



Original Article

Vitamin D Status and Mineral Metabolism in Patients with Acute Spinal Cord Injury: A Hospital-Based Cross-Sectional Study

Dr Tushar Bayla¹, Dr Tirpti Verma², Dr Kamal Kant Sain³, Dr Nikhil Agarwal⁴, Dr Ranjana Gothwal⁵, Dr Geeta Pachori⁶

¹Postgraduate Resident, Department of Biochemistry, SMS Medical College and Attached Hospitals, Jaipur

²Associate Professor, Department of Biochemistry, SMS Medical College and Attached Hospitals, Jaipur

³Associate Professor, Department of Physical Medicine and Rehabilitation, RUHS College of Medical Sciences, Jaipur

⁴Assistant Professor, Department of Physical Medicine and Rehabilitation, SMS Medical College and Attached Hospitals, Jaipur

⁵Postgraduate Resident, Department of Physical Medicine and Rehabilitation, Mahatma Gandhi Medical College and Hospital, Jaipur

⁶Senior Professor, Department of Pathology, JLN Medical College, Ajmer

OPEN ACCESS

Corresponding Author:

Dr Nikhil Agarwal

Assistant Professor, Department
of Physical Medicine and
Rehabilitation, SMS Medical
College and Attached Hospitals,
Jaipur, Rajasthan, India;

Email: coolniks87@gmail.com

Received: 16-06-2026

Accepted: 05-07-2026

Available online: 22-07-2026

ABSTRACT

Background: Vitamin D deficiency is highly prevalent worldwide and is particularly common among patients with acute spinal cord injury (SCI) owing to immobilization, reduced sunlight exposure, and altered bone metabolism. The interaction between serum vitamin D, calcium, parathyroid hormone (PTH), and alkaline phosphatase (ALP) plays an important role in skeletal homeostasis following SCI. However, data from the Indian population remain limited.

Aim: To evaluate serum vitamin D, calcium, intact parathyroid hormone, and alkaline phosphatase levels in patients with acute spinal cord injury and to determine the correlation between vitamin D and these biochemical parameters.

Materials and Methods: A hospital-based cross-sectional study was conducted in the Departments of Biochemistry and Physical Medicine and Rehabilitation at a tertiary care centre in Jaipur from July 2024 to August 2025. Two hundred consecutive adults with acute traumatic spinal cord injury were enrolled. Serum 25-hydroxyvitamin D [25(OH)D], total calcium, intact parathyroid hormone, and alkaline phosphatase were measured using standardized laboratory methods. Data were summarized descriptively; group comparisons and correlation analyses were performed as appropriate, with a two-sided $p < 0.05$ considered statistically significant.

Results: Among 200 patients, vitamin D deficiency (< 20 ng/mL) was observed in 121 (60.5%), and 41 (20.5%) had vitamin D insufficiency; thus, 162 (81.0%) had 25(OH)D levels below 30 ng/mL. Hypocalcaemia was present in 61 (30.5%) patients, and iPTH was above the laboratory reference interval in 26 (13.0%). Serum ALP was within the reference interval in 178 (89.0%) patients. Mean iPTH differed significantly across vitamin D categories ($p < 0.001$). Serum 25(OH)D showed a moderate inverse correlation with iPTH ($r = -0.49$, $p < 0.001$), a weak positive correlation with calcium ($r = 0.16$, $p = 0.03$), and no significant correlation with ALP ($r = 0.07$, $p = 0.32$).

Conclusion: Vitamin D deficiency was common in this cohort of patients with acute spinal cord injury and was associated with higher iPTH concentrations. These findings support early assessment of the vitamin D–calcium–PTH axis while prospective studies determine whether correction improves skeletal or rehabilitation outcomes.

Keywords: Acute spinal cord injury; Vitamin D deficiency; 25-Hydroxyvitamin D; Parathyroid hormone; Calcium; Alkaline phosphatase; Bone metabolism.

