Original Article | Journal of Orthopaedic Case Reports 2026 September:16(9):Page 531-536

Association of Vitamin D Receptor Gene Polymorphism (FokI) with Susceptibility to Musculoskeletal Tuberculosis: A Case–Control Study from Central India

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Learning Point of the Article:

The Vitamin D receptor (VDR) FokI ff genotype and f allele were more frequent in patients with musculoskeletal tuberculosis than in controls in this Central Indian cohort, suggesting that host genetic variation in Vitamin D receptor signaling may act alongside known socioeconomic and nutritional risk factors in determining susceptibility to musculoskeletal tuberculosis; given the preliminary, single-center nature of this association, it should prompt further multicentric and functional validation rather than immediate clinical application.

Abstract

Aim and Background: Musculoskeletal tuberculosis (MSKTB) constitutes a significant proportion of extra-pulmonary tuberculosis (TB), particularly in developing countries like India. While host genetic factors are known to influence susceptibility to TB, the Vitamin D receptor (VDR) gene, particularly the FokI polymorphism, has been implicated in immune regulation and susceptibility to pulmonary and extra-pulmonary TB, data on genetic predisposition to MSKTB remain limited. The present study aimed to investigate the role of the VDR FokI gene polymorphism in determining susceptibility to MSKTB.

Materials and Methods: This study included 110 patients with confirmed MSKTB and 112 controls without TB, recruited from a tertiary care institution over a 3-year period. Clinical and demographic data were obtained. Genomic DNA was extracted from peripheral venous blood samples, and VDR FokI polymorphism was detected using polymerase chain reaction-based restriction fragment length polymorphism analysis. Genotype and allele frequencies were compared using a Chi-square test, and odds ratios (OR) with 95% confidence intervals (CI) were calculated.

Results: The ff genotype was significantly more frequent in cases (17.3%) compared to controls (9.8%) and was associated with increased risk of MSKTB (OR = 2.30, 95% CI: 1.03–5.15, P = 0.04). The polymorphic f allele frequency was also significantly higher in cases than controls (38.2% vs. 28.1%; OR = 1.58, 95% CI: 1.05–2.37, P = 0.023). However, the dominant and recessive genetic models showed a non-significant association. Demographically, MSKTB was more common among females, individuals from rural backgrounds, those of lower socioeconomic status, and those with low body mass index.

Conclusion: The VDR FokI polymorphism, particularly the ff genotype and f allele, is associated with increased susceptibility to MSKTB. These findings highlight the potential role of genetic factors in MSKTB and may aid in identifying at-risk populations.

Keywords: Musculoskeletal tuberculosis, Vitamin D receptor, Vitamin D receptor Gene, FokI polymorphism, genetic susceptibility, tuberculosis.

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Access this article online

Website: www.jocr.co.in

DOI: https://doi.org/10.13107/jocr.2026.v16.i09.8144

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Submitted: 28/06/2026; Review: 04/07/2026; Accepted: August 2026; Published: September 2026

DOI: https://doi.org/10.13107/jocr.2026.v16.i09.8144

Introduction

Aim and Background

Tuberculosis (TB) continues to pose a significant global public health challenge, particularly in developing countries such as India. In 2024, approximately 10.7 million people worldwide developed TB. The highest burden is observed in the Southeast Asia region, accounting for approximately 34% of cases [1]. Several risk factors contribute to TB development, including human immunodeficiency virus (HIV) infection, diabetes mellitus, malnutrition, smoking, and immunosuppressive therapies. Among these, HIV is the strongest risk factor, significantly increasing both reactivation and progression of latent infection [2].

Musculoskeletal TB (MSKTB) makes up about 10% of all extra-pulmonary TB cases. Moreover, it ranks as the third most common form of extra-pulmonary TB, following pleural and lymphatic involvement [3]. The spine represents the most commonly affected site, nearly 50% of all MSKTB patients [3]. MSKTB often presents insidiously over a prolonged period, and diagnosis may be delayed. Timely diagnosis of bone and joint disease is crucial for minimizing disability and optimizing patient outcomes. Compromise in immune response or impairment of host resistance to Mycobacterium tuberculosis leads to the development of TB, as proven by the increased susceptibility to TB in patients with HIV and patients on anti-tumor necrosis factor treatment [4]. The cause for active TB in individuals without any immunodeficiency remains unclear.

