapy (9–18 months) with surgical intervention. Neurosurgery is indicated for progressive neurological deficits, three-column instability, or severe kyphosis (>40°). Currently, the posterior-only approach with pedicle screw fixation is preferred for its robust stabilization and lower perioperative morbidity.

Conclusion: Successful STB management requires a multidisciplinary approach, expanded access to rapid molecular diagnostics, and strict adherence to surgical indications to prevent irreversible neurological sequelae.

Keyword: Spinal Tuberculosis, Pott's Disease, GeneXpert, Spinal Fusion, Antitubercular Therapy.

1. Introduction

Tuberculosis (TB) remains a formidable global health challenge, causing significant mortality particularly in low- and middle-income countries [1]. While pulmonary TB is the most frequent manifestation, extrapulmonary dissemination occurs in a significant number of cases. Among these, spinal tuberculosis (STB) historically known as Pott's disease represents the most prevalent form of musculoskeletal tuberculosis, accounting for approximately 50% of skeletal TB cases [1-3]. Despite advances in global health, the epidemiological burden of STB remains substantial, with some regions experiencing a sustained upward trend in confirmed cases [4].

The clinical course of STB is notoriously insidious, often characterized by non-specific symptoms that delay timely diagnosis. This delay is clinically critical, as the disease progresses to cause severe vertebral destruction, spinal instability, cold abscess formation, and progressive kyphotic deformity. If left untreated, the mechanical collapse and subsequent spinal cord compression often culminate in debilitating neurological deficits, including paralysis or paraplegia. The susceptibility to and severity of STB are further exacerbated by various risk factors, including HIV co-infection, immunosuppression, malnutrition, and socio-economic challenges such as poverty [1-3].

Management of STB requires a multifaceted approach. While antitubercular chemotherapy remains the cornerstone of treatment, the emergence of multi-drug resistant spinal tuberculosis (MDR-STB) poses a significant threat to treatment efficacy [2,5]. Surgical intervention comprising debridement, bone grafting, and internal fixation is frequently indicated for patients presenting with severe kyphosis, spinal instability, or impaired neurological function. Given the complex pathogenesis and the evolving landscape of diagnostic and therapeutic strategies, a comprehensive synthesis of current literature is essential to improve clinical outcomes and guide effective management protocols [6,7].

2. Epidemiology

The global burden of spinal tuberculosis remains substantial, with Southeast Asia and Western Pacific regions accounting for approximately 62% of the world's total TB incidence. In 2023, the estimated global incidence reached 10.8 million cases, with Indonesia contributing nearly 10% of the global total (approximately 1,060,000 cases) [8,9]. While extrapulmonary tuberculosis (EPTB) represents 15–20% of all TB cases in Indonesia, spinal involvement is the most frequent site, found in 50–60% of patients with musculoskeletal TB [3,10]. The disease shows a bimodal age distribution; in endemic regions, up to 70% of cases occur in patients under 30 years old, whereas in developed nations, the median age of presentation has shifted to over 50 years [4,11].

Specific risk factors and co-morbidities significantly alter these epidemiological trends. HIV infection remains the most potent driver, as co-infected individuals have a 20-fold increased risk of developing active TB, with a significantly higher propensity for spinal dissemination [1]. Furthermore, the prevalence of multidrug-resistant TB (MDR-TB) among spinal cases is estimated at 3.5% in new cases and up to 18% in previously treated patients globally [5,8]. Recent 2024-2025 surveillance in Indonesia indicates a male-to-female ratio of approximately 1.5:1, with a significant concentration of cases in urban centers where population density and malnutrition act as primary catalysts for transmission [9,10].

3. Pathogenesis

The pathogenesis of spinal tuberculosis (STB) is a multifactorial process involving complex interactions between the systemic dissemination of *Mycobacterium tuberculosis*, the host's specialized immune response, and the unique anatomical features of the spinal column [12,13].

3.1 Routes of Hemodynamic Seeding and Dissemination

Hematogenous dissemination is the primary mechanism of infection in STB. The infection typically initiates in the lungs, where bacilli may escape alveolar macrophage containment upon a decline in host immunity and enter the systemic circulation. Approximately 67% to 80% of STB cases are linked to a primary pulmonary focus [13].