INTRODUCTION

Spinal cord injury (SCI) produces motor, sensory, and autonomic impairment and is accompanied by rapid skeletal unloading. Bone resorption increases early after injury, bone mineral density declines predominantly in the sublesional skeleton, and long-term fracture risk is increased.¹⁻³

Vitamin D is a secosteroid hormone central to calcium and phosphate homeostasis, skeletal mineralization, neuromuscular function, and immune regulation. Serum 25-hydroxyvitamin D [25(OH)D] is the principal clinical marker of vitamin D status. Deficiency reduces intestinal calcium absorption and may provoke compensatory elevation of parathyroid hormone (PTH), thereby increasing skeletal calcium mobilization.⁴⁻⁷

Patients with SCI may be vulnerable to low vitamin D status because of restricted mobility, reduced sunlight exposure, nutritional limitations, and altered body composition. During the early post-injury period, biochemical markers of bone resorption can rise markedly while formation markers, including total alkaline phosphatase (ALP), may remain within reference limits. Simultaneous assessment of 25(OH)D, calcium, intact PTH (iPTH), and ALP may therefore provide complementary information on mineral metabolism.^{1,8,9}

Vitamin D deficiency is also highly prevalent in India despite abundant sunlight. Darker skin pigmentation, limited effective ultraviolet-B exposure, predominantly indoor lifestyles, low dietary vitamin D intake, and limited food fortification have all been implicated. Reviews from India have reported a substantial burden of low 25(OH)D across age groups and settings.^{10,11}

Studies in SCI populations have reported frequent vitamin D inadequacy at admission to rehabilitation and in chronic SCI. However, Indian data from patients in the acute phase remain limited, and relatively few studies have examined 25(OH)D together with calcium, iPTH, and ALP.¹²⁻¹⁴

Therefore, the present hospital-based cross-sectional study evaluated vitamin D status and its relationship with serum calcium, iPTH, and ALP in adults with acute traumatic SCI admitted to a tertiary care teaching hospital.

AIM AND OBJECTIVES

Aim

To evaluate serum vitamin D status and its correlation with serum calcium, intact parathyroid hormone, and alkaline phosphatase in patients with acute spinal cord injury.

Objectives

1. To estimate serum 25-hydroxyvitamin D [25(OH)D], calcium, intact parathyroid hormone (iPTH), and alkaline phosphatase (ALP) levels in patients with acute spinal cord injury.
2. To determine the prevalence of vitamin D deficiency among patients with acute spinal cord injury.
3. To evaluate the correlation between serum vitamin D and calcium, iPTH, and ALP.
4. To compare serum vitamin D levels according to demographic and injury-related characteristics.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based cross-sectional observational study was conducted in the Department of Biochemistry, Sawai Man Singh Medical College and Attached Hospitals, Jaipur, Rajasthan, India, in collaboration with the Department of Physical Medicine and Rehabilitation. Recruitment occurred from July 2024 to August 2025 after approval by the Institutional Ethics Committee. Reporting was guided by the STROBE statement for cross-sectional studies.¹⁵

Study Population: A total of 200 consecutive adult patients with clinically and radiologically confirmed acute spinal cord injury (SCI) admitted to the Department of Physical Medicine and Rehabilitation during the study period were enrolled in the study.

Inclusion Criteria: The following patients were included in the study:

- Adults aged 18 years or older.
- Patients with acute traumatic spinal cord injury.
- Patients admitted during the acute phase of injury.
- Patients who provided written informed consent or whose legally authorized representatives provided consent.

Exclusion Criteria: Patients were excluded if they had:

- History of vitamin D or calcium supplementation before enrolment.
- Chronic kidney disease, chronic liver disease, metabolic bone disease, parathyroid disorders, or malignancy.

- Current treatment with corticosteroids, anticonvulsants, bisphosphonates, or other drugs affecting calcium metabolism.
- Incomplete clinical records or inadequate blood samples.