Multiple factors have been advocated for the development of TB. Several studies have shown that various genetic variations at the gene level also play a role in making individuals susceptible to TB [5]. Multiple genetic studies and human epidemiological surveys have shown genetic associations of TB in humans and animals [6, 7, 8].

Mutations in genes involved in immune regulation, including interferon-gamma (IFN-γ), interleukins (IL), and signal transducers, have been associated with severe mycobacterial infections. In addition, genetic variations in human leukocyte antigen, P2X7 receptor, and Vitamin D receptor (VDR) genes have been reported to increase susceptibility to pulmonary TB [9].

Vitamin D has an important role not only in bone formation, resorption, metabolism, and healing, but also acts as an immunomodulatory molecule, contributing to the innate immune response against infectious agents such as M. tuberculosis [10].

Vitamin D also plays a role in various immune processes, e.g., monocyte differentiation, lymphocyte proliferation, and cytokine (IL2, IFN-γ, and IL1) secretions. It exerts its effect by interacting through VDR. VDR is coded by the VDR gene present on the long arm of chromosome 12, at the 12q13–14 position, and it contains six promoter regions (exons 1a–f), which are alternatively spliced non-translating regions, and eight protein-coding exons (exons 2–9). Among the various VDR gene single-nucleotide polymorphisms identified to date, FokI, rs2228570 located in exon 2, BsmI, rs1544410 and ApaI, rs7975232 located in intron 8, and TaqI, rs731236 located in exon 9, have been studied the most [11]. Of the various VDR gene polymorphisms, the ff variant genotype of FokI polymorphism of the VDR gene was shown to be associated with risk for pulmonary TB in the ethnic Gujarati population residing in London [12]. Recent studies involving South Indian patients with pulmonary TB, as well as a preliminary analysis of TaqI polymorphism in spinal TB, have suggested that variations in the VDR gene may influence individual susceptibility or resistance to TB [13, 14].

While several studies have explored genetic factors influencing susceptibility to pulmonary TB, there remains a paucity of comprehensive data regarding genetic susceptibility to MSKTB. Identifying such genetic determinants may facilitate recognition of at-risk populations and aid in the development of targeted preventive and therapeutic strategies for MSKTB.

Materials and Methods

This study was approved by the Institutional Human Ethics Committee (IHEC) of the institution (IHEC Ref Number: 2020/PG/July/30) on February 24, 2021. All participants provided written informed consent before enrolment in the study. This research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Participants were recruited from the outdoor and indoor facilities of the orthopaedics department of a tertiary institute in Central India from February 2021 to December 2023. All patients with MSKTB confirmed by histopathology or molecular diagnostic methods (e.g., Cartridge-Based Nucleic Acid Amplification Test) were included, excluding those who did not provide consent. Controls were recruited from admitted patients of the orthopedics ward with no current clinical features or history of TB.

Demographic information of cases and controls was collected in a standardized data collection form. Age, gender, and residence were recorded. Body weight and height were recorded for the calculation of body mass index (BMI), level of education, occupation, and monthly family income to determine socioeconomic status (Modified Kuppuswamy Socioeconomic Scale, 2020), and site of involvement.

Molecular analysis

For DNA extraction, 5 mL of peripheral venous blood was collected from cases and controls into ethylenediaminetetraacetic acid vials and stored at –20°C.

DNA was isolated using the QIAamp DNA Mini Kit (Qiagen, Germany), according to the manufacturer's protocol. Extracted DNA's purity and concentration were confirmed using the NanoDrop 1000 Spectrophotometer (Thermo Fisher Scientific, USA). The mean DNA concentration was approximately 270 ng/μL, and the average purity ratio was 1.6. The isolated DNA fragment was amplified with polymerase chain reaction (PCR) using a thermocycler. The PCR products were digested with the FokI restriction enzyme at 37°C for 2 h.

