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Biochemical Pattern of Mineral Bone Disorder and Its Association with Fracture Risk Assessed by FRAX Tool in Chronic Kidney Disease Patients

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Abstract | **Original Research Article**

Background: Chronic kidney disease (CKD) is a major and increasing global health problem affecting 10–13% of the population, including a rising burden in Bangladesh. Progressive renal dysfunction leads to disturbances in mineral metabolism causing CKD–mineral bone disorder and increased fracture risk. This study aimed to evaluate the biochemical pattern of CKD–mineral bone disorder and its association with fracture risk assessed by the FRAX tool in CKD patients. ***Methods:*** This cross-sectional study (May 2019–April 2020) was conducted in the Department of Nephrology, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh, enrolling 161 CKD patients (≥18 years) to assess mineral bone disorder and fracture risk with FRAX (Indian model, without BMD). Demographic, clinical, and biochemical data were collected, CKD staged per KDIGO, serum analyzed by standard methods, and data processed in SPSS v22.0 ($p < 0.05$) with ethical approval. ***Results:*** Among 161 CKD patients, vitamin D abnormality (95.0%), elevated iPTH (88.2%), phosphate abnormality (68.9%), and calcium abnormality (53.4%) were highly prevalent. Most patients were in CKD stage G5 (47.8%), and diabetes mellitus was the leading cause of CKD (42.2%). No significant association was observed between CKD stage or most biochemical parameters and FRAX-based fracture risk. However, abnormal iPTH levels were significantly associated with increased major osteoporotic and hip fracture risk ($p < 0.05$). ***Conclusion:*** Disordered mineral metabolism was highly prevalent among CKD patients, and abnormal iPTH levels were significantly associated with increased FRAX-based fracture risk.

Keywords: Biochemical Pattern, Mineral Bone Disorder, Fracture Risk, FRAX Tool, Chronic Kidney Disease.

INTRODUCTION

Chronic kidney disease (CKD) is now a major public health problem affecting an estimated 10–13% of the global population, with a steadily increasing prevalence in recent years [1]. The Global Burden of Disease Study 2010 ranked CKD as the 18th leading cause of death globally, rising from 27th in 1990, indicating a marked increase in disease burden over the past two decades. In Bangladesh, CKD is also showing a rising trend in prevalence or renal insufficiency [2], although population-based data remain limited. A rural population study in Bangladesh reported an overall CKD prevalence of 19% [3].

As renal function declines, there is progressive disruption of mineral homeostasis, resulting in abnormalities of calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism. These biochemical disturbances lead to disordered bone remodeling and extra-skeletal calcification, collectively termed chronic kidney disease–mineral bone disorder (CKD-MBD) [1]. CKD-MBD is now recognized as a systemic syndrome encompassing biochemical abnormalities (calcium, phosphate, PTH, vitamin D, klotho, FGF23, and sclerostin), vascular calcification, and bone disorders [4].

Bone remodeling is a continuous physiological process involving osteoclast and osteoblast activity, regulated by hormonal and local factors. Imbalance in this process leads to bone loss and increased fracture risk. In CKD patients, disturbances in calcium and phosphate

regulation contribute to mineral and bone disorders and ectopic calcification [5]. These biochemical and hormonal abnormalities begin early in CKD progression; increased urinary phosphate excretion and alterations in PTH, FGF23, sclerostin, klotho, and vitamin D metabolites can be observed even at relatively preserved renal function [4]. However, skeletal consequences and increased fracture risk are more evident in advanced CKD stages, particularly stage 3b and beyond when eGFR falls below 45 ml/min/1.73 m² [4].

Among the key regulators of bone metabolism in CKD, parathyroid hormone (PTH) plays a central role. In CKD, PTH secretion becomes dysregulated, leading to increased bone turnover, cortical thinning, porosity, and skeletal fragility. Elevated alkaline phosphatase, a marker of increased bone turnover, is also associated with higher fracture risk [6]. Both low and high PTH levels are linked to increased fracture risk, with studies showing significantly higher fracture incidence at very high PTH levels (>900 pg/ml) [4].

Hyperphosphatemia is another important contributor to CKD-MBD, as rising serum phosphate levels in CKD stimulate both PTH and FGF23 production, thereby contributing to skeletal fragility [7]. Vitamin D also plays a crucial role in calcium and phosphate homeostasis and bone mineralization. Although optimal levels of vitamin D in CKD remain controversial, KDIGO guidelines recommend maintaining 25(OH)D levels above 30 ng/mL [8]. Despite supplementation strategies, vitamin D deficiency or insufficiency remains highly prevalent in CKD patients [4]. Evidence suggests that vitamin D levels are independently associated with bone metabolism and bone mineral density in CKD populations [9,10].