Dissemination to the vertebral bodies occurs through two distinct vascular pathways:

1. Arterial Spread: Infection often starts in the highly vascularized, cancellous region of the anterior vertebral body. Because segmental branches of the aorta supply adjacent vertebrae, the infection frequently involves two or more contiguous levels. Furthermore, paradiscal arteries branch out to reach the subchondral area of the upper and lower endplates of the disc. Consequently, subchondral bone involvement on either side of the disc known as paradiscal involvement is the most commonly reported variety [13,14].

2. Venous Spread: *Mycobacterium tuberculosis* can also disseminate via Batson's paravertebral venous plexus, a valveless network connecting the spinal veins to thoracic, abdominal, and pelvic vessels. Fluctuations in intra-abdominal or intrathoracic pressure (e.g., from coughing or straining) facilitate retrograde bacterial transport directly to the spine. This venous route is typically associated with centrally originating osseous lesions and non-contiguous "skipped" vertebral involvement [7,13].

Although less frequent, lymphatic spread to paravertebral nodes (which is more prevalent in children due to their well-developed lymphatic systems) and direct extension from adjacent infected tissues, such as ribs or paraspinal soft tissues, can also occur [12,13].

3.2 Host Immune Response and Evasion

Upon spinal invasion, bacilli are recognized by innate immune cells, such as macrophages, through pattern recognition receptors. Normally, macrophages engulf the bacilli to form phagosomes. However, the lipid-rich cell wall of *Mycobacterium tuberculosis* inhibits phagosome-lysosome fusion, allowing the bacteria to survive and replicate intracellularly [12,13].

Cell-mediated immunity is pivotal for controlling the infection. Helper T (Th1) cells secrete interferon-gamma (IFN-γ) to activate macrophages and promote intracellular killing. To evade this mechanism, the bacilli employ sophisticated strategies, such as secreting proteins (ESAT-6 and CFP-10) that interfere with antigen presentation, and inducing regulatory T cells (Tregs) that release inhibitory cytokines like interleukin-10 (IL-10) to suppress the Th1 response [12,13].

3.3 Molecular Mechanisms of Bone Destruction

Bone destruction in STB is heavily mediated by the host's inflammatory response. Macrophages and T lymphocytes activated by the infection secrete a large number of pro-inflammatory cytokines, including Tumor Necrosis Factor-alpha (TNF-α), Interleukin-1 (IL-1), and IL-6. These cytokines directly or indirectly stimulate the recruitment, differentiation, and activation of osteoclast precursors, thereby significantly enhancing osteoclastic bone resorption [12].

3.4 Pathological Progression and Structural Failure

As the disease progresses into the granulomatous phase, macrophages differentiate into epithelioid cells and fuse to form multinucleated Langhans giant cells, which are characteristic of tuberculous granulomas [12,13]. The central area of these granulomas eventually undergoes caseous necrosis, forming a soft, yellowish-white material. This caseating necrosis leads to the formation of "cold abscesses," which contain high concentrations of bacilli and lack vascular supply [13,14].

Structurally, vertebral bodies are highly susceptible to colonization due to their cancellous composition and slow blood flow. While STB initially shows relative preservation of the intervertebral disc because the bacteria lack proteolytic enzymes, the eventual destruction of the cartilaginous endplates disrupts nutrient diffusion, accelerating disc degeneration. The loss of mechanical strength in the infected cancellous bone leads to the collapse of the anterior column under normal axial loads. This structural failure causes an angulated structural kyphosis known as gibbus deformity. The combination of bone collapse, mechanical instability, and epidural abscess formation can compress the spinal cord, leading to neurological deficits clinically recognized as Pott's paraplegia [1,7,13].

4. Clinical Manifestations

The clinical presentation of spinal tuberculosis (STB) is typically insidious, with symptoms often progressing over several months before a definitive diagnosis is reached. The manifestations are characterized by a combination of constitutional, mechanical, and neurological symptoms, although their presence can be inconsistent [4,15].

Constitutional symptoms, reflecting the systemic nature of the infection, include low-grade fever (often with evening exacerbation), night sweats, malaise, anorexia, and significant weight loss [3,14]. However, recent large-scale studies indicate that these classic systemic features may only be present in a subset of patients; for instance, less than one-third of patients may present with a documented fever [4, 16].

