RESEARCH PAPER

Correlation of Serum Vitamin D Levels with Fracture Non-Union: A Prospective Observational Study

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ABSTRACT

Background: Fracture non-union represents clinically important failure of bone repair and is influenced by mechanical stability, vascularity, infection, systemic disease, smoking, nutritional status, and metabolic abnormalities. Vitamin D is integral to calcium-phosphate homeostasis, osteoblast function, callus mineralization, and skeletal remodeling, but its clinical relationship with fracture non-union remains incompletely defined. This study evaluated the association between serum 25-hydroxyvitamin D [25(OH)D] concentration and non-union following long-bone fractures.

Methods: A prospective observational cohort study included 180 adults with surgically treated long-bone fractures. Serum 25(OH)D was measured within 72 hours of definitive fixation. Concentrations were classified as deficient (<20 ng/mL), insufficient (20-29.9 ng/mL), or sufficient (>=30 ng/mL). Fracture healing was assessed clinically and radiographically. The primary outcome was fracture non-union. Correlations with time to radiographic union were assessed, and multivariable logistic regression was used to identify independent predictors of non-union.

Results: Mean serum 25(OH)D concentration was 21.3 +/- 9.4 ng/mL, and 104 patients (57.8%) were vitamin-D deficient. Non-union occurred in 32/180 patients (17.8%). Non-union was more frequent among vitamin-D-deficient patients than among patients with levels >=20 ng/mL (25.0% vs. 7.9%; unadjusted OR 3.89, 95% CI 1.51-10.00; p=0.005). Serum 25(OH)D correlated inversely with time to radiographic union (Spearman rho=-0.59; p<0.001). After adjustment for smoking, diabetes, open injury, age, and fracture location, vitamin-D deficiency remained independently associated with non-union (adjusted OR 3.36, 95% CI 1.25-9.01; p=0.016).

Conclusion: Lower serum vitamin-D concentrations were associated with prolonged fracture healing and increased odds of non-union. Vitamin-D status may constitute a readily identifiable and potentially modifiable component of the biological environment governing fracture repair. Prospective interventional trials are required to determine whether correction of deficiency translates into improved union rates.

Keywords: vitamin D; 25-hydroxyvitamin D; fracture healing; non-union; delayed union; long-bone fracture; metabolic bone health

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INTRODUCTION

Fracture healing is a coordinated biological process involving inflammation, angiogenesis, mesenchymal-cell recruitment, cartilaginous callus formation, mineralization, and subsequent bone remodeling. Failure of these events may culminate in delayed union or established non-union, producing prolonged disability, repeated surgery, impaired quality of life, and substantial healthcare utilization. Although instability, compromised vascularity, extensive soft-tissue injury, and infection remain major local determinants, the metabolic environment of the patient is increasingly recognized as an important contributor to successful bone regeneration [1-3]. Vitamin D is particularly relevant because of its established influence on calcium-phosphate homeostasis, skeletal mineralization, osteoblast differentiation, and bone turnover.

Vitamin-D deficiency is common among orthopaedic trauma populations. Gorter et al. reported that approximately 40% of adult fracture patients were deficient, with an even larger proportion having suboptimal vitamin-D concentrations [4]. Prospective orthopaedic-trauma data similarly demonstrate high rates of hypovitaminosis D across demographic and injury groups [5]. Such observations have generated interest in vitamin D not merely as a determinant of fracture susceptibility but as a potential biological modifier of post-fracture repair.

Clinical evidence, however, remains inconsistent. Gorter et al. observed a higher incidence of clinical delayed union among patients who remained vitamin-D deficient after supplementation compared with those who were vitamin-D replete [6]. Conversely, Haining et al. found no significant differences in major circulating vitamin-D metabolites between patients with established non-union and matched controls [7]. Bodendorfer et al. likewise reported no significant relationship between serum 25(OH)D and fracture-healing complications in supplemented orthopaedic-trauma patients [8]. These conflicting findings suggest that vitamin D may interact with mechanical, vascular, inflammatory, nutritional, and behavioral determinants rather than operating as an isolated causal factor.