CKD is also associated with a significantly increased risk of fractures compared to the general population, with a 1.5–2-fold higher risk in stage 3 CKD and up to 4-fold higher risk in dialysis patients [11]. Despite this increased risk, the optimal method for fracture risk prediction in CKD remains uncertain. While bone mineral density (BMD) assessment using DEXA and CT is useful, interpretation is limited due to differential involvement of cortical and trabecular bone in CKD and inability of DEXA to distinguish between them. KDIGO 2017 guidelines support BMD use for predicting peripheral fractures in CKD stage 3a–5D, although its predictive value for vertebral fractures remains limited [12].

The FRAX tool, developed by the University of Sheffield, estimates the 10-year probability of major osteoporotic and hip fractures based on clinical risk factors, with or without BMD. Its utility in CKD has been demonstrated in a Canadian study of CKD stage 4 patients, where FRAX effectively discriminated between patients with and without fractures and performed better than BMD for non-spine fracture prediction [11]. However, the best predictive accuracy is achieved when FRAX is combined with BMD assessment.

In this context, there is limited evidence from South Asian populations regarding the pattern of CKD-related mineral bone disorder and its relationship with fracture risk assessed by FRAX. Therefore, the present study was conducted to evaluate the biochemical pattern of mineral bone disorder and its association with FRAX-based fracture risk in patients with chronic kidney disease.

Objective

- To evaluate the biochemical pattern of mineral bone disorder and its association with FRAX-based fracture risk among patients with chronic kidney disease.

METHODOLOGY & MATERIALS

This cross-sectional observational study was conducted in the Department of Nephrology, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh, from May 2019 to April 2020. A total of 161 adult patients with chronic kidney disease (CKD) were enrolled based on predefined inclusion and exclusion criteria to evaluate the biochemical patterns of mineral bone disorder and their association with fracture risk using the FRAX tool.

Inclusion Criteria

i. Patients aged ≥18 years
ii. Diagnosed cases of chronic kidney disease (CKD)

Exclusion Criteria

i. Patients with cognitive impairment
ii. Patients receiving medications known to affect vitamin D absorption or metabolism (e.g., anticonvulsants, isoniazid, rifampicin, theophylline, glucocorticoids, calcium, vitamin D or its analogues, calcimimetics, bisphosphonates)
iii. Patients with primary parathyroid disorders or liver disease
iv. Terminally ill patients

After obtaining informed written consent, data on demographic, clinical, and laboratory variables were collected using a predesigned semi-structured questionnaire. The variables included age, sex, body mass index (BMI), waist and hip circumference, blood pressure, and biochemical parameters such as serum calcium, phosphate, vitamin D, intact parathyroid hormone (iPTH), alkaline phosphatase, uric acid, total protein, and albumin. CKD staging was classified according to KDIGO guidelines (G1–G5). Venous blood samples were collected under aseptic conditions, allowed to clot, and centrifuged. The separated serum was stored

appropriately until biochemical analysis. Laboratory investigations were performed using standard enzymatic and immunoassay techniques. Serum creatinine, urea, calcium, phosphate, uric acid, total protein, and albumin were measured using enzymatic methods. Serum iPTH was analyzed using a chemiluminescent immunometric assay, while 25-hydroxyvitamin D was assessed using radioimmunoassay. All biochemical analyses were performed using a fully automated biochemistry analyzer. Fracture risk was estimated using the FRAX tool (Indian model) without bone mineral density input, calculating 10-year probabilities of major osteoporotic and hip fractures. Data were analyzed using SPSS version 22.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage. Associations between variables were assessed using appropriate statistical tests including t-test, chi-square test, and ANOVA. A p-value <0.05 was considered statistically significant. The study was conducted after obtaining ethical approval from the institutional ethics committee. All participants were informed about the study objectives, procedures, potential risks, and confidentiality, and written informed consent was obtained. Participants retained the right to withdraw from the study at any time without affecting their treatment.

