

CBCT-Based Assessment of Jaw Bone Alterations in Patients with Chronic Kidney Disease and Mineral Bone Disorder

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ABSTRACT

Background: Chronic kidney disease–mineral and bone disorder (CKD-MBD) causes abnormalities in mineral metabolism and bone turnover that may also affect the jaws. CBCT provides three-dimensional assessment of mandibular cortical and trabecular changes. **Aim:** To assess CBCT-based jaw bone alterations in patients with CKD-MBD and compare radiomorphometric and qualitative findings with individuals without CKD-MBD. **Methods:** This comparative observational study included 120 adults, comprising 80 patients with CKD-MBD and 40 age- and sex-matched controls. CBCT examinations were assessed for mandibular cortical measurements, including the mental index, antegonial index, panoramic mandibular index, and cortical thickness. Qualitative changes in cortical morphology, trabecular architecture, lamina dura, focal radiolucencies, and soft-tissue/vascular calcifications were also evaluated. Clinical and biochemical parameters, including calcium, phosphate, alkaline phosphatase, PTH, and eGFR, were recorded. **Results:** Patients with CKD-MBD demonstrated significantly lower mandibular cortical measurements than controls ($P < 0.001$). Cortical porosity, lamina dura attenuation/loss, altered trabecular architecture, reduced osseous density, focal radiolucent changes, and soft-tissue/vascular calcifications were significantly more frequent in the CKD-MBD group. Several CBCT abnormalities increased with advancing CKD stage. Mandibular cortical measurements showed significant inverse correlations with PTH and alkaline phosphatase and positive correlations with eGFR. **Conclusion:** CKD-MBD is associated with significant quantitative and qualitative alterations of mandibular bone detectable on CBCT. Systematic assessment of these changes may provide useful adjunctive information for dental evaluation and treatment planning. However, CBCT findings should be interpreted alongside clinical and biochemical assessment.

Keywords: Chronic kidney disease; CKD-MBD; cone-beam computed tomography; mandibular bone; radiomorphometry; PTH; trabecular bone.

Key Message

CKD-MBD is associated with measurable alterations in mandibular cortical and trabecular bone that can be demonstrated using CBCT. Quantitative radiomorphometric assessment combined with evaluation of cortical morphology, trabecular architecture, lamina dura integrity, and calcific changes may provide useful adjunctive information for dental assessment and treatment planning in affected patients.

INTRODUCTION

Chronic kidney disease (CKD) is associated with progressive disturbances in mineral metabolism and

skeletal homeostasis. Impaired phosphate excretion, altered vitamin D metabolism, calcium imbalance, and secondary hyperparathyroidism contribute to chronic kidney disease–mineral and bone disorder (CKD-MBD), which encompasses abnormalities of mineral metabolism, bone turnover and mineralization, and vascular or soft-tissue calcification [1–3]. Although these abnormalities primarily affect the systemic skeleton, the jaws may also demonstrate clinically relevant structural changes.

The maxillofacial manifestations of CKD-MBD include reduction or loss of the lamina dura, mandibular cortical thinning or erosion, altered

trabecular architecture, reduced osseous density, and, in severe secondary hyperparathyroidism, focal radiolucent lesions associated with excessive bone turnover [4–7]. These changes are particularly relevant in dentistry because jawbone quality may influence assessment and planning of implant placement, tooth extraction, periodontal surgery, and other invasive dentoalveolar procedures.

Panoramic radiography has traditionally been used to evaluate mandibular bone changes through radiomorphometric parameters such as the mandibular cortical index (MCI), mental index (MI), antegonial index (AI), and panoramic mandibular index (PMI) [8,9]. However, two-dimensional imaging is affected by magnification, geometric distortion, and anatomical superimposition. Cone-beam computed tomography (CBCT) provides multiplanar and three-dimensional visualization of the maxillofacial skeleton and permits more detailed assessment of cortical thickness, cortical morphology, trabecular architecture, and other osseous abnormalities. CBCT-based studies have demonstrated alterations in mandibular radiomorphometric parameters and trabecular organization in patients with chronic renal disease [10–15].

The relationship between mandibular radiological findings and systemic CKD-MBD parameters is also of dental and diagnostic interest. Previous research has demonstrated associations between mandibular radiomorphometric indices and biochemical markers, particularly PTH, calcium, phosphate, and alkaline phosphatase, in patients with secondary hyperparathyroidism associated with CKD [16–18]. Such associations suggest that jawbone findings may provide an anatomical correlate of systemic skeletal involvement, although CBCT cannot replace biochemical or nephrological assessment.

Recognition of CKD-MBD-related jaw alterations is therefore important for oral and maxillofacial radiologists and dental clinicians. A systematic CBCT assessment may identify cortical and trabecular abnormalities that are relevant to dental treatment planning and may also reveal incidental calcific changes within the scanned field [19,20].

However, available studies differ in their populations, CKD severity, dialysis status, imaging methodology, and radiomorphometric parameters, and relatively few studies have comprehensively evaluated both quantitative and qualitative jaw alterations in relation to CKD-MBD severity.