Local and mechanical symptoms are the most frequent initial complaints. Chronic back pain is reported in nearly all cases (90-100%), typically localized to the site of vertebral involvement, which most commonly affects the lower thoracic and lumbar segments [4,14]. As the disease causes destruction of the anterior vertebral column, structural instability may manifest as a localized kyphotic deformity, or gibbus. Furthermore, the formation of cold abscesses is a hallmark of STB; these may present as paraspinal masses or track along the psoas muscle, appearing as a non-tender palpable mass in the inguinal region [3,15].

Neurological deficits represent the most debilitating clinical stage, occurring in approximately 20-45% of patients depending on the level of the lesion and the degree of spinal canal compromise [4,14]. Patients may present with a spectrum of Pott's paraplegia, ranging from early motor weakness and hyperreflexia to complete sensory loss and sphincter dysfunction. Atypical presentations have also been documented, such as spinal TB masquerading as pyogenic meningitis, emphasizing the need for high clinical suspicion in endemic regions [15,17].

5. Diagnostic Workup

The diagnostic trajectory for spinal tuberculosis (STB) requires a meticulous synthesis of clinical suspicion, high-resolution neuroimaging, and advanced molecular or histopathological confirmation. Given the paucibacillary nature of the disease and its insidious progression, reliance on a single diagnostic modality often leads to delayed intervention and irreversible neurological sequelae [1,18].

5.1 Conventional Radiography (X-ray)

Plain radiography remains the primary screening tool due to its accessibility. However, its sensitivity is notably low, as bony changes only become apparent after 30% to 50% of bone mineral loss has occurred [19,20]. The paradiscal type is the most common radiographic presentation, characterized by the narrowing of the intervertebral disc space and irregularity of the vertebral endplates. In advanced stages, classic findings include angulated kyphosis (gibbus deformity) and widened paravertebral shadows, which indicate abscess formation and potential mechanical collapse [20,21].

5.2 Computed Tomography (CT)

Computed Tomography provides superior spatial resolution for evaluating cortical bone destruction and structural instability, particularly in the posterior elements of the vertebrae, which are often better visualized on CT than MRI [21]. For the neurosurgeon, CT is indispensable for identifying bone sequestra and stippled calcifications within paraspinal soft tissue masses features that are highly pathognomonic for tuberculous infection and help differentiate it from pyogenic spondylitis [22]. Furthermore, CT-guided percutaneous needle biopsy remains a vital, minimally invasive tool for obtaining tissue from deep-seated spinal segments for microbiological and pathological analysis [18,22].

5.3 Magnetic Resonance Imaging (MRI)

MRI with gadolinium enhancement is the established gold standard due to its unparalleled soft-tissue contrast and ability to detect bone marrow edema before structural destruction occurs [19,20]. A hallmark of STB on MRI is the relative preservation of the intervertebral disc in the early-to-intermediate stages, attributed to the lack of proteolytic enzymes in *M. tuberculosis* [22]. Contrast studies typically reveal thin and smooth wall enhancement of "cold" abscesses, differentiating them from the thick, irregular enhancement seen in pyogenic infections [7,18]. Additionally, MRI is essential for identifying skipped lesions (non-contiguous vertebral involvement), which occur via Batson's paravertebral venous plexus, and for evaluating the degree of subligamentous spread beneath the anterior longitudinal ligament [20,22].

5.4 Molecular Rapid Testing and Microbiology

The integration of GeneXpert MTB/RIF has shifted the diagnostic paradigm, allowing for the detection of *M. tuberculosis* DNA and rifampicin resistance within 24 hours [18,23]. However, the overall yield of smear microscopy for acid-fast bacilli (AFB) is often low (frequently <36%) due to the paucibacillary state of spinal lesions. While liquid culture (MGIT) remains the benchmark for drug susceptibility testing, its clinical utility is limited by a long incubation period of up to 8 weeks and relatively low sensitivity in extrapulmonary samples [7,18].

5.5 Laboratory Indices and Predictive Models

While systemic inflammatory markers like Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP) are non-specific, they remain critical for monitoring therapeutic response. Modern diagnostic strategies now incorporate predictive nomograms and laboratory ratios. Research by Hu et al. emphasizes the utility of the T-SPOT.TB assay and specific antigen ratios in differentiating active STB from latent infection. These models utilize conventional variables such as hemoglobin, albumin, and blood urea nitrogen (BUN) to facilitate early clinical diagnosis in resource-limited settings where advanced molecular imaging is unavailable. [18,23].