RESULTS

Table 1: Demographic and Clinical Characteristics of the Study Participants (N = 161)

Variable		Frequency (n)	Percentage (%)
Age (years)	≤30	11	6.8
	31–40	25	15.5
	41–50	50	31.1
	51–60	53	32.9
	>60	22	13.7
	Mean ± SD	49.9 ± 11.4	
Gender	Male	85	52.8
	Female	76	47.2
BMI (kg/m²)	Underweight (<18.5)	9	5.6
	Normal (18.5–24.9)	98	60.9
	Overweight (25.0–29.9)	54	33.5
	Mean ± SD	23.5 ± 3.4	16.1 – 32.8
Waist circumference (cm)		87.3 ± 9.7	61.0 – 116.8
Hip circumference (cm)		93.9 ± 9.9	71.0 – 127.1
Systolic BP (mmHg)		136 ± 15	100 – 180
Diastolic BP (mmHg)		85 ± 7	60 – 100

The study included 161 CKD patients, with the highest proportion aged 51–60 years (32.9%), followed closely by 41–50 years (31.1%), and a mean age of 49.9 ± 11.4 years. Males were slightly more common than females (52.8% vs 47.2%), with a male-to-female ratio of approximately 1.1:1. Most participants had a normal BMI (60.9%), while 33.5% were overweight and 5.6% were underweight. The mean BMI was 23.5 ± 3.4 kg/m². Mean waist and hip circumferences were 87.3 ± 9.7 cm and 93.9 ± 9.9 cm, respectively. The mean systolic and diastolic blood pressures were 136 ± 15 mmHg and 85 ± 7 mmHg, respectively.

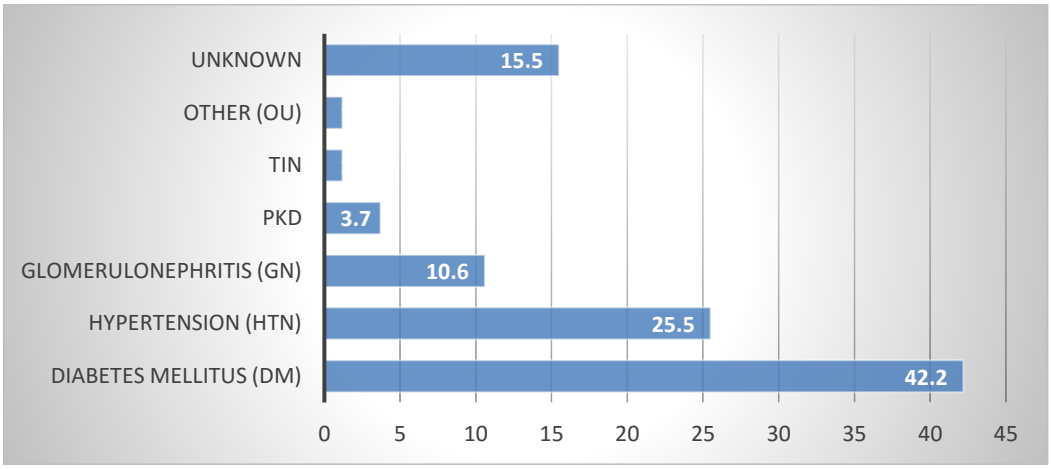

Figure 1: Distribution of Primary Diseases Among Study Participants (N = 161)

Diabetes mellitus was the leading primary cause of CKD in the study population (42.2%), followed by hypertension (25.5%) and glomerulonephritis (10.6%). A notable proportion of patients had unknown etiology (15.5%), while polycystic kidney disease (3.7%) and tubulointerstitial nephritis (1.2%) were less common. Other causes accounted for 1.2% of cases.

Table 2: Biochemical Profile of Study Participants (N = 161)

Parameter	Mean ± SD	Range
Serum calcium (mg/dl)	8.65 ± 1.07	2.30–10.90
Serum phosphate (mg/dl)	5.12 ± 1.94	1.90–15.00
Vitamin D	21.43 ± 9.73	11.20–94.20
iPTH (pg/ml)	183.10 ± 190.71	0–1727
ALP (IU/L)	118.62 ± 81.64	16.40–596
Uric acid (mg/dl)	6.99 ± 2.34	2.10–14.90
Total protein (g/dl)	6.77 ± 1.83	1.60–12.00
Albumin (g/dl)	3.98 ± 0.88	1.29–6.68

The biochemical analysis showed a mean serum calcium level of 8.65 ± 1.07 mg/dl with elevated serum phosphate levels (5.12 ± 1.94 mg/dl). Mean vitamin D level was 21.43 ± 9.73, while iPTH levels demonstrated marked variability with a mean of 183.10 ± 190.71 pg/ml. The mean alkaline phosphatase level was 118.62 ± 81.64 IU/L. Mean serum uric acid, total protein, and albumin levels were 6.99 ± 2.34 mg/dl, 6.77 ± 1.83 g/dl, and 3.98 ± 0.88 g/dl, respectively.