Therefore, it is of interest to assess CBCT-based jaw bone alterations in patients with CKD-MBD compared with individuals without CKD and to determine their relationship with CKD severity and relevant biochemical markers of mineral and bone metabolism.

MATERIALS AND METHODS

Study design and setting

A comparative observational study was conducted in the Department of Dental Surgery in collaboration with the Department of Nephrology at Kakatiya Medical

College and Hospital, Warangal, India. The study was conducted over 12 months, from April 2025 to March 2026. The study was designed to compare CBCT-based jaw bone characteristics between patients with established chronic kidney disease–mineral and bone disorder (CKD-MBD) and individuals without CKD or known metabolic bone disease.

Study population and sample size

A total of 120 participants were included, comprising 80 patients with CKD-MBD (study group) and 40 age- and sex-matched individuals without CKD-MBD (control group).

A sample size of 80 patients in the CKD-MBD group was considered appropriate to detect clinically relevant differences in mandibular cortical measurements between patients with CKD-MBD and controls, while the 40-member control group provided an adequate comparative reference for the principal CBCT parameters. The final sample size also allowed evaluation of associations between radiomorphometric findings and biochemical indicators of CKD-MBD.

Study group

Patients were recruited from the nephrology service and referred for dental/maxillofacial evaluation. CKD-MBD was defined on the basis of established CKD together with documented abnormalities of mineral and bone metabolism managed as part of routine nephrological care.

Patients with different stages of CKD were eligible, allowing evaluation of the relationship between renal disease severity and jawbone alterations. Dialysis status and duration of dialysis were recorded for patients receiving renal replacement therapy.

Control group

The control group consisted of individuals attending the dental service who had no known history of CKD, renal replacement therapy, metabolic bone disease, or systemic condition known to substantially affect bone metabolism. Controls were frequency-matched to the study group according to age and sex.

Inclusion criteria

Participants were eligible if they:

1. Were aged ≥ 18 years.
2. Had a clinically established diagnosis of CKD for inclusion in the study group.
3. Had documented CKD-MBD-related mineral or bone abnormalities managed by the treating nephrologist.
4. Had a CBCT examination of the jaws obtained for a clinically indicated dental/maxillofacial purpose.
5. Had adequate image quality for assessment of the predefined radiomorphometric parameters.
6. Were willing to participate and provide informed consent.

For the control group, participants were required to have no known CKD, CKD-MBD, metabolic bone disease, or other systemic disorder substantially affecting skeletal metabolism.

Exclusion criteria

Patients were excluded if they had a history of primary metabolic bone disease unrelated to CKD, previous major mandibular surgery or fracture affecting the assessment region, known primary hyperparathyroidism, active malignancy involving the jaws, severe maxillofacial developmental abnormality, or CBCT images with substantial artefacts that prevented reliable assessment.

Patients receiving medications or having systemic conditions likely to produce major independent alterations in jawbone metabolism were excluded where these could not be appropriately accounted for.

Clinical and biochemical assessment

For patients in the CKD-MBD group, relevant demographic and clinical information was recorded, including age, sex, CKD stage, duration of CKD, dialysis status, duration of dialysis where applicable, and relevant medical comorbidities.

The most recent clinically available biochemical measurements obtained within the predefined assessment period were recorded. These included:

- Serum calcium
- Serum phosphate
- Serum alkaline phosphatase
- Intact parathyroid hormone (PTH)
- Serum creatinine
- Estimated glomerular filtration rate (eGFR)

These parameters were used to characterize the systemic mineral and bone profile and to examine their relationship with CBCT findings.

CBCT examination

CBCT examinations were evaluated using standardized multiplanar reconstructions. The assessment focused primarily on the mandible because mandibular cortical and trabecular structures provide reproducible anatomical landmarks for radiomorphometric evaluation.

Images were assessed in axial, sagittal, and coronal planes, with cross-sectional reconstructions used where necessary to identify and measure cortical and trabecular alterations. All examinations included in the analysis were required to demonstrate adequate visualization of the mandibular regions selected for measurement.

Quantitative CBCT assessment

The principal quantitative radiomorphometric parameters were:

Mental index (MI): Mandibular cortical thickness was measured in the region of the mental foramen on cross-sectional CBCT images, using a standardized

perpendicular measurement of the inferior mandibular cortical border.

Antegonial index (AI): Cortical thickness was assessed in the antegonial region using a standardized measurement perpendicular to the mandibular inferior border.

Panoramic mandibular index (PMI): The ratio between mandibular cortical thickness and the corresponding distance from the inferior cortical border to the inferior margin of the mental foramen was calculated where the anatomical landmarks permitted reliable assessment.

Mandibular cortical index (MCI): The mandibular cortex was classified according to its morphological appearance, ranging from relatively normal continuous cortex to varying degrees of cortical erosion or porosity.

