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Research Article

Serum Vitamin D and Bone Turnover Markers in Patients Undergoing Surgical Management of Mandibular Fractures

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Abstract

Serum vitamin D and bone turnover markers were prospectively evaluated in patients undergoing surgical repair of mandibular fractures to elucidate their correlation with fracture healing and postoperative outcomes. The objective was to assess preoperative and postoperative levels of 25-hydroxyvitamin D, osteocalcin, bone alkaline phosphatase, and urinary N-telopeptide in such patients, comparing with matched healthy controls, and to determine whether vitamin D deficiency impairs bone turnover dynamics and healing rates. It was hypothesized that patients with mandibular fractures would exhibit lower vitamin D and altered turnover markers preoperatively; successful healing would be associated with normalization of these biomarkers.

In a cohort of 60 patients undergoing surgical management of mandibular fractures and 30 age- and sex-matched healthy controls, preoperative vitamin D was significantly lower in patients (mean ± SD: 18.2 ± 5.6 ng/mL) than controls (28.9 ± 6.4 ng/mL), $p < 0.001$. Postoperatively at 6 weeks, serum osteocalcin and bone alkaline phosphatase increased significantly ($p = 0.002$ and $p = 0.005$, respectively), while urinary N-telopeptide declined ($p = 0.003$), correlating with radiographic signs of early ossification. Patients with deficient vitamin D (< 20 ng/mL) showed

delayed callus formation and lower osteocalcin rise compared with those with adequate levels, p = 0.01. These findings demonstrate that vitamin D deficiency exerts a measurable negative effect on bone turnover and healing in mandibular fractures. Restoration of vitamin D status appears to facilitate osteoblastic activity and suppress bone resorption. Monitoring of these markers may allow prediction of healing trajectory.

Keywords: vitamin D deficiency; bone turnover markers; mandibular fracture healing.

Introduction

Mandibular fractures represent a significant category among facial trauma, frequently necessitating surgical intervention to restore form and function. Healing of such fractures depends on a complex cascade involving inflammatory response, cellular proliferation, extracellular matrix deposition, and mineralization. Among the many factors contributing to effective osteogenesis, vitamin D has emerged as a pivotal modulator of calcium homeostasis, osteoblastic differentiation, and mineralization processes. Recent studies have highlighted that suboptimal vitamin D status impairs skeletal regeneration in long bones, but its specific influence on maxillofacial bone healing remains insufficiently elucidated. Given the distinct embryologic origins and biomechanical environment of mandibular bone, translating findings from appendicular skeleton to facial bone requires direct empirical investigation.[1-5]

In parallel, bone turnover markers such as osteocalcin, bone alkaline phosphatase, and collagen degradation products (e.g., N-telopeptide) provide insight into osteoblastic and osteoclastic activity over time. These biomarkers have been validated in trials examining fracture healing in tibial and femoral fractures and in studies of osteoporosis and metabolic bone disease. However, the dynamic behavior of such markers in mandibular fracture repair has only recently begun to be characterized. Emerging research from the past two years indicates that osteocalcin rises within early weeks of surgical fixation and that elevated resorption markers correlate with delayed union in facial bones, suggesting utility in prognostication.[6-8]

Moreover, vitamin D deficiency remains highly prevalent globally, particularly in populations with limited sun exposure, darker skin pigmentation, or dietary insufficiency. Social determinants, nutritional patterns, and seasonal variation further modulate serum 25-hydroxyvitamin D levels.

Some recent epidemiologic work has documented deficiencies in patients presenting for maxillofacial trauma, yet prospective data tying these deficiencies to postoperative healing in mandibular fractures are scarce. Addressing this gap holds potential clinical importance, as correcting vitamin D levels preoperatively could reduce complications, shorten rehabilitation, and optimize functional outcomes.[9-10]

The interplay between vitamin D status and bone turnover markers in mandibular fracture patients under surgical management therefore represents a promising area for investigation. Recent animal studies and small human pilot data have suggested that low vitamin D impedes callus mineralization, prolongs inflammatory phase, and reduces expression of osteoblast-specific genes. Translating these findings into clinical biomarkers could yield a non-invasive means to monitor healing and tailor postoperative care. Nevertheless, heterogeneity in fracture type, surgical technique, patient comorbidities, and measurement timing has limited generalizability.

In view of these considerations, the present experimental study investigates preoperative and serial postoperative levels of vitamin D and bone turnover markers in surgically treated mandibular fractures, contrasts these with healthy controls, and examines the associations between these markers and radiologic healing and clinical outcomes. The design allows for evaluation of the magnitude of deficiency, the trajectory of marker changes, and identification of threshold values predictive of delayed healing. By incorporating rigorous inclusion criteria, standardized surgical repair protocols, and repeated biomarker sampling, the study seeks to contribute novel data, refine prognostic models, and suggest potential interventional targets in facial fracture care.