Clinical Assessment: A detailed history and clinical examination were recorded on a predesigned case record form. Variables included age, sex, residence, diet, body mass index, mechanism and vertebral level of injury, duration since trauma, completeness of injury, and American Spinal Injury Association (ASIA) Impairment Scale grade, determined according to the International Standards for Neurological Classification of Spinal Cord Injury.¹⁶

Blood Sample Collection: After 12–14 hours of overnight fasting, approximately 5 mL of venous blood was collected under aseptic precautions into a plain vial. Samples were allowed to clot and centrifuged at 3500 rpm for 10 minutes. Serum was analysed promptly or stored under assay-specific recommended conditions.

The study workflow and participant enrolment are summarized in Figure 1.

	STUDY POPULATION (n = 200) Patients with acute onset SCI, ≥18 years Department of PMR, SMS Hospital, Jaipur	
	↓	
	WRITTEN INFORMED CONSENT OBTAINED	
	↓	
	DETAILED CLINICAL HISTORY & EXAMINATION Demographics: Age, Sex, Religion, Diet, Occupation, Education, Income Habits: Smoking, Alcohol Injury Details: Date, Duration, Mode of trauma, Vertebral level	
	↓	
	BLOOD SAMPLE COLLECTION 5 mL venous blood from antecubital vein After 12–14 hours of overnight fasting Collected in plain vial (without anticoagulant)	
	↓	
	SAMPLE PROCESSING Allowed to clot at room temperature for 1 hour Centrifuged at 3500 rpm for 10 minutes Serum separated for investigations	
	↓	
ROUTINE INVESTIGATIONS • Complete Blood Count (CBC) • Erythrocyte Sedimentation Rate (ESR)	SPECIAL INVESTIGATIONS • Serum Vitamin D [25(OH)D] — ADVIA Centaur XP • Serum Calcium — AU 5811 (Arsenazo III) • Serum iPTH — ADVIA Centaur XP • Serum ALP — Modified IFCC Kinetic	
	↓	
	DATA COMPILATION & STATISTICAL ANALYSIS Qualitative data: Frequencies & Percentages Quantitative data: Mean ± SD Correlation: Pearson / Spearman Tests: t-test, ANOVA, Chi-square Software: SPSS v26.0 p < 0.05 significant	
	↓	
	INTERPRETATION, INFERENCE & CONCLUSION	

Figure 1: Methodology Flowchart

Biochemical Analysis:

Serum 25-Hydroxyvitamin D [25(OH)D]: Serum 25(OH)D was measured by automated competitive chemiluminescent immunoassay on the ADVIA Centaur XP Immunoassay System (Siemens Healthineers, Germany). Vitamin D status was categorized as deficient (<20 ng/mL), insufficient (20–29.9 ng/mL), or sufficient (≥30 ng/mL), using the prespecified Endocrine Society thresholds.⁷

Serum Calcium: Serum total calcium was measured using the Arsenazo III photometric method on the Beckman Coulter AU5811 Clinical Chemistry Analyzer (Beckman Coulter Inc., USA). The reference interval adopted by the laboratory was 8.5–10.5 mg/dL.

Serum Alkaline Phosphatase (ALP): Serum ALP was estimated by a modified International Federation of Clinical Chemistry kinetic method using the Beckman Coulter AU5811 analyser. The laboratory reference interval was 44–147 U/L.

Serum Intact Parathyroid Hormone (iPTH): Serum intact parathyroid hormone (iPTH) was measured using a two-site sandwich chemiluminescent immunoassay on the ADVIA Centaur XP Immunoassay System. The reference interval adopted was 15–65 pg/mL.

Table 1. Laboratory Methods Used

Parameter	Method	Instrument	Reference Range
Serum 25(OH)D	Competitive chemiluminescent immunoassay	ADVIA Centaur XP	≥30 ng/mL
Serum Calcium	Arsenazo III photometric method	Beckman Coulter AU5811	8.5–10.5 mg/dL
Serum ALP	Modified IFCC kinetic method	Beckman Coulter AU5811	44–147 U/L
Serum iPTH	Two-site sandwich chemiluminescent immunoassay	ADVIA Centaur XP	15–65 pg/mL

Outcome Measures: The primary outcome was the prevalence of vitamin D deficiency among patients with acute spinal cord injury. The secondary outcomes included:

- Serum calcium concentration.
- Serum intact parathyroid hormone concentration.
- Serum alkaline phosphatase activity.
- Correlation between serum vitamin D and biochemical parameters.
- Association of serum vitamin D with demographic and injury-related characteristics.