Qualitative CBCT assessment

The following jawbone alterations were systematically recorded:

- Cortical thinning or irregularity
- Cortical porosity
- Altered mandibular cortical morphology
- Loss or attenuation of the lamina dura
- Altered trabecular architecture
- Generalized reduction in apparent osseous density
- Focal radiolucent osseous alterations
- Soft-tissue or vascular calcifications within the scanned maxillofacial field

The presence or absence of each finding was documented using predefined assessment criteria.

Image assessment and reliability

CBCT images were independently evaluated by two trained oral and maxillofacial radiologists who were blinded to the biochemical results at the time of radiological assessment. Quantitative measurements were recorded independently and compared for agreement. In cases of disagreement regarding qualitative findings, consensus was reached after joint review.

Intraobserver reliability was assessed by repeat evaluation of a randomly selected subset of CBCT examinations after an interval sufficient to minimize recall. Agreement for categorical variables was assessed using Cohen's kappa, while intraclass correlation coefficients were used for continuous radiomorphometric measurements.

Study outcomes

The **primary outcome** was the difference in mandibular cortical measurements between patients with CKD-MBD and controls.

The **secondary outcomes** included:

- Distribution of mandibular cortical morphology according to MCI.
- Frequency of lamina dura alterations.

- Frequency of trabecular bone abnormalities.
- Frequency of focal radiolucent changes.
- Frequency of soft-tissue/vascular calcifications.
- Correlation between CBCT radiomorphometric measurements and PTH, calcium, phosphate, alkaline phosphatase, and eGFR.
- Relationship between CBCT findings and CKD severity/dialysis status.

Statistical Analysis

Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as frequencies and percentages. Differences in continuous CBCT measurements between the CKD-MBD and control groups were assessed using the independent *t* test for normally distributed data or the Mann–Whitney *U* test where appropriate.

Categorical CBCT findings were compared using the chi-square test or Fisher's exact test. Correlations between radiomorphometric measurements and biochemical variables were evaluated using Pearson or Spearman correlation analysis according to data distribution.

Multivariable regression analysis was used where appropriate to identify independent associations between selected CBCT abnormalities and clinical/biochemical variables after adjustment for relevant demographic factors. Interobserver and intraobserver reliability were assessed using intraclass correlation coefficients for continuous measurements and Cohen's kappa for categorical assessments.

All statistical tests were two-sided, and a *P* value <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki and applicable institutional ethical standards. Institutional Ethics Committee approval was obtained before initiation of the study. Written informed consent was obtained from all participants before inclusion. CBCT examinations were performed only when clinically indicated, and no additional radiation exposure was undertaken solely for research purposes. Participant confidentiality was maintained throughout data collection, analysis, and reporting.

RESULTS

A total of 120 participants were included in the final analysis, comprising 80 patients with CKD-MBD and 40 age- and sex-matched controls. The mean age of the CKD-MBD group was higher than that of the control group, although the difference was not statistically significant. Most participants in both groups were male. Among patients with CKD-MBD, stage 4 and stage 5 disease constituted the predominant categories, and more than half were receiving maintenance dialysis. Biochemical assessment demonstrated abnormalities of calcium-phosphate metabolism, elevated alkaline phosphatase, and markedly increased intact PTH concentrations in the CKD-MBD group. CBCT assessment demonstrated significantly reduced mandibular cortical measurements in patients with CKD-MBD compared with controls. Abnormal mandibular cortical morphology, lamina dura attenuation or loss, and trabecular alterations were also substantially more frequent among patients with CKD-MBD. Soft-tissue or vascular calcifications within the scanned field were observed predominantly in the CKD-MBD group. Greater severity of selected CBCT abnormalities was associated with higher PTH and alkaline phosphatase levels and lower eGFR.

Table 1: Demographic and clinical characteristics of study participants

The demographic and baseline clinical characteristics of the 120 participants are presented below.

Characteristic	CKD-MBD group (n=80)	Control group (n=40)	<i>P</i> value
Age (years), mean $\pm$ SD	55.8 $\pm$ 10.7	53.2 $\pm$ 9.8	0.190
Age ≥ 60 years, n (%)	29 (36.3)	12 (30.0)	0.501
Male sex, n (%)	48 (60.0)	23 (57.5)	0.795
Female sex, n (%)	32 (40.0)	17 (42.5)	0.795
Duration of CKD (years), mean $\pm$ SD	6.2 $\pm$ 3.1	—	—
Stage 3 CKD, n (%)	10 (12.5)	—	—
Stage 4 CKD, n (%)	25 (31.3)	—	—
Stage 5 CKD, n (%)	45 (56.3)	—	—
Receiving maintenance dialysis, n (%)	47 (58.8)	—	—
Duration of dialysis (years), mean $\pm$ SD	3.4 $\pm$ 1.9	—	—
Diabetes mellitus, n (%)	39 (48.8)	9 (22.5)	0.005
Hypertension, n (%)	58 (72.5)	15 (37.5)	<0.001

The two groups were broadly comparable with respect to age and sex. CKD-MBD patients predominantly had advanced renal disease, with 56.3% having stage 5 CKD and 58.8% receiving maintenance dialysis. Diabetes and hypertension were significantly more frequent among patients with CKD-MBD.