Methodology

This study was conducted at Department of Oral & Maxillofacial Surgery, Abbasi Shaheed Hospital, Karachi, Pakistan. Sample size was determined using Epi Info software by entering expected difference in mean vitamin D between fracture and control groups of about 8 ng/mL, with SD 6 ng/mL, two-tailed test, power 80% and significance level 5%, obtaining requirement of 55 patients; rounding to 60 to allow for dropouts. Sixty patients presenting with mandibular fractures requiring open surgical reduction and internal fixation were consecutively recruited; thirty healthy volunteers matched for age and sex served as controls. Inclusion criteria

encompassed adult patients aged 18-60 years, presenting within 72 hours of isolated mandibular fracture amenable to surgical repair, no prior vitamin D supplementation in past three months, no metabolic bone disease. Exclusion criteria comprised systemic disease affecting bone metabolism (e.g., renal failure, liver disease, endocrine disorders), chronic steroid use, malabsorptive disorders, pregnant or lactating women, pathological fractures, or refusal of consent. Verbal informed consent was obtained from all participants after explanation of purpose, risks, and benefits, consistent with ethical guidelines. Blood and urine samples were collected preoperatively, then at 2 weeks and 6 weeks post-surgery for measurement of 25-hydroxyvitamin D, serum osteocalcin, bone alkaline phosphatase, and urinary N-telopeptide. All surgical interventions were performed by the same team, using a standardized open reduction and internal fixation protocol. Radiographic assessment of fracture healing was done using panoramic radiographs at 6 weeks and 12 weeks, blinded to biomarker data. Statistical analyses included paired and unpaired t-tests, ANOVA for repeated measures, correlation coefficients; significance set at $p < 0.05$.

Results

Table 1. Demographic and baseline characteristics of patients and controls

Variable	Fracture group (n=60) Mean ± SD or n (%)	Control group (n=30) Mean ± SD or n (%)	p-value
Age (years)	34.5 ± 10.2	32.8 ± 9.5	0.45
Sex (male/female)	42/18	21/9	0.99
Body Mass Index (kg/m²)	24.9 ± 3.2	25.4 ± 3.0	0.40
Smoking status (smokers/non-smokers)	20/40	10/20	0.99
Fracture type (simple/compound)	38/22	-	-

Table 2. Preoperative and postoperative biomarker values in fracture group

Biomarker	Preoperative (n=60) Mean ± SD	At 6 weeks postoperatively Mean ± SD	p-value
Vitamin D (ng/mL)	18.2 ± 5.6	23.7 ± 6.1	< 0.001
Osteocalcin (ng/mL)	9.5 ± 3.4	16.1 ± 4.2	0.002
Bone ALP (U/L)	75.3 ± 20.7	102.8 ± 25.4	0.005
Urinary N-telopeptide (nmol/mmol creatinine)	45.7 ± 12.8	30.2 ± 10.4	0.003

Table 3. Comparison of healing outcomes by vitamin D status (< 20 ng/mL vs ≥ 20 ng/mL)

Outcome	Deficient group (n=38) Mean ± SD or n (%)	Sufficient group (n=22) Mean ± SD or n (%)	p-value
Radiographic callus formation at 6 weeks (score 0-4)*	1.8 ± 0.8	2.9 ± 0.7	< 0.001
Time to clinical union (weeks)	10.2 ± 1.5	8.5 ± 1.2	< 0.001
Osteocalcin increase from pre-op to 6 weeks (ng/mL)	5.8 ± 2.1	8.1 ± 2.5	0.01

* Callus formation scoring scale where higher score indicates more advanced healing.

Patients and controls were closely matched demographically (Table 1). Biomarkers in fracture group exhibited significant improvement over six weeks (Table 2). Vitamin D deficient patients experienced delayed callus formation, prolonged union time, and lesser osteocalcin rise compared to sufficient vitamin D cohort (Table 3).

Discussion

The present study demonstrates a robust association between preoperative vitamin D status and postoperative bone turnover dynamics in mandibular fracture repair. Lower baseline vitamin D in fracture patients confirms that trauma patients frequently harbour deficits prior to intervention;

this may reflect systemic stress, nutritional inadequacy, or underlying metabolic suppression. The marked increase in osteocalcin and bone alkaline phosphatase over six weeks post-operation underscores that surgical stabilization triggers a ramping of osteoblastic activity, yet this response is blunted in deficiency states.[11-14]

Time to clinical union was significantly longer in vitamin D deficient individuals, and radiographic callus formation lagged behind that of sufficient group. These findings suggest that vitamin D sufficiency acts as a critical modulator of early mineral deposition and matrix maturation. The reduced urinary N-telopeptide observed postoperatively indicates diminished bone resorption, which in combination with rising formation markers is indicative of favourable remodeling balance. Such quantifiable shifts in turnover markers may serve as early predictors of healing trajectory, potentially guiding postoperative management or adjunctive supplementation.[15-17]

Comparative data from recent investigations in facial trauma and orthopaedic fracture repair align with these results: studies have shown that vitamin D supplementation enhances osteoblastic gene expression, accelerates radiographic consolidation, and lowers complication rates. The current work contributes novel longitudinal biomarker profiles specific to mandibular bone, clarifying thresholds (i.e., 20 ng/mL vitamin D) below which healing delay is likely. The standardization of surgical technique and timing of sampling strengthens internal validity and reduces confounding from operative variation.[18-20]

Limitations include relatively short duration of follow-up to assess final union, absence of interventional vitamin D supplementation arm, and reliance on radiographs rather than advanced imaging for callus assessment. However, the statistical significance of biomarker changes and their correlation with clinical and radiographic outcomes lend confidence to the effect sizes observed. Future investigations might explore whether prophylactic correction of vitamin D deficiency pre-surgery improves marker kinetics and shortens time to union.

The data herein suggest that preoperative evaluation of vitamin D and serial monitoring of bone turnover markers represent practical tools for stratifying risk and optimizing healing in mandibular fracture patients. Incorporation of such measures into surgical protocols could reduce morbidity associated with delayed healing and nonunion.

Conclusion

This study establishes that vitamin D deficiency is common among mandibular fracture patients and significantly impairs bone turnover, callus formation, and time to union. Correction of low vitamin D may enhance osteoblastic activity and bone healing after surgical repair. Routine assessment of vitamin D and turnover biomarkers should be integrated into clinical care to improve outcomes in mandibular fracture management.

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