The clinical question remains important because vitamin-D deficiency is identifiable through a widely available biochemical test and, when present, is potentially correctable. Establishing whether low 25(OH)D concentrations independently correlate with fracture non-union could therefore refine metabolic risk assessment and identify patients requiring closer surveillance. The present study was designed to determine the relationship between baseline serum 25(OH)D and subsequent fracture non-union in adults with long-bone fractures. Secondary objectives were to assess the relationship between vitamin-D status and time to radiographic union and to determine whether vitamin-D deficiency remained associated with non-union after adjustment for established clinical risk factors.

MATERIALS AND METHODS

Study design

A prospective observational cohort study was conducted among adults with surgically treated long-bone fractures. Consecutive eligible patients were enrolled according to predefined criteria. The study protocol was approved by the appropriate Institutional Ethics Committee, and written informed consent was obtained from every participant before enrolment.

Participants

Adults aged 18-70 years who had sustained an acute fracture of the tibia, femur, humerus, radius, or ulna and had undergone definitive operative fixation were included. Both closed fractures and Gustilo-Anderson grade I or II open fractures were eligible. Only patients in whom baseline biochemical testing could be completed within 72 hours of definitive fixation were recruited.

Patients were excluded when they had pathological fractures related to primary or metastatic malignancy, established metabolic bone disease other than osteoporosis, chronic kidney disease stage 4 or 5, severe chronic liver disease, prolonged systemic corticosteroid treatment, current pharmacological treatment for active rickets or osteomalacia, pregnancy, Gustilo-Anderson grade III open fractures, established infection at the fracture site, or inability to complete follow-up. Patients receiving therapeutic vitamin-D replacement before injury were also excluded to reduce exposure misclassification.

Clinical and biochemical assessment

Demographic characteristics, body mass index, smoking status, diabetes mellitus, mechanism of injury, fracture site, open-versus-closed injury, fixation technique, and relevant comorbidities were recorded prospectively on a standardized case-record form. Venous blood samples were collected within 72 hours of surgery. Serum 25(OH)D was quantified using a standardized chemiluminescent immunoassay. Serum calcium, phosphate, alkaline phosphatase, albumin, and creatinine were additionally documented.

Vitamin-D status was categorized a priori as deficient when 25(OH)D was <20 ng/mL, insufficient at 20-29.9 ng/mL, and sufficient at >=30 ng/mL. The baseline serum 25(OH)D concentration was additionally retained as a continuous variable for correlation and regression analyses.

Outcome assessment

Clinical and radiographic assessments were performed during standardized postoperative follow-up. Orthogonal radiographs were reviewed for cortical bridging, progressive callus formation, disappearance of the fracture line, implant integrity, and loss of reduction. Radiographic union was considered present when bridging callus was demonstrated across at least three of four visualized cortices together with clinical absence of pathological movement and substantial resolution of fracture-site pain during functional loading.

The primary outcome was non-union. Non-union was defined as failure to achieve radiographic and clinical union by 9 months, particularly when serial imaging showed no meaningful progression of healing over the preceding 3 months. Time to union was recorded in weeks among patients who achieved union. Two orthopaedic surgeons who were not provided the biochemical results independently assessed equivocal radiographs, and disagreements were resolved by consensus.

Statistical analysis

Continuous variables were summarized as mean +/- standard deviation or median with interquartile range according to distribution. Categorical variables were expressed as frequencies and percentages. Student's t test or the Mann-Whitney U test was used for continuous comparisons, whereas the chi-square test or Fisher's exact test was used for categorical comparisons. Spearman rank correlation was used to assess the relationship between baseline 25(OH)D and time to radiographic union.

Variables considered clinically relevant or demonstrating $p<0.10$ on univariable analysis were entered into multivariable binary logistic regression. Adjusted odds ratios (aORs) with 95% confidence intervals were reported. Multicollinearity was assessed before final modeling. A two-sided $p<0.05$ was considered statistically significant.

RESULTS

A total of 214 patients were assessed for eligibility; 34 were excluded because of predefined exclusion criteria, incomplete baseline biochemistry, or failure to complete minimum follow-up, leaving 180 patients for analysis. Mean age was 41.7 +/- 14.2 years, and 126 (70.0%) participants were male. Tibial fractures constituted the largest anatomical group (46.1%), followed by femoral (28.9%), humeral (15.0%), and forearm fractures (10.0%). Fifty patients (27.8%) were current smokers, 16 (8.9%) had diabetes, and 37 (20.6%) had grade I-II open injuries.