Table 2: Biochemical characteristics of patients with CKD-MBD and controls

The biochemical profile demonstrated substantial differences between participants with CKD-MBD and controls, particularly in parameters reflecting mineral and bone metabolism.

Biochemical parameter	CKD-MBD group (n=80)	Control group (n=40)	P value
Serum calcium (mg/dL), mean ± SD	8.42 ± 0.71	9.18 ± 0.48	<0.001
Serum phosphate (mg/dL), mean ± SD	5.12 ± 1.21	3.54 ± 0.62	<0.001
Alkaline phosphatase (IU/L), mean ± SD	188.6 ± 82.4	92.7 ± 24.6	<0.001
Intact PTH (pg/mL), median (IQR)	468 (286–782)	54 (38–72)	<0.001
Serum creatinine (mg/dL), mean ± SD	5.84 ± 2.16	0.89 ± 0.18	<0.001
eGFR (mL/min/1.73 m ²), mean ± SD	19.7 ± 12.4	94.8 ± 16.2	<0.001

Patients with CKD-MBD had significantly lower serum calcium and significantly higher phosphate, alkaline phosphatase, PTH, and creatinine concentrations than controls. Mean eGFR was markedly reduced in the CKD-MBD group, consistent with the underlying renal impairment.

Table 3: Comparison of quantitative mandibular CBCT measurements

Quantitative CBCT assessment demonstrated significantly reduced mandibular cortical dimensions among patients with CKD-MBD.

CBCT parameter	CKD-MBD group (n=80)	Control group (n=40)	P value
Mental index (mm), mean ± SD	2.61 ± 0.54	3.42 ± 0.49	<0.001
Antegonial index (mm), mean ± SD	2.54 ± 0.51	3.18 ± 0.46	<0.001
Panoramic mandibular index, mean ± SD	0.29 ± 0.07	0.38 ± 0.06	<0.001
Mean mandibular cortical thickness (mm)	2.67 ± 0.52	3.48 ± 0.47	<0.001

The mean mental index, antegonial index, panoramic mandibular index, and overall mandibular cortical thickness were significantly lower in the CKD-MBD group than in controls. The greatest relative difference was observed for the mental index.

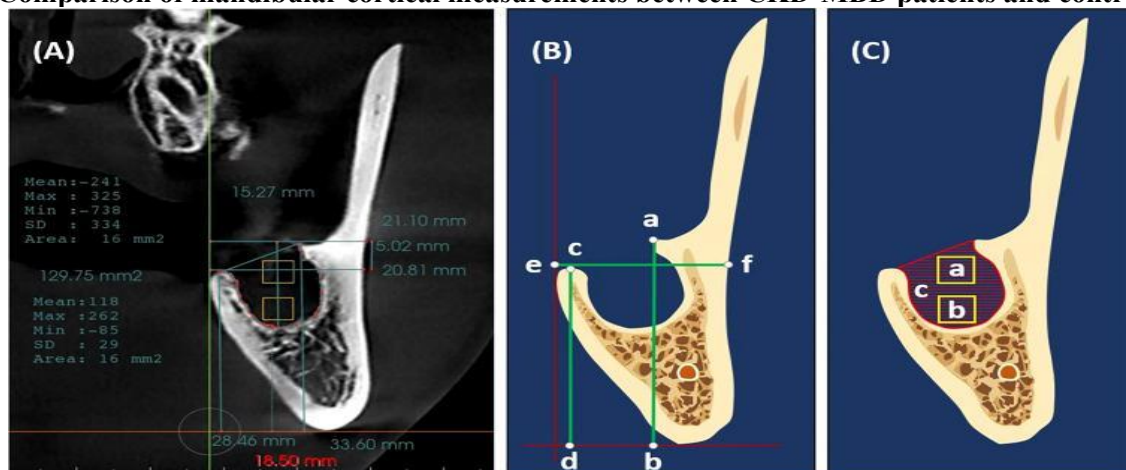
Table 4: Distribution of mandibular cortical morphology and qualitative CBCT alterations