5.6 Histopathology

In cases where microbiological yields are insufficient, histopathological examination remains the definitive diagnostic benchmark. The identification of caseating granulomas, Langhans giant cells, and epithelioid histiocytes provides conclusive evidence of STB. Histopathology often yields the earliest definitive result compared to traditional cultures, making it an essential component of the neurosurgical workup, especially in atypical presentations or cases masquerading as spinal malignancies [1,18].

6. Management

The management of spinal tuberculosis (STB) is a multidisciplinary protocol that integrates long-term antitubercular chemotherapy with strategically planned neurosurgical interventions. The core philosophy is the "Middle Path" regime a clinical strategy that balances conservative medical therapy with timely surgery to optimize neural decompression, deformity correction, and spinal stabilization [14,25].

6.1 Pharmacological Protocols and Duration Controversies

Antitubercular therapy (ATT) remains the absolute cornerstone for eradicating *Mycobacterium tuberculosis* in paucibacillary bone lesions. The standard protocol initiates with a 2-month intensive phase using a four-drug combination: Rifampicin (R), Isoniazid (H), Pyrazinamide (Z), and Ethambutol (E), followed by a continuation phase of Rifampicin and Isoniazid. While a 6-month course is standard for pulmonary TB, there is a strong consensus in neurosurgical literature that STB requires an extended duration due to limited drug penetration into necrotic bone and sequestra. Recent cohort studies advocate for treatment spanning 9 to 12 months, extending to 18 months in cases with multi-level destruction or slow clinico-radiological response [13,26].

Efficacy is monitored via serial Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP); a failure of these markers to normalize within 4–8 weeks necessitates an urgent evaluation for multidrug-resistant strains (MDR-TB) or mechanical failure [14, 27]. The management of MDR-STB requires a significantly more aggressive and prolonged pharmacological strategy. When clinical non-responsiveness is observed or when molecular assays (GeneXpert) or drug susceptibility testing (DST) confirm resistance to rifampicin or isoniazid, the standard first-line regimen must be replaced by individualized protocols incorporating second-line antitubercular agents, such as bedaquiline, fluoroquinolones, and linezolid. In accordance with the latest World Health Organization guidelines, the treatment duration for MDR-STB is extended to 18–24 months to ensure complete mycobacterial eradication and prevent postoperative relapse [8, 26]. Furthermore, in cases of extensive vertebral involvement under MDR-STB conditions, radical surgical debridement becomes even more vital to reduce the high bacterial load that may be sequestered within necrotic tissues, which are often poorly penetrated by second-line drugs [5, 27].

6.2 Surgical Decision-Making and Clinical Indications

Neurosurgical intervention is indicated to prevent irreversible neurological sequelae and progressive sagittal malalignment. Absolute indications include progressive neurological deficits (Frankel/ASIA Grade A–C) that do not improve with ATT, or acute deterioration due to mechanical compression from bone fragments. Mechanical instability from extensive three-column destruction also mandates stabilization [2, 28]. Furthermore, kyphosis correction is a primary surgical goal when angulation exceeds 40° in adults or when pediatric patients exhibit "Spine at Risk" signs such as retropulsion, lateral translation, or toppling signs which serve as strong predictors for late-onset paraplegia [14]. Relative indications include persistent mechanical pain, failure of medical management after 3–6 months, and large, symptomatic cold abscesses (psoas or paravertebral) that threaten vital structures [27,28]. The comprehensive step-by-step decision-making protocol is illustrated in the proposed management algorithm (Figure 1).