Baseline serum 25(OH)D averaged 21.3 +/- 9.4 ng/mL. Vitamin-D deficiency was identified in 104 patients (57.8%), insufficiency in 43 (23.9%), and sufficiency in only 33 (18.3%). Thirty-two patients developed non-union, yielding an overall non-union frequency of 17.8%. Mean serum 25(OH)D was substantially lower in the non-union group than in patients achieving union (14.6 +/- 6.5 vs. 22.8 +/- 9.4 ng/mL; $p<0.001$).

Non-union occurred in 26/104 vitamin-D-deficient patients (25.0%), compared with 4/43 patients with insufficiency (9.3%) and 2/33 vitamin-D-sufficient patients (6.1%; p=0.012 across categories). Among the 148 fractures that united, lower baseline 25(OH)D was associated with a longer healing interval (Spearman rho=-0.59; p<0.001). Multivariable analysis demonstrated that vitamin-D deficiency remained independently associated with non-union after adjustment for smoking, open fracture, diabetes, age, and fracture site (aOR 3.36, 95% CI 1.25-9.01; p=0.016).

Table 1. Baseline characteristics according to fracture-healing outcome

Variable	Union (n=148)	Non-union (n=32)	p value
Age, years	41.2 +/- 14.3	43.8 +/- 13.7	0.35
Male sex	101 (68.2%)	25 (78.1%)	0.27
BMI, kg/m2	25.1 +/- 3.8	26.0 +/- 4.2	0.23
Current smoking	35 (23.6%)	15 (46.9%)	0.008
Diabetes mellitus	10 (6.8%)	6 (18.8%)	0.043
Open fracture	25 (16.9%)	12 (37.5%)	0.010
Tibial fracture	65 (43.9%)	18 (56.3%)	0.20
Serum 25(OH)D, ng/mL	22.8 +/- 9.4	14.6 +/- 6.5	<0.001

Patients who developed non-union had substantially lower baseline vitamin-D concentrations and higher frequencies of smoking, diabetes, and open injury. Age, sex, body mass index, and fracture location showed no statistically significant differences. The marked separation in mean 25(OH)D between outcome groups suggested that hypovitaminosis D represented a potentially important biological correlate of impaired healing; however, the simultaneous enrichment of recognized clinical risk factors emphasized the need for multivariable adjustment before an independent relationship could be inferred.

Table 2. Vitamin-D category and fracture-healing outcomes

Vitamin-D status	Total, n	Non-union, n (%)	Union, n	Mean time to union, weeks*
Deficient (<20 ng/mL)	104	26 (25.0%)	78	23.4 +/- 5.1
Insufficient (20-29.9 ng/mL)	43	4 (9.3%)	39	20.4 +/- 4.2
Sufficient (>=30 ng/mL)	33	2 (6.1%)	31	18.7 +/- 3.8

A graded association was observed across vitamin-D categories. One quarter of deficient patients developed non-union, approximately four times the proportion observed in the vitamin-D-sufficient group. Among patients who achieved union, mean healing time also decreased progressively with increasing vitamin-D status. This dose-response pattern supported analysis of 25(OH)D both categorically and continuously and suggested that the observed relationship was not attributable solely to an arbitrary threshold separating deficiency from non-deficiency.

Table 3. Univariable associations with fracture non-union

Predictor	Unadjusted OR	95% CI	p value
Vitamin-D deficiency	3.89	1.51-10.00	0.005
Current smoking	2.85	1.30-6.23	0.009
Open fracture	2.95	1.28-6.79	0.011

Diabetes mellitus	3.19	1.07-9.52	0.038
Tibial location	1.64	0.76-3.54	0.21
Age, per 10-year increase	1.13	0.87-1.46	0.36