Statistical Analysis: Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables were summarized as frequencies and percentages. Continuous variables were summarized as mean ± standard deviation because the reported analyses treated them as approximately normally distributed; distributions were examined using histograms, Q–Q plots, and the Shapiro–Wilk test. Two independent groups were compared using the independent-samples t-test, and three or more groups using one-way analysis of variance (ANOVA). When an omnibus ANOVA was significant, Bonferroni-adjusted pairwise comparisons were planned. Categorical variables were compared using the chi-square test or Fisher exact test when expected cell counts were small. Pearson correlation coefficients were used to quantify linear associations between serum 25(OH)D and continuous biochemical or clinical variables after checking linearity and influential outliers. All tests were two-sided, and $p < 0.05$ was considered statistically significant. Exact p-values are reported where available; no adjustment was applied to the exploratory correlation analyses.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee, Sawai Man Singh Medical College, Jaipur (Approval Ref. No. 1227/MC/EC/2024/S.No. 250; 25 October 2024). Written informed consent was obtained from all participants or their legally authorized representatives before enrolment. The study was conducted in accordance with the Declaration of Helsinki.¹⁷

RESULTS

Baseline Characteristics of the Study Population: A total of 200 patients with acute spinal cord injury (SCI) were included in the study. The mean age of the study population was 36.56 ± 10.92 years (range: 18–65 years). The highest proportion of patients belonged to the 26–35 years (30.0%) age group, followed by the 36–45 years (28.0%) age group. Male patients constituted 81.0% ($n = 162$) of the study population, while females accounted for 19.0% ($n = 38$). Most participants were residents of rural areas (78.0%) and 57.5% followed a vegetarian diet. Falls from height (48.0%) were the leading cause of spinal cord injury, followed by road traffic accidents (28.0%). Dorsal spinal injuries were the most common (43.0%), followed by lumbar (32.0%) and cervical injuries (20.5%). According to the American Spinal Injury Association (ASIA) Impairment Scale, 119 patients (59.5%) were classified as ASIA Grade A. Complete spinal cord injury was present in 136 patients (68.0%), whereas 64 patients (32.0%) had incomplete injury.

Vitamin D Status: The mean serum 25-hydroxyvitamin D [25(OH)D] concentration was 19.71 ± 10.99 ng/mL. Vitamin D deficiency (<20 ng/mL) was observed in 121 patients (60.5%), whereas 41 patients (20.5%) had vitamin D insufficiency (20–29.9 ng/mL). Only 38 patients (19.0%) had sufficient vitamin D levels (≥ 30 ng/mL). Overall, 162 patients (81.0%) demonstrated suboptimal vitamin D levels below 30 ng/mL. The distribution of vitamin D status among the study participants is illustrated in Figure 2.

Serum Calcium, Intact Parathyroid Hormone and Alkaline Phosphatase: Mean serum calcium was 8.83 ± 0.75 mg/dL. Hypocalcaemia (<8.5 mg/dL) was present in 61 (30.5%) patients and hypercalcaemia in 1 (0.5%). Mean iPTH was 41.82 ± 19.36 pg/mL; 26 (13.0%) patients had values above 65 pg/mL, 161 (80.5%) were within the reference interval, and 13 (6.5%) were below it. Mean ALP was 96.74 ± 31.63 U/L; 178 (89.0%) values were within the reference interval, 12 (6.0%) were elevated, and 10 (5.0%) were below it.