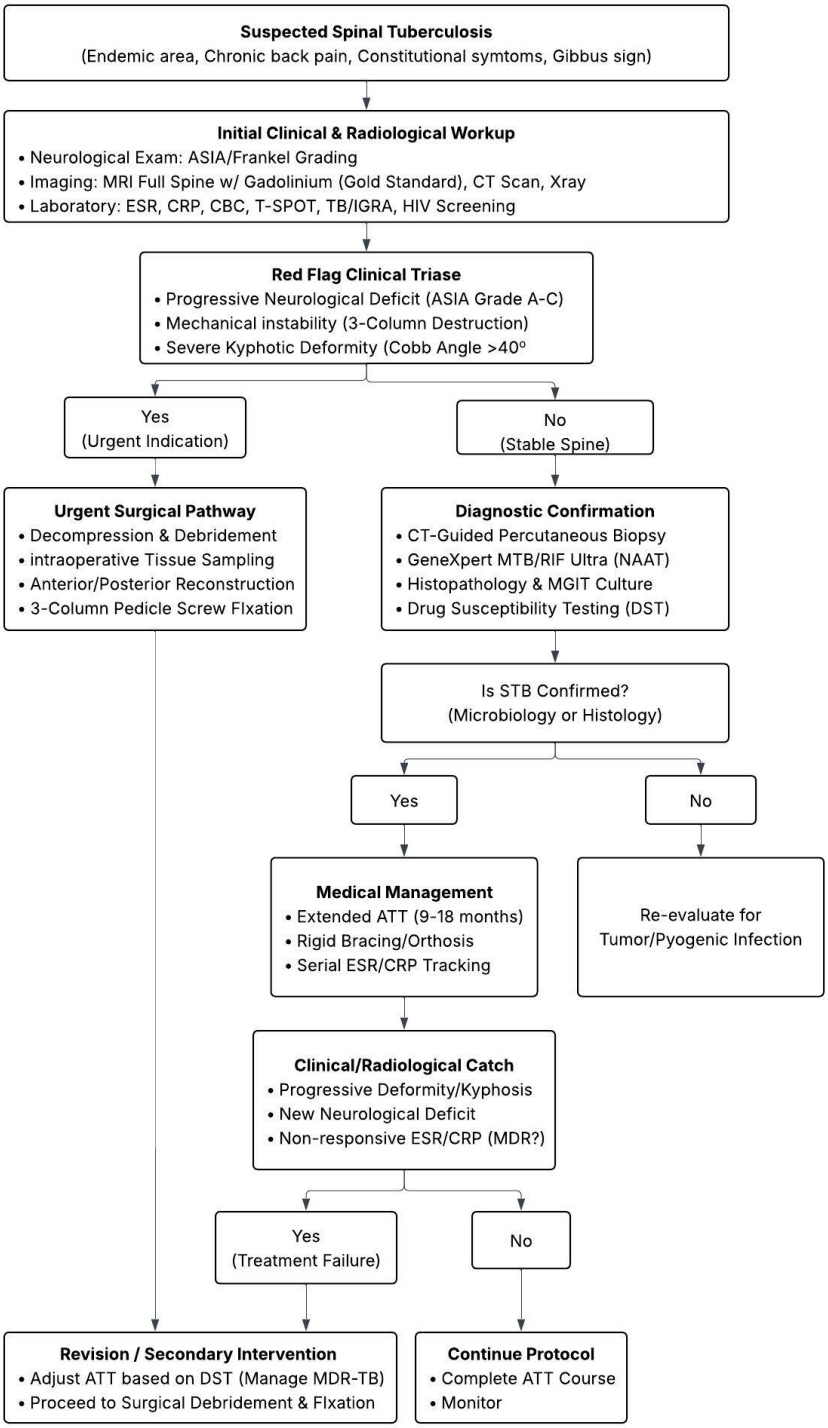


Figure 1. Guideline-Based Diagnostic and Management Algorithm for Spinal Tuberculosis

6.3 Operative Approaches and Radical Debridement

Operative strategies prioritize radical debridement, involving the aggressive removal of caseous material, necrotic bone, and infected disc tissue until healthy, bleeding bone is reached. This process reduces the mycobacterial burden and optimizes the local vascular bed for ATT penetration [2].

1. Anterior Approach: Historically favored for direct access to the vertebral body, the anterior approach (transthoracic or retroperitoneal) allows for radical debridement and anterior grafting. However, it is associated with significant perioperative morbidity, including pulmonary complications [2,28].

2. Posterior-Only Approach: This is the current neurosurgical preference. Utilizing pedicle screw fixation, this approach provides superior three-column stability and allows for circumferential or transpedicular decompression. It is highly effective in achieving and maintaining kyphosis correction with lower surgical risks compared to the anterior route [2,28,29].

6.4 Structural Reconstruction and Fusion Materials

Following debridement, structural reconstruction is mandatory to promote solid bony fusion.

1. Bone Grafting: Autologous bone (iliac crest or rib) remains the gold standard for its osteogenic properties, though allografts are increasingly used to minimize donor-site morbidity [2,27]

2. Titanium Mesh Cages: These have proven safe and effective for anterior column support. Despite historical concerns regarding foreign bodies in infected sites, titanium cages maintain vertebral height without increasing the risk of persistent infection when used alongside radical debridement and potent ATT [2,27].

