Journal of Advances in Drug Research

ISSN: 2230-7761
www.jadr.co.in

Volume 17, Issue 04 | Received 20.09.2026 | Accepted 21.09.2026 | Online 25.09.2026

CARDIAC ARRHYTHMIAS IN SPINAL TUBERCULOSIS

A narrative review of potential mechanisms clinical assessment and evidence gaps

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Abstract

Background: Spinal tuberculosis can coexist with neurological impairment, systemic illness and cardiovascular comorbidity. Arrhythmias detected during its course may reflect several overlapping processes, including pre-existing heart disease, autonomic disturbance, metabolic abnormalities, medication effects and, occasionally, myocardial tuberculosis. The strength of evidence differs substantially across these explanations.

Objective: To critically examine evidence relevant to cardiac arrhythmias in spinal tuberculosis and identify a clinically useful framework for assessment and future research.

Methods: A focused narrative review used targeted web searches to identify indexed biomedical publications and professional guidance available by 21 September 2026. Sources were selected for relevance to spinal tuberculosis, tuberculosis treatment and repolarization, myocardial involvement, spinal cord dysfunction and electrocardiographic assessment. Direct spinal evidence was distinguished from evidence extrapolated from other populations. No meta-analysis or formal risk-of-bias assessment was undertaken.

Synthesis: The selected literature supports the clinical relevance of treatment-associated QT prolongation and demonstrates altered cardiac autonomic regulation after traumatic spinal cord injury. However, neither finding establishes the incidence or mechanism of arrhythmias in spinal tuberculosis. QT prolongation is a marker of repolarization disturbance rather than an arrhythmia diagnosis. Published myocardial tuberculosis cases support a differential diagnosis but cannot quantify population risk. Baseline rhythm documentation, treatment timelines, electrolyte assessment and clinically indicated ambulatory monitoring are therefore central to interpretation.

Conclusion: Cardiac rhythm abnormalities in spinal tuberculosis require individualized attribution. Prospective studies should distinguish documented pre-existing arrhythmia from arrhythmia first detected after tuberculosis diagnosis, use comparable monitoring across groups and assess neurological, cardiac and treatment-related exposures separately.

Keywords: spinal tuberculosis; tuberculous spondylitis; cardiac arrhythmia; QT interval; autonomic dysfunction; electrocardiography; Holter monitoring.

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www.jadr.co.in

Volume 17, Issue 04 | Received 20.09.2026 | Accepted 21.09.2026 | Online 25.09.2026

Introduction

Spinal tuberculosis presents a clinical setting in which vertebral disease, neurological dysfunction and prolonged treatment may coexist. A retrospective study of 893 patients described substantial neurological involvement and concurrent pulmonary disease, illustrating the heterogeneity of this population [1]. Such heterogeneity matters when interpreting an abnormal electrocardiogram: a rhythm finding in a patient with spinal tuberculosis does not, by itself, identify the spinal infection as its cause.

Tuberculosis treatment guidance provides the broader therapeutic context, but decisions about drug selection and duration depend on disease site, susceptibility and individual circumstances [2]. Cardiac safety research on bedaquiline and delamanid has mainly addressed drug-resistant tuberculosis rather than a dedicated spinal population [3,4]. Separately, investigations of traumatic spinal cord injury demonstrate changes in autonomic regulation and cardiac rhythm [5,6]. These bodies of evidence are relevant, but their applicability to slowly progressive tuberculous compression must be examined rather than assumed.

The purpose of this review is to connect these evidence streams while preserving their limitations. It examines potential mechanisms, the distinction between arrhythmia and electrophysiological risk markers, and the requirements for meaningful clinical observation. Its contribution is an evidence-based framework for attribution and study design; it does not propose a validated spinal tuberculosis arrhythmia score.

Review approach

Targeted web searches were conducted on 21 September 2026 using combinations and exact phrases including “spinal tuberculosis”, “arrhythmia”, “tuberculous myocarditis”, “bedaquiline delamanid”, “QT effects”, “cardiac arrhythmias associated with spinal cord injury” and “heart rate variability”. Searches located PubMed and PubMed Central records, publisher pages, and World Health Organization and professional society materials. Bibliographic details were checked against the available publication records. Publisher text, abstracts and indexed extracts were examined where accessible; full text was not accessible for every source.