Comparison of Biochemical Parameters According to Vitamin D Status: Patients with vitamin D deficiency had significantly higher mean serum iPTH concentrations (48.73 ± 18.85 pg/mL) than those with vitamin D insufficiency (35.76 ± 15.14 pg/mL) or vitamin D sufficiency (26.36 ± 13.12 pg/mL) (ANOVA, $p < 0.001$). Although serum calcium concentrations were lower among vitamin D-deficient patients (8.75 ± 0.71 mg/dL), the difference did not reach statistical significance ($p = 0.09$). Similarly, no statistically significant difference in serum ALP concentrations was observed among the three vitamin D groups ($p = 0.35$).

Correlation Analysis: Pearson correlation analysis demonstrated a moderate negative correlation between serum 25-hydroxyvitamin D [25(OH)D] and serum intact parathyroid hormone (iPTH) ($r = -0.49$, $p < 0.001$), indicating that lower vitamin D concentrations were associated with higher iPTH levels. A weak positive correlation was observed between serum vitamin D and serum calcium ($r = +0.16$, $p = 0.03$). No statistically significant correlation was found between serum vitamin D and serum alkaline phosphatase (ALP) ($r = +0.07$, $p = 0.32$), body mass index (BMI) ($r = -0.11$, $p = 0.12$), age ($r = 0.00$, $p = 0.96$), or duration since trauma ($r = -0.03$, $p = 0.64$). The correlation coefficients and corresponding p -values are presented in Table 6.

Subgroup Analysis: No statistically significant differences in mean serum vitamin D concentrations were observed according to sex ($p = 0.64$), residence ($p = 0.11$), dietary habits ($p = 0.59$), vertebral level of injury ($p = 0.84$), or type of spinal cord injury ($p = 0.11$), indicating that vitamin D deficiency was consistently prevalent across all demographic and injury-related subgroups.

Table 2. Baseline Characteristics of Study Participants (n = 200)

Variable	Value
Mean age (years)	36.56 ± 10.92
Male	162 (81.0%)
Female	38 (19.0%)
Rural residence	156 (78.0%)
Urban residence	44 (22.0%)
Vegetarian	115 (57.5%)
Non-vegetarian	85 (42.5%)
Complete SCI	136 (68.0%)
Incomplete SCI	64 (32.0%)
ASIA Grade A	119 (59.5%)

Figure 2: Distribution According to Vitamin D Status

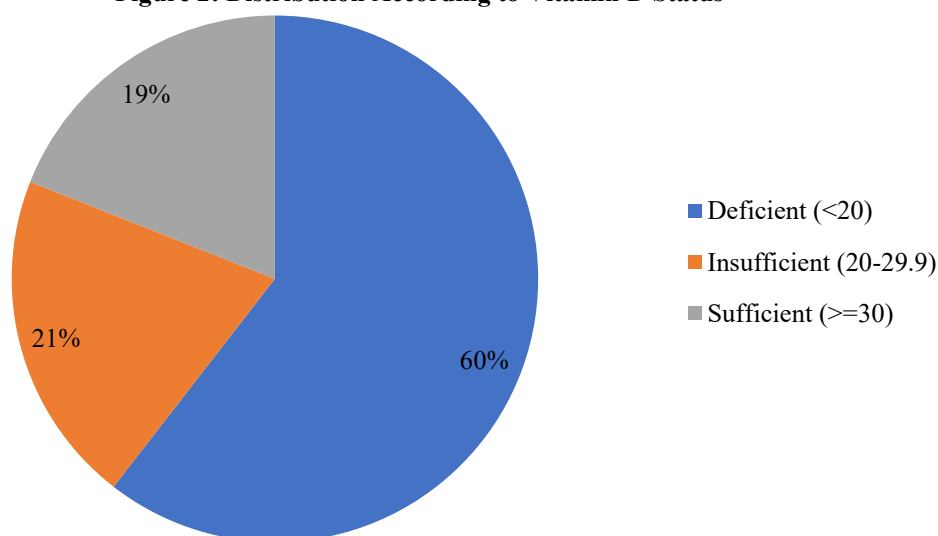


Table 3. Distribution of Vitamin D Status

Vitamin D Status	Frequency	Percentage
Deficient (<20 ng/mL)	121	60.5
Insufficient (20–29.9 ng/mL)	41	20.5
Sufficient (≥30 ng/mL)	38	19.0