6.5 Technological Advancements

The precision of STB surgery has been enhanced by 3D printing, which allows for patient-specific guide plates for accurate screw placement and custom implants for complex reconstructions. Minimally Invasive Surgery (MIS) including percutaneous endoscopic debridement and Video-Assisted Thoracoscopic Surgery (VATS) is increasingly utilized to reduce blood loss and accelerate recovery in select cases. Furthermore, the emergence of robotic-assisted navigation improves instrumentation safety in distorted spinal anatomy caused by severe infection [13,29].

7. Challenges and Prespectives in High-Burden Endemic Regions

The clinical and epidemiological landscape of spinal tuberculosis in high-burden endemic regions, particularly in Southeast Asia, contrasts starkly with that of developed nations. In these settings, managing STB extends beyond pathological complexities and is deeply intertwined with socio-economic and systemic healthcare barriers [4,8].

The most prominent challenge is the extraordinarily high rate of delayed presentation. Patients frequently arrive at tertiary referral centers in the advanced stages of the disease. Rather than presenting with early localized back pain, clinicians routinely encounter patients exhibiting severe, rigid kyphotic deformities, massive multiloculated cold abscesses, or profound neurological deficits, including established Pott's paraplegia [9,14]. This delay is driven by a combination of geographical barriers, low health literacy, and prolonged initial treatment for non-specific back pain by traditional healers or at primary care facilities lacking specific diagnostic tools.

Diagnostic bottlenecks further exacerbate this issue. While MRI with gadolinium and GeneXpert MTB/RIF are globally recognized as the gold standards, their availability in endemic countries is largely concentrated in major urban hospitals and specialized neurosurgical centers [18]. In peripheral and rural settings, clinicians are often forced to rely on conventional radiography and clinical acumen. Consequently, by the time a patient is referred to a spine surgeon, the window for conservative pharmacological management has often closed. The resulting three-column mechanical instability and neural compromise necessitate complex, high-risk reconstructive surgeries, placing an immense financial and logistical strain on local healthcare systems [2,28].

Furthermore, the management of STB in these regions is increasingly threatened by the rising incidence of multidrug-resistant tuberculosis (MDR-TB). The mandatory prolonged duration of antitubercular therapy (9 to 18 months) requires rigorous patient adherence. However, compliance is frequently compromised by the socio-economic impact of the disease, including loss of livelihood and the cost of repeated hospital visits [5,27]. This non-compliance not only fosters MDR-STB but also leads to high rates of postoperative recurrence and hardware failure.

To mitigate these regional challenges, a paradigm shift is required. Healthcare policies must prioritize the decentralization of rapid molecular diagnostic tools to secondary care levels and establish streamlined, early-referral pathways to specialized neurosurgical units. Integrating telemedicine and community-based screening could bridge the gap in early detection, ultimately shifting the regional treatment focus from aggressive surgical salvage back to early, effective medical management.

8. Conclusion

Spinal tuberculosis (STB) remains a formidable neurosurgical challenge that demands a high index of clinical suspicion due to its paucibacillary nature and the risk of irreversible neurological damage. A definitive diagnosis is best achieved through a multimodal approach, integrating the superior soft-tissue resolution of MRI as the gold standard with rapid molecular assays like GeneXpert MTB/RIF and early histopathological confirmation. Management is governed by the "Middle Path" philosophy, which balances a foundation of prolonged antitubercular therapy (9–18 months) with targeted surgical intervention for patients presenting with progressive neurological deficits, mechanical instability, or severe kyphosis. Contemporary neurosurgical trends increasingly favor the posterior-only approach with pedicle screw fixation for its robust stability and lower morbidity, while the integration of 3D printing and minimally invasive surgery (MIS) represents the next frontier in optimizing patient recovery and structural outcomes.

Acknowledgements

None.

Conflict of Interest

The authors declare no conflicts of interest in preparing this article.

References

[1] I. Glassman, K. H. Nguyen, J. Giess, C. Alcantara, M. Booth, and V. Venketaraman, "Pathogenesis, diagnostic challenges, and risk factors of Pott's disease," *Clin. Pract.*, vol. 13, no. 1, pp. 155–165, Jan. 2023, doi: 10.3390/clinpract13010014.