Sources were retained when they informed spinal disease characteristics, a plausible cardiac mechanism, medication safety or a relevant measurement method. The synthesis prioritizes identifiable original studies and authoritative guidance. Foundational electrophysiology standards were retained because measurement comparability is central to the research question. Selection was purposive and non-exhaustive, without a registered protocol, duplicate screening, a complete database export or formal certainty grading. Consequently, this article is a narrative review and cannot establish the absence of relevant studies outside the retrieved literature.

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What the evidence establishes

Direct spinal tuberculosis research and cardiac safety research answer different questions. Cao and colleagues described clinical, diagnostic and treatment characteristics of 893 spinal tuberculosis cases, but the reported study was not designed to estimate arrhythmia incidence through standardized rhythm surveillance [1]. Its size therefore cannot compensate for the absence of the specific cardiac outcome required here. Similarly, a treatment trial measuring serial QT intervals estimates changes in repolarization under its eligibility and monitoring conditions, rather than the total burden of atrial and ventricular arrhythmias in spinal disease [3].

The studies summarized in Table 1 should therefore be interpreted according to population and outcome. Evidence from traumatic spinal cord injury contributes biological plausibility; pharmacological studies inform exposure-related risk; myocardial case reports establish clinical possibilities. None of these categories alone demonstrates a characteristic arrhythmia phenotype attributable to spinal tuberculosis.

Table 1 Selected evidence and its applicability to spinal tuberculosis

Source	Population and design	Contribution	Limit of inference
Cao et al 2025 [1]	893 spinal tuberculosis cases; retrospective study	Defines clinical and neurological heterogeneity	No standardized arrhythmia incidence endpoint
Dooley et al 2021 [3]	Randomized tuberculosis drug safety trial	Examines QT effects of bedaquiline and delamanid	Selected patients; not a spinal tuberculosis cohort
Tanneau et al 2022 [4]	Pharmacometric analysis	Relates drug and metabolite exposure to QTcF	Model predictions do not establish clinical arrhythmia rates
Malmqvist et al 2015 [5]	50 patients with acute traumatic cord injury	Documents differences in heart rate variability	Traumatic injury differs from tuberculous compression
Bartholdy et al 2014 [6]	Acute traumatic cord injury; serial rhythm assessment	Supports attention to rhythm after cord injury	Cannot supply spinal tuberculosis prevalence estimates
Vennamaneni et al 2022 and Shaikh et al 2023 [7,8]	Myocardial tuberculosis case reports	Support consideration of myocardial involvement	Cases cannot quantify risk or prove a spinal mechanism

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Potential mechanisms and competing explanations

Autonomic disturbance associated with neurological involvement

In a study of 50 patients with acute traumatic spinal cord injury, Malmqvist and colleagues used repeated 24-hour Holter recordings and found differences in heart rate variability according to lesion level and completeness [5]. Bartholdy and colleagues also investigated rhythm abnormalities during the first month after traumatic injury [6]. These observations support evaluating cardiac autonomic function when a spinal lesion causes clinically important cord dysfunction. They do not demonstrate that vertebral tuberculosis without neurological involvement produces the same effects.

For spinal tuberculosis, the proposed pathway is interruption or distortion of autonomic control in patients with cord involvement. This remains an extrapolation. A vertebral level on magnetic resonance imaging is not interchangeable with a neurological level, and an epidural collection is not equivalent to functional interruption of sympathetic pathways. Future analyses should record the neurological examination and cord compression separately from the location and extent of bone destruction. This distinction would make an observed association more interpretable and reduce the temptation to assign a cardiac mechanism solely from imaging.

Medication exposure and ventricular repolarization

The randomized ACTG A5343 study evaluated bedaquiline, delamanid and their combination during multidrug treatment. Participants with baseline QTc above 450 ms were excluded; clofazimine was disallowed and levofloxacin replaced moxifloxacin. The reported combination effect on QTc was modest and no more than additive under these conditions [3]. These eligibility and background-regimen restrictions are essential to interpretation. The findings should not be generalized without qualification to patients with baseline repolarization abnormalities or multiple additional QT-prolonging medicines.