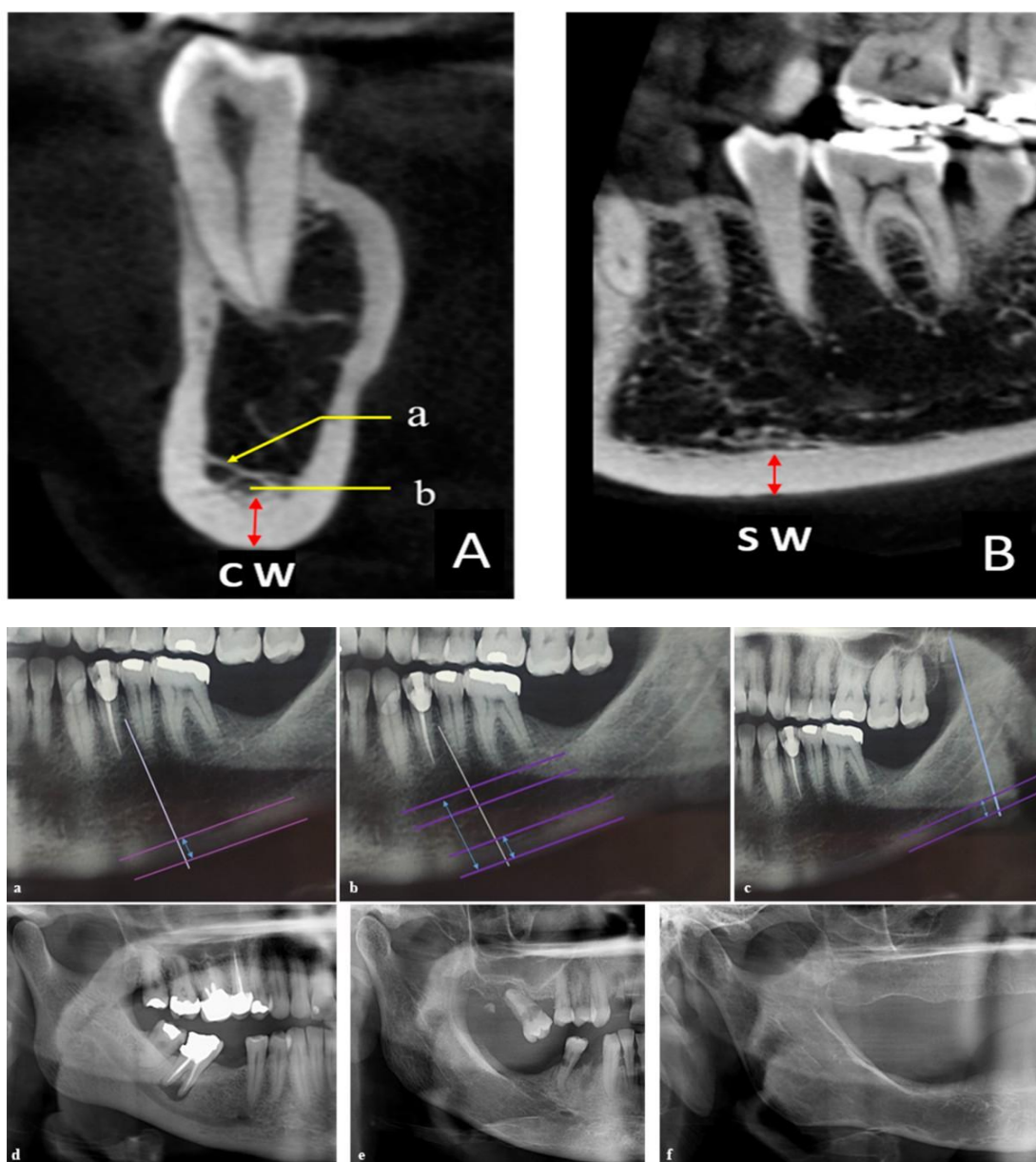
Qualitative assessment demonstrated a substantially greater burden of mandibular and dentoalveolar abnormalities in patients with CKD-MBD.

CBCT finding	CKD-MBD group (n=80)	Control group (n=40)	P value
Normal mandibular cortex, n (%)	23 (28.8)	30 (75.0)	<0.001
Mild cortical porosity/erosion, n (%)	31 (38.8)	8 (20.0)	0.036
Moderate-to-severe cortical porosity/erosion, n (%)	26 (32.5)	2 (5.0)	<0.001
Lamina dura attenuation/loss, n (%)	39 (48.8)	6 (15.0)	<0.001
Altered trabecular architecture, n (%)	42 (52.5)	8 (20.0)	0.001
Generalized reduced osseous density, n (%)	29 (36.3)	4 (10.0)	0.003
Focal radiolucent osseous alteration, n (%)	11 (13.8)	1 (2.5)	0.047
Soft-tissue/vascular calcification, n (%)	24 (30.0)	3 (7.5)	0.006

Normal mandibular cortical morphology was significantly less frequent among patients with CKD-MBD. Moderate-to-severe cortical porosity or erosion, lamina dura attenuation or loss, trabecular alterations, reduced osseous density, focal radiolucent changes, and soft-tissue/vascular calcifications were all more frequently observed in the CKD-MBD group.

Figure 1: Comparison of mandibular cortical measurements between CKD-MBD patients and controls





The graphical comparison demonstrates consistently lower mandibular cortical measurements among patients with CKD-MBD across the evaluated radiomorphometric parameters.