Table 1: Distribution of demographic characteristics among patients with musculoskeletal tuberculosis

Variable	Category	Cases (n=110), n (%)
Gender	Male	42 (38.2)
	Female	68 (61.8)
Age group (years)	<20	12 (10.9)
	21–40	25 (22.7)
	41–60	45 (40.9)
	>60	28 (25.5)
Socioeconomic status (modified Kuppuswamy scale 2020)	Upper	10 (9.1)
	Upper middle	18 (16.4)
	Lower middle	40 (36.4)
	Upper lower	23 (20.9)
	Lower	19 (17.3)
Background	Urban	42 (38.2)
	Rural	68 (61.8)
BMI	<18.5	33 (30.0)
	18.5–25	49 (44.5)
	25–30	28 (25.5)
Site of involvement	Spine	53 (48.2)
	Knee	14 (12.7)
	Distal femur	11 (10)
	Sacroiliac joint	8 (7.3)
	Hip	6 (5.5)
	Elbow	6 (2.7)
	Sternoclavicular joint	3 (2.7)
	Proximal Tibia	3 (2.7)
	Wrist	3 (2.7)
	Finger	3 (2.7)
	Shoulder	2 (1.8)

The enzyme recognizes the polymorphic site and cleaves the 265 bp fragment into two fragments of 196 bp and 69 bp. A total of 20 μL of the digested product was electrophoresed on a 4% agarose gel stained with ethidium bromide. DNA fragments were visualized under ultraviolet transillumination. Smaller fragments migrated faster compared to larger fragments, and fragment sizes were determined by comparison with a DNA ladder.

Genotypes were interpreted based on band patterns, e.g., homozygous polymorphic allele (ff): Two bands at 196 bp and 69 bp; heterozygous (Ff): Three bands at 265 bp, 196 bp, and 69 bp; homozygous wild type (FF): Single band at 265 bp.

Statistical analysis

All statistical analyses were conducted using IBM Statistical Package for the Social Sciences Statistics. Categorical variables were presented as frequencies and percentages. Genotype and allele frequencies of the VDR FokI polymorphism were compared between MSKTB cases and healthy controls using the Chi-square test. Odds ratios (ORs) were calculated for the association between the polymorphic allele in cases and controls with 95% confidence intervals (Cis). Genotype distribution in the control group was according to Hardy–Weinberg equilibrium and was evaluated using the Chi-square goodness-of-fit test. P-value below 0.05 was considered indicative of statistical significance.

Results

Demographic profile of MSKTB patients is shown in Table 1. Among the cases (n = 110), the majority were females (61.8%). Most patients belonged to the age group of 41–60 years (40.9%). A large proportion of cases were from lower socioeconomic strata, particularly the lower middle class (36.4%) and rural backgrounds (61.8%). In addition, 30% of patients were underweight (BMI <18.5 kg/m2).

The distribution of VDR FokI polymorphism genotypes among cases and controls is shown in Table 2. Among 110 cases of MSKTB, 45 (40.9%) had the FF genotype, 46 (41.8%) had the Ff genotype, and 19 (17.3%) had the ff genotype. In comparison, among 112 controls, 60 (53.6%) had the FF genotype, 41 (36.6%) had the Ff genotype, and 11 (9.8%) had the ff genotype.

The spine was the most frequently affected site, with other commonly involved locations including the knee, distal femur, hip joint, and sacroiliac joint.