[2] J. Gan, C. Zhang, D. Tang, and X. Du, "Surgical treatment of spinal tuberculosis: An updated review," *Eur. J. Med. Res.*, vol. 29, no. 1, p. 588, Dec. 2024, doi: 10.1186/s40001-024-02198-4.

[3] E. H. Tobin and E. A. Rausch-Phung, "Tuberculous spondylitis (Pott disease)," in *StatPearls*. Treasure Island, FL, USA: StatPearls Publishing, Feb. 15, 2026. [Online]. Available: https://www.ncbi.nlm.nih.gov/books/NBK538331/

[4] M. Cao, S. Jiao, Q. Zhang, B. Zhu, and S. Shi, "Clinical and epidemiological analysis of 893 patients with spinal tuberculosis: An 11-year investigation of a general hospital in East China," *BMC Musculoskelet. Disord.*, vol. 26, no. 1, p. 830, Sep. 2025, doi: 10.1186/s12891-025-09053-5.

[5] S. Yang *et al.*, "Multi-drug resistant spinal tuberculosis epidemiological characteristics of in-patients: A multicentre retrospective study," *Epidemiol. Infect.*, vol. 148, p. e11, Jan. 2020, doi: 10.1017/S0950268820000011.

[6] M. Mohanty *et al.*, "Bibliometric analysis of research on spinal tuberculosis in last 5 years," *World J. Virol.*, vol. 14, no. 2, p. 102668, 2025, doi: 10.5501/wjv.v14.i2.102668.

[7] V. M. S. Ross, B. Pinzón, D. M. Palacios-Ortiz, Z. De La Rosa-Noriega, J. A. Rafeh, and L. F. Jurado, "Spinal tuberculosis, pathophysiology and radiological presentation, three case reports," *Biomedica*, vol. 45, no. 4, pp. 511–520, Sep. 2025, doi: 10.7705/biomedica.7685.

[8] World Health Organization, *Global tuberculosis report 2024*. Geneva, Switzerland: World Health Organization, 2024. [Online]. Available: https://www.who.int/teams/global-tuberculosis-programme/tb-reports/global-tuberculosis-report-2024

[9] Kementerian Kesehatan Republik Indonesia, "Indonesia's movement to end TB," 2025. [Online]. Available: https://kemkes.go.id/

[10] I. N. Kadariswantiningsih, R. N. Ramadhan, and D. D. C. H. Rampengan, "Prevalence of extrapulmonary tuberculosis in Indonesia: Protocol for systematic review and meta-analysis," *BMJ Open*, vol. 15, no. 5, p. e098140, May 2025, doi: 10.1136/bmjopen-2024-098140.

[11] R. Ueta, A. França Filho, H. Neto, M. Leonel, and F. Vieira, "Epidemiological profile of spinal tuberculosis and treatment performed in a SUS reference service from 2005 to 2024," *Coluna/Columna*, vol. 25, 2026, doi: 10.1590/s1808-185120262501295091.

[12] M. Liu, W. Nong, Z. He, H. Qin, W. Peng, and D. Yu, "Pathogenesis and therapeutic advances in spinal tuberculosis: A comprehensive review," *IJBLS*, vol. 12, no. 1, pp. 92–97, Sep. 2025, doi: 10.54097/tjrqkh87.

[13] Y. X. Lu *et al.*, "Review on the pathogenesis and treatment progress of spinal tuberculosis," *J. Biosci. Med.*, vol. 14, pp. 316–331, 2026, doi: 10.4236/jbm.2026.141024.

[14] R. Shanmuganathan, K. Ramachandran, A. P. Shetty, and R. M. Kanna, "Active tuberculosis of spine: Current updates," *N. Am. Spine Soc. J.*, vol. 16, p. 100267, Aug. 2023, doi: 10.1016/j.xnsj.2023.100267.

[15] D. Kumar, P. K. Patra, G. Mishra, and S. K. S, "Silently gradually progressing paraparesis Pott's spine lumbar vertebra with bilateral psoas abscess," *J. Orthop. Case Rep.*, vol. 16, no. 3, pp. 116–121, 2026, doi: 10.13107/jocr.2026.v16.i03.6910.