Table 4. Biochemical Characteristics of the Study Population

Parameter	Mean ± SD	Abnormal Findings
Vitamin D (ng/mL)	19.71 ± 10.99	81% below 30 ng/mL
Calcium (mg/dL)	8.83 ± 0.75	61 (30.5%) low
Intact PTH (pg/mL)	41.82 ± 19.36	26 (13.0%) elevated
ALP (U/L)	96.74 ± 31.63	12 (6.0%) elevated; 10 (5.0%) low

Table 5. Comparison of Biochemical Parameters According to Vitamin D Status

Vitamin D Status	PTH (pg/mL)	Calcium (mg/dL)	ALP (U/L)
Deficient	48.73 ± 18.85	8.75 ± 0.71	94.31 ± 31.76
Insufficient	35.76 ± 15.14	9.04 ± 0.81	101.75 ± 35.10
Sufficient	26.36 ± 13.12	8.86 ± 0.75	99.07 ± 26.92
p-value	<0.001	0.09	0.35

Table 6. Correlation of Serum Vitamin D with Clinical and Biochemical Variables

Variable	Correlation coefficient (r)	p-value
Serum Calcium	+0.16	0.03
Serum iPTH	−0.49	<0.001
Serum ALP	+0.07	0.32
BMI	−0.11	0.12
Age	0.00	0.96
Duration since trauma	−0.03	0.64

DISCUSSION

This cross-sectional study found vitamin D deficiency in 60.5% of adults with acute SCI and 25(OH)D concentrations below 30 ng/mL in 81.0%. Lower 25(OH)D was associated with higher iPTH and, more weakly, with lower calcium, whereas ALP showed no significant correlation. The findings describe biochemical associations at a single time point and do not establish causality or treatment benefit.

Vitamin D Deficiency in Acute Spinal Cord Injury

Vitamin D deficiency was observed in 60.5% of patients, and a further 20.5% had insufficiency. The burden is clinically relevant because low vitamin D status may coexist with the rapid skeletal changes that follow SCI.

Waliullah et al. reported that 76% of young adults with acute thoracolumbar SCI had 25(OH)D levels below 30 ng/mL at admission. In a rehabilitation cohort of 100 patients with SCI, Nemunaitis et al. found vitamin D inadequacy or severe deficiency in 93%. Differences in case mix, thresholds, season, geography, and timing after injury may explain variation between studies.^{12,13}

The high prevalence in the present cohort may reflect both SCI-related reductions in mobility and sunlight exposure and the high background prevalence of vitamin D deficiency in India.^{10,11}

Serum Calcium

The mean serum calcium concentration in the present study was 8.83 ± 0.75 mg/dL, and approximately one-third of patients had hypocalcaemia. However, only a weak positive correlation was observed between serum vitamin D and calcium concentration.

Serum calcium can remain within or near the reference range despite low 25(OH)D because PTH-mediated renal calcium conservation and skeletal calcium mobilization help maintain extracellular calcium. Accordingly, total calcium alone is an insensitive screening marker for vitamin D deficiency.

Serum Intact Parathyroid Hormone

One of the most important findings of the present study was the significant inverse relationship between serum vitamin D and iPTH. Vitamin D-deficient patients had significantly higher serum iPTH concentrations than patients with vitamin D

insufficiency or sufficiency ($p < 0.001$). Pearson correlation analysis demonstrated a moderate negative correlation between serum vitamin D and iPTH ($r = -0.49$).

The inverse 25(OH)D–PTH relationship is biologically plausible: reduced vitamin D-dependent intestinal calcium absorption can stimulate PTH secretion, which promotes renal calcium conservation and bone resorption.^{4,7}

Hummel et al. likewise reported an inverse association between 25(OH)D and PTH in adults with chronic SCI. Their cohort differed from the present acute-care population, but the direction of association was consistent.¹⁴

Serum Alkaline Phosphatase

The mean serum ALP concentration was 96.74 ± 31.63 U/L, and most participants had values within the normal laboratory reference range. No statistically significant correlation was observed between serum vitamin D and ALP.