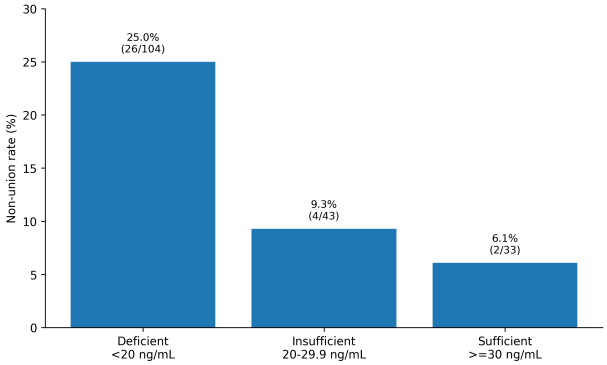
On unadjusted analysis, vitamin-D deficiency, smoking, open injury, and diabetes were each associated with increased odds of non-union. The magnitude of the vitamin-D association was comparable with other recognized biological and injury-related variables. Tibial location and increasing age were not significant predictors in this cohort. Because vitamin-D status may itself correlate with lifestyle, comorbidity, nutrition, and injury characteristics, these unadjusted estimates were subsequently examined within a multivariable model rather than interpreted as causal effects.

Table 4. Multivariable logistic regression for fracture non-union

Predictor	Adjusted OR	95% CI	p value
Vitamin-D deficiency (<20 ng/mL)	3.36	1.25-9.01	0.016
Current smoking	2.45	1.05-5.72	0.039
Open fracture	2.78	1.17-6.60	0.021
Diabetes mellitus	2.11	0.69-6.44	0.19
Tibial fracture	1.31	0.58-2.97	0.52
Age, per 10 years	1.08	0.82-1.43	0.58

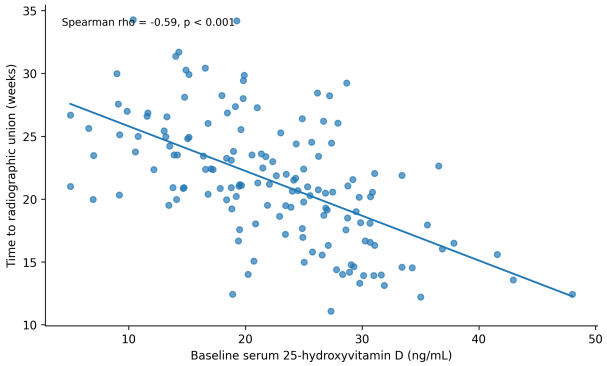
After simultaneous adjustment for relevant demographic, biological, and injury-related variables, vitamin-D deficiency remained associated with more than threefold greater odds of non-union. Smoking and open fracture also retained independent associations, whereas diabetes lost statistical significance, partly reflecting its limited prevalence and wide confidence interval. These findings suggested that low 25(OH)D provided information beyond several conventional risk factors, although the observational design precluded determination of whether correction of deficiency would directly reduce non-union.

FIGURE 1. NON-UNION RATE ACCORDING TO BASELINE VITAMIN-D STATUS.



The figure demonstrates a stepwise reduction in non-union frequency with improving baseline vitamin-D status. The largest absolute difference occurred between deficient and non-deficient patients, with non-union affecting 25.0% of patients below 20 ng/mL compared with fewer than 10% of patients in either higher category. Although the pattern is compatible with a biological gradient, the small number of events in the sufficient group requires cautious interpretation and independent validation in larger cohorts.

FIGURE 2. RELATIONSHIP BETWEEN BASELINE SERUM 25(OH)D AND TIME TO RADIOGRAPHIC UNION.



Among fractures that achieved union, increasing serum 25(OH)D was associated with progressively shorter radiographic healing time. The inverse correlation was moderately strong (Spearman rho=-0.59, p<0.001), indicating that vitamin-D status may relate not only to binary non-union but also to the tempo of otherwise successful healing. The dispersion of observations at comparable vitamin-D concentrations nevertheless illustrates the multifactorial nature of fracture repair and argues against using serum 25(OH)D as an isolated prognostic biomarker.

DISCUSSION

The principal finding of this study was an association between reduced serum 25(OH)D and impaired fracture healing. Vitamin-D-deficient patients demonstrated a higher frequency of non-union, while increasing 25(OH)D correlated with shorter time to radiographic union. Importantly, deficiency remained associated with non-union after adjustment for smoking, open injury, diabetes, age, and fracture location. These findings support the concept that systemic metabolic health contributes to fracture repair alongside mechanical stability and local vascular biology.