A subsequent model-based analysis examined the relationship between drug and metabolite concentrations and QTcF, supporting exposure-dependent assessment [4]. Pharmacometric evidence helps explain why safety depends on the actual regimen and exposure history. It does not prove that an individual episode of palpitations is drug-induced, and a modeled change in QTc is not a clinical ventricular tachyarrhythmia. Studies of spinal tuberculosis should therefore record medication start and stop dates, dose changes and concomitant drugs alongside ECG timing.

Drug-susceptible and drug-resistant treatment groups should not be treated as a single cardiac exposure category. Recommendations for tuberculosis care are regimen- and population-dependent [2]. The American Heart Association statement also emphasizes that medication-related rhythm effects extend beyond QT prolongation to several arrhythmia types [9]. Attribution should consider all relevant medicines, including cardiac and supportive therapies, instead of assuming that any event during antituberculosis treatment originates from the principal tuberculosis drug.

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Electrolyte abnormalities and systemic illness

Reduced repolarization reserve can become clinically important when several risk factors coexist. The scientific statement on prevention of torsade de pointes identifies electrolyte abnormalities, bradycardia and combinations of QT-prolonging drugs as relevant factors [10]. In a spinal tuberculosis study, potassium, magnesium and calcium measurements would therefore be interpretable safety variables when linked to the timing of ECG changes. An isolated laboratory result obtained well before or after an event is less informative for causal attribution.

Systemic illness also complicates rhythm interpretation. Fever, pain, anemia, hypoxemia and volume disturbance should be considered when evaluating tachycardia, rather than classifying every elevated heart rate as a primary arrhythmia. For research, these conditions are potential covariates or competing explanations. The retrieved evidence does not establish a spinal tuberculosis-specific causal role for iron or zinc concentrations in arrhythmia development. If measured, such biomarkers should have a prespecified exploratory purpose and should not be presented as validated predictors.

Myocardial tuberculosis and pre-existing cardiac disease

Published reports by Vennamaneni and colleagues and Shaikh and colleagues describe the diagnostic challenges of myocardial tuberculosis [7,8]. They support including myocardial involvement in the differential diagnosis when cardiac findings warrant investigation. However, a case report cannot establish how frequently this occurs in spinal tuberculosis, and tuberculosis at a vertebral site does not confirm infection of the myocardium. The suspected cardiac diagnosis requires its own clinical and investigative justification.

An additional competing explanation is pre-existing structural or electrical heart disease. A first recorded episode after tuberculosis diagnosis may represent previously unrecognized disease. This is particularly relevant when admission triggers more intensive cardiac testing than the patient previously received. The available spinal cohort also includes patients with substantial comorbidity [1]. Research that fails to document prior ECG findings, cardiac diagnoses and treatment may incorrectly attribute background disease to tuberculosis.

Clinical and electrophysiological assessment

Defining the outcome before interpreting it

Cardiac outcomes should be specified at the level of the rhythm diagnosis. Atrial fibrillation, supraventricular tachycardia, ventricular ectopy, ventricular tachycardia, sinus pauses and atrioventricular block should not be collapsed into an undefined label such as “ECG changes”. QTc prolongation belongs in a separate repolarization outcome category. Symptom burden, episode duration, hemodynamic consequences and treatment requirements provide additional dimensions of clinical importance [9–11].

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This distinction is particularly important for severity assessment. A patient with rare asymptomatic ectopic beats and a patient with sustained ventricular tachycardia have different outcomes, even if both meet a broad definition of arrhythmia. Likewise, a normal short ECG does not establish the absence of intermittent events. The ambulatory ECG consensus emphasizes selecting monitoring according to the clinical question and symptom frequency [11]. Comparisons between patient groups are only meaningful when their opportunities for detecting an event are sufficiently similar.

Combining rhythm monitoring with clinical context

A practical assessment begins with a documented rhythm history, medication review and a standard ECG. Further testing should respond to symptoms, baseline abnormalities and suspected structural disease. Ambulatory monitoring can relate intermittent symptoms to rhythm and characterize event burden [11]. Echocardiography, cardiac biomarkers or advanced imaging may help investigate an indicated cardiac differential, but this review does not establish a requirement to perform every investigation in every patient with spinal tuberculosis.