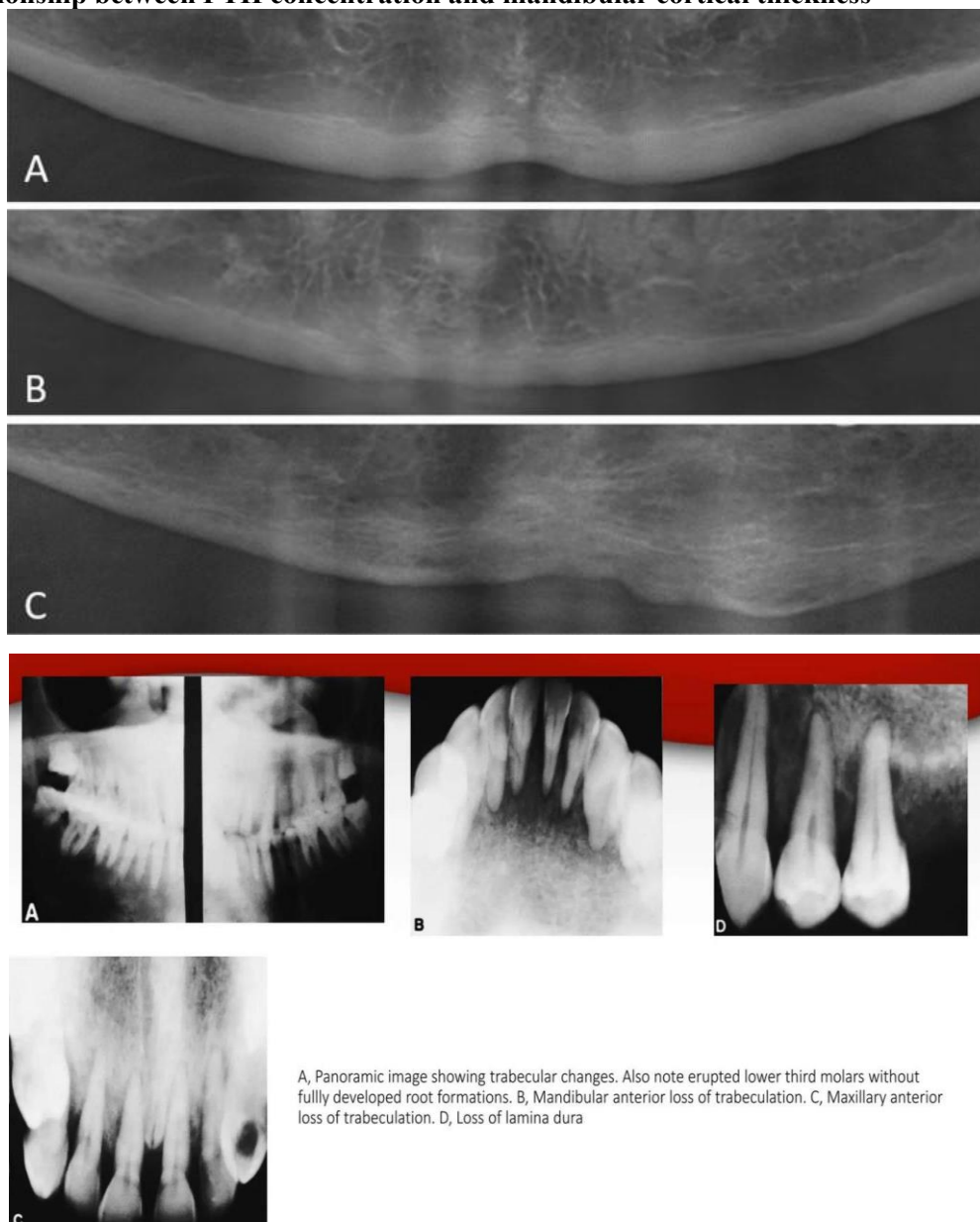
Table 5: Association between CKD severity and mandibular cortical alterations

The frequency of significant cortical abnormalities increased progressively with greater CKD severity.

CBCT finding	Stage 3 CKD (n=10)	Stage 4 CKD (n=25)	Stage 5 CKD (n=45)	P for trend
Moderate-to-severe cortical porosity/erosion, n (%)	1 (10.0)	7 (28.0)	18 (40.0)	0.031
Lamina dura attenuation/loss, n (%)	2 (20.0)	10 (40.0)	27 (60.0)	0.018
Altered trabecular architecture, n (%)	3 (30.0)	12 (48.0)	27 (60.0)	0.041
Reduced osseous density, n (%)	2 (20.0)	8 (32.0)	19 (42.2)	0.047
Soft-tissue/vascular calcification, n (%)	1 (10.0)	5 (20.0)	18 (40.0)	0.028

The prevalence of moderate-to-severe cortical porosity, lamina dura alteration, trabecular abnormality, reduced osseous density, and soft-tissue/vascular calcification increased progressively from stage 3 to stage 5 CKD. Significant trends were observed for all evaluated abnormalities.

Figure 2: Relationship between PTH concentration and mandibular cortical thickness



The analysis demonstrated an inverse relationship between intact PTH concentration and mandibular cortical thickness, with greater PTH elevation associated with lower cortical measurements.