Total ALP was not significantly related to 25(OH)D in this study. This is compatible with longitudinal evidence showing marked increases in bone-resorption markers after acute SCI while bone-formation markers may change only modestly. A scoping review also found heterogeneous evidence for total or bone-specific ALP after SCI, limiting the interpretability of total ALP as a stand-alone early bone-turnover marker.^{9,18}

Clinical Implications

The findings support consideration of early assessment of 25(OH)D, calcium, and iPTH in patients with acute SCI, particularly when other osteoporosis risk factors are present. However, this study did not evaluate supplementation, fractures, bone mineral density, or rehabilitation outcomes; therefore, treatment recommendations and claims of improved outcomes should be based on clinical guidelines and prospective intervention studies rather than these cross-sectional data alone.

Strengths of the Study

Strengths include the sample size, consecutive recruitment, assessment during the acute phase, and concurrent measurement of several components of mineral metabolism. The study also contributes data from an Indian tertiary-care population, a setting in which evidence remains limited.

Limitations

This study has several limitations. Its cross-sectional design precludes causal or temporal inference. It was conducted at a single tertiary-care centre, which may limit generalizability. Season of sampling, sunlight exposure, dietary vitamin D intake, renal function indices, albumin-adjusted or ionized calcium, and assay calibration data were not incorporated into the reported analyses. Bone-specific ALP, CTX, PINP, fibroblast growth factor-23, and dual-energy X-ray absorptiometry were not measured. Multiple exploratory comparisons were performed without multiplicity adjustment, so nominally significant findings, particularly the weak calcium correlation, should be interpreted cautiously. Prospective multicentre studies with serial biochemical and bone-density assessments are warranted.

CONCLUSION

In this cohort of 200 adults with acute traumatic SCI, 60.5% were vitamin D deficient and 81.0% had 25(OH)D levels below 30 ng/mL.

Serum 25(OH)D was moderately inversely correlated with iPTH and weakly positively correlated with total calcium, while no significant correlation was observed with total ALP. These associations are consistent with altered vitamin D–PTH physiology but cannot establish causality.

Early biochemical assessment may help identify patients with vitamin D deficiency or disturbances of mineral metabolism. Whether screening and correction improve bone density, fracture risk, or rehabilitation outcomes should be determined in prospective controlled studies.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the faculty and staff of the Departments of Biochemistry and Physical Medicine and Rehabilitation, SMS Medical College and Attached Hospitals, Jaipur, for their support. The authors also thank Dr Shailendra Vashistha (Assistant Professor, Transplant Immunology HLA Lab, Dept of IHTM, GMC, Kota) and the VAssist Research team (www.thevassist.com) for their contribution in manuscript editing and submission process. The authors sincerely thank all patients who participated in the study.

CONFLICT OF INTEREST: The authors declare no conflict of interest.

SOURCE OF FUNDING: The authors declare that no external funding was received for this study.

AUTHOR CONTRIBUTIONS: Tushar Bayla: conceptualization, investigation, data curation, formal analysis, and drafting. Tirpti Verma: supervision, methodology, interpretation, and critical revision. Kamal Kant Sain, Nikhil Agarwal, and Ranjana Gothwal: participant recruitment, clinical assessment, data acquisition, and critical revision. Geeta Pachori: scientific review, interpretation, and critical revision. All authors approved the final manuscript and accept accountability for the work.