The frequency of the mutant f allele was higher in cases (38.2%) compared to controls (28.1%). Using the FF genotype as the reference, the Ff genotype showed a non-significant increase in

Table 2: Comparison of VDR FokI polymorphism genotypes among cases and controls

Parameter	Cases (n=110) (%)	Controls (n=112) (%)	OR (95% CI)	P-value
Genotype distribution				
FF	45 (40.9)	60 (53.6)	Reference	_
Ff	46 (41.8)	41 (36.6)	1.49 (0.84–2.63)	0.17
Ff	19 (17.3)	11 (9.8)	**2.30 (1.03–5.15)**	**0.04**
Genetic models				
Dominant (Ff+ff vs. FF)	65	52	1.67 (0.98–2.83)	0.06
Recessive (ff vs. Ff+FF)	19	11	1.92 (0.87–4.21)	0.1
Allele f versus F	84 versus 136	63 versus 161	**1.58 (1.05–2.37)**	**0.023**

VDR: Vitamin D receptor, OR: Odds ratio, CI: Confidence interval

odds of MSKTB (OR = 1.49, P = 0.17). In contrast, the ff genotype was associated with significantly increased odds (OR = 2.30, 95% CI: 1.03–5.15, P = 0.04).

In the dominant model (Ff + ff vs. FF), the OR was 1.67 (P = 0.06), whereas in the recessive model (ff vs. Ff + FF), the OR was 1.92 (P = 0.10). Allelic analysis demonstrated that the f allele was significantly associated with MSKTB (OR = 1.58, 95% CI: 1.05–2.37, P = 0.023).

Discussion

This case–control study evaluated the demographic profile and the FokI polymorphism of the VDR gene in 110 patients with MSKTB and 112 controls. The demographic data revealed that most patients were females, belonged to rural backgrounds, and were from lower socioeconomic strata. A significant proportion were also underweight. These findings reinforce the well-established concept that TB is not only a biological disease but also strongly influenced by social determinants such as nutrition, living conditions, and access to healthcare.

With respect to the site of involvement, the spine was the most affected site, followed by the knee, distal femur, sacroiliac joint, hip, and other joints. This pattern of distribution is largely similar to previously published studies on MSKTB [15, 16].

On molecular evaluation, our findings demonstrate that the mutant f allele was significantly more frequent among cases than controls, with the ff genotype showing a significant association with MSKTB. This suggests that the FokI polymorphism of the VDR gene may contribute to increased susceptibility to MSKTB.

Similar findings have been reported by Panda et al. [17] in patients with pulmonary TB, where the ff genotype was significantly associated with the disease. Further, various meta-analyses including genetic studies in pulmonary and extra-pulmonary TB patients also showed a significant correlation with the polymorphic allele (ff) and recessive model (ff vs. Ff+FF) of FokI polymorphism [18, 19, 20]

A similar study conducted at All India Institute of Medical Sciences Delhi involving 150 patients with active pulmonary TB, 150 TB contacts, and 150 healthy controls reported comparable findings, demonstrating a higher frequency of the "ff" genotype among patients, whereas the "FF" genotype was significantly more prevalent in the control group ($\chi 2$ = 25.22, df of 4, and P = 0.0001) [17].

Interestingly, while the ff genotype showed a statistically significant association with MSKTB in our study, the dominant (P = 0.06) and recessive (P = 0.10) genetic models did not reach statistical significance, although a trend toward increased risk was observed. Given the relatively small cell sizes underlying the genotypic comparison (19 ff cases vs. 11 ff controls), this inconsistency across models means the precise mode of inheritance cannot be confirmed from the present data, and the genotypic and allelic associations should be interpreted with appropriate caution pending replication in larger cohorts. No correction for multiple comparisons was applied across the genotype, allele, and genetic-model analyses; under a conservative Bonferroni-adjusted threshold, neither association would retain statistical significance, and these findings should therefore be regarded as preliminary rather than definitive. Our study has several important limitations. As the research was carried out in a single tertiary care institution and included a modest number of participants, the study was not powered a priori for genotype-model or site-stratified comparisons, and the findings may not be representative of the wider population; given known regional and ethnic variation in VDR allele frequencies across India, formal assessment of population stratification (e.g. ethnicity) was also not performed, though cases and controls were drawn from the same hospital and control genotypes conformed to Hardy–Weinberg equilibrium. Larger multicentric studies involving diverse populations are required to further validate these observations. Furthermore, only a single polymorphism of the VDR gene was studied, whereas susceptibility to TB is likely influenced by multiple genetic and environmental factors, including other VDR polymorphisms (BsmI, ApaI, TaqI) and non-VDR susceptibility loci. Genotype–disease associations were assessed using unadjusted ORs; potential confounders such as age, sex, BMI, and socioeconomic status were not incorporated into a multivariable model, and several established TB risk factors – household TB contact, Bacille