Table 6: Correlation of CBCT radiomorphometric parameters with biochemical variables in CKD-MBD patients

Correlation analysis demonstrated significant associations between selected CBCT measurements and biochemical indicators of CKD-MBD.

CBCT parameter	PTH	Alkaline phosphatase	Phosphate	Calcium	eGFR
Mental index	-0.56 (<0.001)	-0.43 (<0.001)	-0.31 (0.005)	0.24 (0.032)	0.48 (<0.001)
Antegonial index	-0.49 (<0.001)	-0.38 (0.001)	-0.28 (0.012)	0.21 (0.061)	0.42 (<0.001)
Panoramic mandibular index	-0.45 (<0.001)	-0.34 (0.002)	-0.25 (0.025)	0.19 (0.091)	0.39 (<0.001)
Cortical thickness	-0.58 (<0.001)	-0.46 (<0.001)	-0.33 (0.003)	0.27 (0.016)	0.51 (<0.001)

Values are correlation coefficient (r) followed by P value.

Mandibular cortical measurements showed moderate inverse correlations with PTH and alkaline phosphatase and positive correlations with eGFR. The strongest association was observed between overall mandibular cortical thickness and PTH ($r = -0.58$, $P < 0.001$). Phosphate showed weaker but statistically significant inverse associations with the principal cortical measurements, whereas serum calcium demonstrated weak positive associations.

DISCUSSION

The present study evaluated CBCT-based jaw bone alterations in patients with chronic kidney disease–mineral and bone disorder (CKD-MBD) and demonstrated consistent changes in both quantitative and qualitative mandibular bone characteristics [4]. Compared with controls, patients with CKD-MBD showed significantly reduced mental index, antegonial index, panoramic mandibular index, and overall mandibular cortical thickness [5]. Cortical porosity, altered trabecular architecture, lamina dura attenuation or loss, reduced osseous density, focal radiolucent changes, and soft-tissue or vascular calcifications were also more frequent [7]. The increasing prevalence of several abnormalities with advancing CKD stage and their association with PTH, alkaline phosphatase, and eGFR further support a relationship between systemic CKD-MBD severity and jawbone alterations [14].

The biochemical profile of the CKD-MBD group was characterized by lower calcium and higher phosphate, alkaline phosphatase, and PTH concentrations. These findings are consistent with the established pathophysiology of CKD-MBD, in which impaired phosphate excretion, altered vitamin D metabolism, calcium imbalance, and secondary hyperparathyroidism contribute to abnormal bone turnover and mineralization [1–3]. The predominance of stage 4 and stage 5 CKD in the present cohort is relevant because mineral and bone abnormalities generally become more pronounced with progressive renal dysfunction.

The most important radiological finding was the significant reduction in mandibular cortical dimensions. All quantitative CBCT parameters were significantly lower in CKD-MBD patients than controls. These findings are consistent with previous investigations demonstrating mandibular cortical alterations in chronic renal disease [6,8–13]. Çağlayan et al. reported osseous and dental changes in patients with chronic renal failure using CBCT, while Ersu et al. demonstrated differences in mandibular radiomorphometric parameters and trabecular organization using CBCT-based assessment [8,9]. Similarly, Queiroz et al. demonstrated significant relationships between mandibular radiomorphometric indices and biochemical parameters in patients with secondary hyperparathyroidism associated with CKD [10].

The qualitative CBCT findings indicate that CKD-MBD-related jaw involvement extends beyond cortical thinning. Nearly half of the CKD-MBD patients demonstrated lamina dura attenuation or loss, while more than half showed altered trabecular architecture. Cortical porosity and reduced apparent osseous density were also substantially more common than in controls. Earlier reports have described loss of lamina dura, altered mandibular cortical morphology, and other maxillomandibular radiographic manifestations in

renal osteodystrophy [4,6,7]. The present findings therefore reinforce the importance of evaluating both cortical and trabecular compartments during dental CBCT interpretation.

The increase in jawbone abnormalities with advancing CKD stage provides additional support for an association with systemic disease severity. Moderate-to-severe cortical porosity, lamina dura alteration, trabecular abnormalities, reduced osseous density, and soft-tissue or vascular calcification were progressively more frequent from stage 3 to stage 5 CKD. This pattern is biologically plausible because progressive renal dysfunction produces increasingly complex abnormalities in mineral metabolism and bone turnover. Previous CBCT and radiographic studies have similarly reported greater mandibular alterations in patients with advanced renal disease [8–13].

A particularly important finding was the inverse relationship between PTH and mandibular cortical measurements. Overall cortical thickness showed the strongest inverse correlation with PTH, while similar relationships were observed for the mental, antegonial, and panoramic mandibular indices. Alkaline phosphatase also demonstrated significant inverse associations, whereas eGFR was positively correlated with cortical measurements. These findings are consistent with the role of secondary hyperparathyroidism in high-turnover renal bone disease. Persistent PTH elevation increases bone remodeling and may contribute to cortical loss and structural alteration [1–3,10,12]. The present results therefore suggest that mandibular radiomorphometric changes may reflect, at least partly, the severity of the underlying systemic bone-metabolic disturbance.