REFERENCES

1. Charmetant C, Phaner V, Condemine A, Calmels P. Diagnosis and treatment of osteoporosis in spinal cord injury patients: a literature review. *Ann Phys Rehabil Med*. 2010;53(10):655-668. doi:10.1016/j.rehab.2010.10.001.
2. Zleik N, Weaver F, Harmon RL, Le B, Radhakrishnan R, Jirau-Rosaly WD, et al. Prevention and management of osteoporosis and osteoporotic fractures in persons with a spinal cord injury or disorder: a systematic scoping review. *J Spinal Cord Med*. 2019;42(6):735-759. doi:10.1080/10790268.2018.1469808.
3. Soleyman-Jahi S, Yousefian A, Maheronnaghsh R, Shokraneh F, Abdollah Zadegan S, Soltani A, et al. Evidence-based prevention and treatment of osteoporosis after spinal cord injury: a systematic review. *Eur Spine J*. 2018;27(8):1798-1814. doi:10.1007/s00586-017-5114-7.
4. Christakos S, Dhawan P, Verstuyf A, Verlinden L, Carmeliet G. Vitamin D: metabolism, molecular mechanism of action, and pleiotropic effects. *Physiol Rev*. 2016;96(1):365-408. doi:10.1152/physrev.00014.2015.
5. Bikle DD. Vitamin D metabolism, mechanism of action, and clinical applications. *Chem Biol*. 2014;21(3):319-329. doi:10.1016/j.chembiol.2013.12.016.
6. Sassi F, Tamone C, D'Amelio P. Vitamin D: nutrient, hormone and immunomodulator. *Nutrients*. 2018;10(11):1656. doi:10.3390/nu10111656.
7. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2011;96(7):1911-1930. doi:10.1210/jc.2011-0385.
8. Jiang SD, Jiang LS, Dai LY. Mechanisms of osteoporosis in spinal cord injury. *Clin Endocrinol (Oxf)*. 2006;65(5):555-565. doi:10.1111/j.1365-2265.2006.02683.x.
9. Roberts D, Lee W, Cuneo RC, Wittmann J, Ward G, Flatman R, et al. Longitudinal study of bone turnover after acute spinal cord injury. *J Clin Endocrinol Metab*. 1998;83(2):415-422. doi:10.1210/jcem.83.2.4581.
10. Ritu G, Gupta A. Vitamin D deficiency in India: prevalence, causalities and interventions. *Nutrients*. 2014;6(2):729-775. doi:10.3390/nu6020729.
11. Aparna P, Muthathal S, Nongkynrih B, Gupta SK. Vitamin D deficiency in India. *J Family Med Prim Care*. 2018;7(2):324-330. doi:10.4103/jfmpe.jfmpe_78_18.
12. Waliullah S, Kumar D, Kumar D, Tewari PG, Kumar V, Srivastava RN. Prevalence of vitamin D deficiency in a young adult with acute spinal cord injury. *Cureus*. 2021;13(3):e13791. doi:10.7759/cureus.13791.
13. Nemunaitis GA, Mejia M, Nagy JA, Johnson T, Chae J, Roach MJ. A descriptive study on vitamin D levels in individuals with spinal cord injury in an acute inpatient rehabilitation setting. *PM R*. 2010;2(3):202-208. doi:10.1016/j.pmrj.2010.01.010.
14. Hummel K, Craven BC, Giangregorio L. Serum 25(OH)D, PTH and correlates of suboptimal 25(OH)D levels in persons with chronic spinal cord injury. *Spinal Cord*. 2012;50(11):812-816. doi:10.1038/sc.2012.67.
15. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *PLoS Med*. 2007;4(10):e296. doi:10.1371/journal.pmed.0040296.
16. Kirshblum SC, Burns SP, Biering-Sørensen F, Donovan W, Graves DE, Jha A, et al. International standards for neurological classification of spinal cord injury (revised 2011). *J Spinal Cord Med*. 2011;34(6):535-546. doi:10.1179/204577211X13207446293695.
17. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. *JAMA*. 2013;310(20):2191-2194. doi:10.1001/jama.2013.281053.
18. Ponzano M, Wiest MJ, Coleman A, Newton E, Pakosh M, Patsakos EM, et al. The use of alkaline phosphatase as a bone turnover marker after spinal cord injury: a scoping review of human and animal studies. *J Spinal Cord Med*. 2023;46(2):167-180. doi:10.1080/10790268.2021.1977905.