[16] R. P. Swari and I. N. G. Wahyudana, "Spinal tuberculosis: A rare case report in a 52-year-old female without a history of pulmonary tuberculosis," *Neurol. Spinale Med. Chir.*, vol. 6, no. 1, pp. 23–25, 2023, doi: 10.4103/nsmc.nsmc_25_22.

[17] P. Karunathilake, S. Bowatte, P. Kumarihamy, D. Madegedara, M. Gunathilake, and B. Narmada, "An unusual presentation of spinal tuberculosis masquerading as pyogenic meningitis: A case report," *Case Rep. Med.*, vol. 2026, p. 4815700, Feb. 2026, doi: 10.1155/carm/4815700.

[18] A. Acharya, K. Panda, S. Panigrahi, S. B. Senapati, A. K. Mahapatra, and K. Sahu, "Spinal tuberculosis: An exhaustive diagnosis," *Int. J. Mycobacteriol.*, vol. 13, no. 1, pp. 96–99, 2024, doi: 10.4103/ijmy.ijmy_14_24.

[19] A. Bhanudas, S. Ronanki, and V. L. Mounika, "Role of plain radiography and MRI in evaluation of spinal tuberculosis," *Int. J. Health Clin. Res.*, vol. 5, no. 2, pp. 389–392, 2022.

[20] R. Kukreja, M. Mital, and P. K. Gupta, "Evaluation of spinal tuberculosis by plain X-rays and magnetic resonance imaging in a tertiary care hospital in Northern India: A prospective study," *Int. J. Contemp. Med. Res.*, vol. 9, no. 5, pp. E1–E6, 2022.

[21] M. Kale, S. Tivaskar, A. Luharia, W. Khatib, and R. Umate, "Radiological evaluation of tuberculosis in the spine - A review article," *J. Pharm. Negat. Results*, vol. 13, no. S08, pp. 63–67, 2022, doi: 10.47750/pnr.2022.13.S08.10.

[22] J. J. Y. Yeo, S. B. S. Wong, and P. M. Yeap, "Revisiting imaging features of spinal tuberculosis," *Singapore Med. J.*, vol. 66, no. 10, pp. 576–581, Oct. 2025, doi: 10.4103/singaporemedj.SMJ-2022-214.

[23] A. T. Talebzadeh and N. Talebzadeh, "Diagnosis, management, and prognosis of spinal tuberculosis: A case report," *Cureus*, vol. 15, no. 2, p. e35262, Feb. 2023, doi: 10.7759/cureus.35262.

[24] X. Hu *et al.*, "A predictive model for early clinical diagnosis of spinal tuberculosis based on conventional laboratory indices: A multicenter real-world study," *Front. Cell. Infect. Microbiol.*, vol. 13, p. 1150632, Mar. 2023, doi: 10.3389/fcimb.2023.1150632.

[25] A. Rava *et al.*, "Conservative treatment of spinal tuberculosis in a retrospective cohort study over 20-year period: High eradication rate and successful health status can be expected," *Ann. Jt.*, vol. 8, p. 34, Aug. 2023, doi: 10.21037/aoj-22-54.

[26] S. K. Abid, R. Kumar, P. K. Yadav, and R. Kumar, "Determining the optimal duration of drug therapy in spinal tuberculosis: A cohort study," *Int. J. Med. Biomed. Stud.*, vol. 8, no. 3, pp. 192–199, 2024, doi: 10.32553/ijmbs.v8i3.2841.

[27] P. Wang, W. Liao, G. Cao, Y. Jiang, J. Rao, and Y. Yang, "Characteristics and management of spinal tuberculosis in tuberculosis endemic area of Guizhou province: A retrospective study of 597 patients in a teaching hospital," *Biomed Res. Int.*, vol. 2020, p. 1468457, Jan. 2020, doi: 10.1155/2020/1468457.

[28] F. Kilinc *et al.*, "Surgical management and clinical outcome of cervical, thoracic and thoracolumbar spinal tuberculosis in a middle-European adult population," *Sci. Rep.*, vol. 13, no. 1, p. 7000, Apr. 2023, doi: 10.1038/s41598-023-34178-9.

[29] Romaniyanto, M. F. Ilyas, A. Lado, D. Sadewa, D. N. Dzikri, and E. A. Budiono, "Current update on surgical management for spinal tuberculosis: A scientific mapping of worldwide publications," *Front. Surg.*, vol. 11, p. 15