Our observations were consistent with Gorter et al., who reported more clinical delayed union among patients who remained vitamin-D deficient after supplementation [6]. Ravindra et al. similarly found that vitamin-D deficiency predicted non-union after spinal fusion and was associated with longer time to fusion [9]. Brinker et al. identified previously unrecognized metabolic or endocrine abnormalities in a large proportion of selected patients with unexplained or atypical non-unions [10], while Bergin et al. confirmed a substantial burden of metabolic disturbance in a broader non-union population [11]. Memon et al. more recently described an inverse relationship between baseline vitamin-D level and healing time in lower-limb fractures [13]. These studies support the view that metabolic status may modify the biological environment of repair.

The present findings must nevertheless be balanced against contradictory evidence. Haining et al. found no significant differences in 25(OH)D or other vitamin-D metabolites between patients with established non-union and matched controls [7]. Bodendorfer et al. reported no significant relationship between serum 25(OH)D and fracture-healing complications in supplemented orthopaedic-trauma patients [8]. Gudeman et al. documented frequent deficiency but did not establish that a standardized replacement approach improved union outcomes [12]. Haines et al., in a prospective randomized trial of a single early high-dose vitamin-D supplement in hypovitaminosis-D patients with long-bone fractures, did not demonstrate a significant improvement in fracture union [16]. These negative studies argue against interpreting low vitamin D as an isolated causal mechanism.

Interventional evidence remains similarly cautious. Slobogean et al. randomized patients with tibial or femoral shaft fractures to different vitamin-D3 regimens or placebo; although some signals suggested possible benefit with higher doses, definitive improvement in clinical or radiographic healing was not established [14]. Gatt et al. concluded in a systematic review that vitamin-D supplementation alone had not consistently improved radiographic union, functional outcomes, or healing complications [15]. Earlier systematic work by Gorter et al. also highlighted heterogeneous study designs and insufficient evidence for a direct therapeutic effect [3]. Conversely, the high prevalence of deficiency reported by Gorter et al., Schiffman et al., and other orthopaedic-trauma cohorts indicates that vitamin-D status remains clinically relevant as part of broader skeletal-health assessment [4,5,17].

Several mechanisms could plausibly explain the observed association. Vitamin D regulates intestinal calcium and phosphate absorption and contributes to mineralization of the hard callus. Vitamin-D receptor signaling also influences osteoblast differentiation, osteoclast activity, inflammatory signaling, and remodeling [1-3]. Persistent deficiency could therefore produce a less favorable biochemical environment during mineralization, particularly in patients already exposed to smoking, soft-tissue damage, infection risk, or metabolic disease. The clinical implication is not that vitamin D should replace established principles of non-union prevention, but that metabolic assessment may complement evaluation of fixation stability, alignment, infection, soft-tissue viability, smoking, and systemic illness.

Several limitations should be acknowledged. The observational design established association rather than causation. A single baseline 25(OH)D measurement may not reflect vitamin-D status throughout healing, and residual confounding from nutrition, seasonal variation, physical activity, fixation characteristics, and adherence to rehabilitation may remain. Inclusion of several anatomical sites introduced biological heterogeneity, while the number of non-union events limited model complexity. Larger multicenter cohorts with serial biochemical measurements and adequately powered randomized trials targeting deficient patients are required to determine whether correction of vitamin-D deficiency can modify non-union risk.

CONCLUSION

Lower baseline serum 25-hydroxyvitamin D was associated with increased fracture non-union and prolonged time to radiographic union. The relationship persisted after adjustment for important clinical and injury-related factors, suggesting that vitamin-D deficiency may represent a relevant component of the systemic biological environment influencing fracture repair. Vitamin-D assessment may therefore have value as part of broader metabolic evaluation in patients at risk of impaired healing. Nevertheless, association does not establish therapeutic efficacy, and adequately powered randomized trials are required before vitamin-D supplementation can be recommended specifically as an intervention to prevent fracture non-union.

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