The weaker associations with serum calcium and phosphate should be interpreted cautiously. CKD-MBD is a dynamic disorder, and isolated biochemical measurements may not adequately represent cumulative exposure or longitudinal mineral imbalance. PTH and alkaline phosphatase may provide more useful indicators of skeletal involvement when interpreted together with other clinical and biochemical parameters [1–3]. Accordingly, CBCT findings should not be interpreted independently but considered alongside the patient's renal status and biochemical profile.

From a dental perspective, the findings have practical implications. Reduced mandibular cortical dimensions and altered trabecular architecture may be relevant when evaluating bone quality before implant placement or dentoalveolar surgery [14]. Lamina dura abnormalities and reduced osseous density may also influence interpretation of periodontal and periapical findings. The identification of vascular or soft-tissue calcifications within a dental CBCT field may provide an additional indication of systemic mineral imbalance and warrants appropriate clinical correlation [15,16]. However, CBCT should remain an adjunctive

dentomaxillofacial imaging modality and should not be considered a substitute for biochemical or nephrological assessment.

The study has limitations. Its single-centre observational design limits generalizability and prevents establishment of causality. The CKD-MBD cohort was clinically heterogeneous with respect to CKD stage and dialysis status [17]. Biochemical measurements represented clinically available assessments rather than longitudinal monitoring, and CBCT cannot directly determine the histological pattern of bone turnover. Furthermore, the study assessed radiological alterations rather than their direct effect on implant survival, surgical complications, or osseous healing [18-20].

Despite these limitations, the consistent reduction in mandibular cortical measurements, increased prevalence of qualitative bone abnormalities, progressive changes with CKD severity, and significant biochemical correlations collectively demonstrate substantial jawbone involvement in CKD-MBD. For dental practitioners and oral and maxillofacial radiologists, systematic evaluation of mandibular cortical and trabecular structures on clinically indicated CBCT examinations may provide valuable adjunctive information for diagnosis and treatment planning in patients with CKD-MBD.

Strengths of the Study

1. The study included a relatively well-defined CKD-MBD cohort with a comparable control group, allowing direct comparison of mandibular bone characteristics.
2. CBCT-based assessment provided three-dimensional evaluation of both quantitative radiomorphometric parameters and qualitative cortical and trabecular alterations.
3. The study evaluated multiple mandibular parameters, including mental index, antegonial index, panoramic mandibular index, and cortical thickness, along with qualitative osseous changes.
4. Radiological findings were correlated with clinically relevant biochemical markers, including PTH, alkaline phosphatase, calcium, phosphate, and eGFR.
5. Assessment by two trained oral and maxillofacial radiologists with evaluation of interobserver and intraobserver reliability strengthened the methodological rigor of the study.
6. The inclusion of patients across different CKD stages allowed assessment of the relationship between CKD severity and jawbone alterations.

CONCLUSION

CBCT-based assessment demonstrated significant jaw bone alterations among patients with chronic kidney disease–mineral and bone disorder compared with individuals without CKD-MBD. Patients with CKD-MBD showed reduced mandibular cortical thickness and significantly lower mental index, antegonial index, and panoramic mandibular index values, together with

a greater frequency of cortical porosity, altered trabecular architecture, lamina dura attenuation or loss, reduced osseous density, focal radiolucent changes, and soft-tissue or vascular calcifications. The increasing prevalence of these abnormalities with advancing CKD stage and their significant association with PTH, alkaline phosphatase, and eGFR indicate a relationship between systemic mineral and bone disturbances and structural changes within the jaws.

From a dental perspective, these findings emphasize the importance of careful assessment of jawbone quality in patients with CKD-MBD, particularly when CBCT is clinically indicated before implant placement, dentoalveolar surgery, or other invasive procedures. CBCT should be regarded as an adjunctive dentomaxillofacial imaging modality and interpreted together with clinical and biochemical assessment. Further multicentre prospective studies with standardized CBCT protocols and longitudinal dental outcomes are warranted to determine the clinical and prognostic significance of these radiomorphometric alterations.

DECLARATIONS

Consent for Publication

Not applicable. No individually identifiable personal data or images are presented in this manuscript.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

Dr. Radhika Besta: Conceptualization, study design, data collection, interpretation of findings, and manuscript preparation.

Dr. Sunitha Kesidi: Clinical coordination, data collection, methodological review, and critical revision of the manuscript.

Dr. Ranga Anand: Radiological assessment, data interpretation, statistical review, and critical revision of the manuscript.

All authors contributed to the study, reviewed the manuscript critically, approved the final version, and agreed to be accountable for all aspects of the work.

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Use of Artificial Intelligence

The authors declare that no generative artificial intelligence tool was used to generate the scientific data, statistical analyses, or research findings reported in this manuscript. Any language or editorial assistance, if applicable, does not alter the scientific content or interpretation of the study.

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