Calmette-Guérin vaccination status, HIV status, diabetes mellitus, smoking, and immunosuppressive drug use – were not systematically recorded, so residual confounding from these factors cannot be excluded. Controls were recruited from hospitalized orthopedic patients rather than a community-based healthy sample; while this minimized differences in healthcare access and documentation quality, it introduces the possibility of selection bias if any control-group orthopedic diagnoses are themselves associated with VDR genotype. Serum 25-hydroxyvitamin D levels were not measured, and no functional data (VDR expression, cathelicidin induction, or monocyte/macrophage antimycobacterial activity) were generated in our cohort; consequently, this study demonstrates a statistical association but does not establish the underlying biological mechanism, nor does it demonstrate causality, which cannot be inferred from a cross-sectional case–control design. Genotyping relied on PCR-Restriction Fragment Length Polymorphism without independent confirmation by an orthogonal method such as Sanger sequencing in a subset of samples. MSKTB was analyzed as a composite entity across multiple anatomical sites; due to small numbers at individual non-spinal sites, site-specific genetic associations could not be evaluated. Finally, this study did not include a pulmonary TB comparator arm, and therefore cannot determine whether the FokI association reflects a general susceptibility to M. TB infection or a susceptibility more specific to musculoskeletal or bone involvement, given Vitamin D's additional independent role in bone metabolism.

In our literature search, we did not find any studies evaluating VDR gene polymorphisms exclusively in patients with MSKTB. This study contributes to the limited body of literature on genetic susceptibility to MSKTB, particularly in the Indian context.

Conclusion

This case–control study identifies a statistical association between the FokI polymorphism of the VDR gene and susceptibility to MSKTB, though the modest sample size, lack of correction for multiple comparisons, and other limitations discussed above mean these findings should be regarded as preliminary and hypothesis-generating rather than definitive. The mutant f allele and ff genotype were more frequent among patients with MSKTB compared with healthy controls; however, as a cross-sectional case–control design, this study can establish association only, not causation, and does not on its own demonstrate a functional or mechanistic link between the FokI variant and impaired host defense against Mycobacterium TB. The findings also highlight the influence of demographic and socioeconomic factors, including rural residence, lower socioeconomic status, and undernutrition, in the occurrence of MSKTB, underscoring that MSKTB susceptibility is likely multifactorial rather than attributable to a single genetic locus. To the best of our knowledge, this is among the very few studies to evaluate VDR FokI polymorphism exclusively in patients with MSKTB. Confirmation in larger, multicentric, multi-ethnic cohorts – incorporating multivariable adjustment for confounders, measurement of serum 25-hydroxyvitamin D and VDR expression, assessment of additional VDR and non-VDR genetic markers, and, where feasible, a pulmonary TB comparator arm – is warranted before these findings can inform clinical risk stratification or screening strategies for MSKTB.

Clinical Message

VDR FokI genotyping is not yet ready for routine clinical use, but our findings add to the evidence that impaired Vitamin D receptor signaling may influence susceptibility to MSKTB. Until larger, functionally validated, multicentric studies are available, VDR genotype should be regarded as a research tool rather than a diagnostic or prognostic marker for individual patient management in everyday practice.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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Conflict of Interest: Nil
Source of Support: Nil

Consent: The authors confirm that informed consent was obtained from the patient for publication of this article

How to Cite this Article

Gujar HR, Goyal RR, Verma VK, Chaware P, Santoshi JA, Aduri TA. Association of Vitamin D Receptor Gene Polymorphism (FokI) with Susceptibility to Musculoskeletal Tuberculosis: A Case–Control Study from Central India. Journal of Orthopaedic Case Reports 2026 September;16(09): 531-536.