QT measurements should use a consistent method, with manual review of doubtful automated values and an explicitly reported correction formula. Serial comparisons should consider heart rate and recording conditions. The torsade prevention statement identifies marked QTc prolongation, including values above 500 ms, as a clinically important warning signal [10]. Such a finding requires clinical review of the tracing, medicines and reversible factors; it is not equivalent to a documented episode of torsade de pointes.

Heart rate variability as an exploratory measure

Heart rate variability can characterize fluctuations between normal beats when acquisition and processing follow defined standards [12]. It is not a direct assay of adrenergic or cholinergic receptor activity. Recordings differ according to duration, rhythm quality, activity and other conditions, so methodological consistency is essential. Findings from spinal cord injury research provide a rationale for exploratory assessment [5], but do not validate an HRV threshold for predicting arrhythmia in spinal tuberculosis.

An appropriate research report should state the recording duration, artifact handling, treatment of ectopic beats and the selected time-domain or frequency-domain measures. HRV analysis in a recording dominated by atrial fibrillation cannot be interpreted as ordinary sinus rhythm autonomic variability. Any association between an HRV measure and a clinical rhythm outcome should be distinguished from diagnostic discrimination or prognostic performance. These are separate questions requiring separate analyses.

Implications for future studies

A prospective cohort could distinguish three clinically useful categories: patients with documented arrhythmia predating spinal tuberculosis; patients with an arrhythmia first documented after its diagnosis; and patients with no arrhythmia detected during a defined

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observation period. These categories describe chronology and detection. The second category should not automatically be called tuberculosis-induced arrhythmia, and the third should not be interpreted as lifelong absence of arrhythmia. A predefined baseline assessment and comparable surveillance would strengthen both classifications.

For incident-event analysis, the population at risk should consist of patients without the specified arrhythmia at baseline. Patients with established arrhythmia can contribute a separate analysis of burden or deterioration. Mixing prevalent and incident events would obscure whether a proposed predictor relates to disease onset, persistence or simply detection. If the question is whether spinal tuberculosis carries additional risk compared with other tuberculosis sites, a suitable non-spinal tuberculosis comparison group would address that question more directly than internal rhythm categories alone.

A feasible core dataset would combine ECG and monitoring results with neurological status, imaging-defined cord involvement, actual medication exposure, key electrolyte measurements and pre-existing cardiac disease. Disease activity and treatment stage should be recorded at each assessment. The study should select one primary outcome in advance, define follow-up and account for surgery, changes in medication and loss to follow-up. A chronology linking these observations would be more informative than an extensive panel of biomarkers collected at a single time point.

Prediction requires particular restraint. Sample size should be justified by the expected event frequency, the intended analysis and the number of candidate parameters, rather than an arbitrary equal allocation to three groups. Small datasets should emphasize descriptive estimates and a limited number of prespecified associations. A proposed prediction model would need internal validation, calibration assessment and subsequent external validation before clinical use. Correlation of a biomarker with arrhythmia cannot by itself demonstrate a causal pathway or justify a screening threshold.

Limitations of the evidence and this review

The central limitation is the mismatch between the target population and much of the available cardiac evidence. Drug trials, traumatic spinal cord injury cohorts and myocardial case reports each inform only part of the question. The narrative search was selective, and access to some publications was limited to abstracts or indexed extracts. No pooled prevalence, comparative effect estimate or formal certainty rating is therefore presented. The review also cannot establish that no relevant spinal-specific research exists.

Future synthesis would benefit from a reproducible database search, multilingual screening and detailed extraction of whether spinal cases were included in broader tuberculosis cohorts. Until such work and prospective clinical data are available, mechanisms inferred from neighboring fields should remain explicitly labeled as hypotheses. The proposed assessment and research framework is an interpretation of the reviewed evidence, not a tested intervention or an endorsed disease-specific guideline.

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Conclusion

Arrhythmias detected in patients with spinal tuberculosis require attention to chronology, neurological involvement, baseline cardiac disease, metabolic conditions and actual medication exposure. The strongest directly relevant pharmacological evidence concerns ventricular repolarization during drug-resistant tuberculosis treatment. Autonomic studies and myocardial case reports provide complementary, indirect evidence. The immediate research priority is consistent rhythm ascertainment in a defined spinal tuberculosis cohort, with clear separation of incident arrhythmia, pre-existing disease and QTc abnormalities. This approach would support clinically meaningful risk assessment without overstating causation.

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