Table 3: Distribution of CKD Stages and FRAX-Based Fracture Risk (N = 161)

CKD Stage	n (%)	Major osteoporotic fracture (%)	Hip fracture (%)
G1	2 (1.2)	0.90 ± 0.00	0.00 ± 0.00
G2	9 (5.6)	1.50 ± 0.89	0.31 ± 0.39
G3a	13 (8.1)	1.37 ± 0.98	0.51 ± 1.02
G3b	19 (11.8)	2.42 ± 2.16	0.45 ± 0.68
G4	41 (25.5)	2.54 ± 2.05	0.49 ± 0.57
G5	77 (47.8)	2.18 ± 1.58	0.41 ± 0.51
p-value		0.19	0.829

Most patients were in CKD stage G5 (47.8%), followed by G4 (25.5%), while only a small proportion were in G1 (1.2%). The mean 10-year probability of major osteoporotic fracture ranged from 0.90% in G1 to 2.54% in G4, while hip fracture risk ranged from 0.00% in G1 to 0.51% in G3a. However, there was no statistically significant association between CKD stage and fracture risk for either major osteoporotic fracture ($p = 0.19$) or hip fracture ($p = 0.829$).

Table 4: Frequency of Abnormal Biochemical Parameters in CKD Patients (N = 161)

Parameter		Abnormal n (%)	Normal n (%)
Serum Calcium	Overall abnormality	86 (53.4)	75 (46.6)
	Hypocalcaemia	70 (43.5)	
	Hypercalcaemia	16 (9.9)	
Serum Phosphate (PO_4)	Overall abnormality	111 (68.9)	50 (31.1)
	Hypophosphatemia	36 (22.4)	
	Hyperphosphatemia	75 (46.6)	
Vitamin D	Overall abnormality	153 (95.0)	8 (5.0)
	Insufficient	75 (46.6)	
	Deficient	78 (48.4)	
iPTH	Overall abnormality	142 (88.2)	19 (11.8)
	Hypoparathyroidism	5 (3.1)	
	Hyperparathyroidism	137 (85.1)	
Alkaline phosphatase	Abnormal	52 (32.3)	109 (67.7)
Uric acid	Abnormal	86 (53.4)	75 (46.6)
Total protein	Abnormal	72 (44.7)	84 (52.2)
Albumin	Abnormal	37 (23.0)	124 (77.0)

Biochemical abnormalities were highly prevalent among CKD patients. Vitamin D deficiency or insufficiency was observed in 95.0% of patients, followed by elevated iPTH levels in 88.2% and phosphate abnormalities in 68.9%. Calcium abnormalities were present in 53.4% of patients, with hypocalcaemia being more common than hypercalcaemia. Abnormal uric acid levels were observed in 53.4%, low total protein in 44.7%, low albumin in 23.0%, and elevated alkaline phosphatase in 32.3%.

Table 5: Association Between Biochemical Parameters and FRAX-Based Fracture Risk (Calcium, Phosphate, ALP, Uric Acid, Total Protein, Albumin)

Parameter	Group	Major osteoporotic fracture (%) (Mean ± SD)	Hip fracture (%) (Mean ± SD)
Calcium	Abnormal	2.22 ± 1.74	0.48 ± 0.65
	Normal	2.15 ± 1.76	0.39 ± 0.52
	p-value	0.749	0.553
Phosphate	Abnormal	2.20 ± 1.79	0.40 ± 0.52
	Normal	2.16 ± 1.64	0.51 ± 0.71
	p-value	0.837	0.369
Alkaline phosphatase	Abnormal	2.33 ± 1.87	0.54 ± 0.75
	Normal	2.12 ± 1.68	0.38 ± 0.49
	p-value	0.577	0.3
Uric acid	Abnormal	2.25 ± 1.84	0.48 ± 0.68
	Normal	2.12 ± 1.62	0.39 ± 0.47
	p-value	0.962	0.903
Total protein	Abnormal	2.20 ± 1.82	0.44 ± 0.59
	Normal	2.24 ± 1.71	0.45 ± 0.61
	p-value	0.536	0.517
Albumin	Abnormal	2.37 ± 2.04	0.57 ± 0.83
	Normal	2.13 ± 1.65	0.39 ± 0.49
	p-value	0.647	0.39

No statistically significant association was observed between serum calcium, phosphate, alkaline phosphatase, uric acid, total protein, or albumin levels and either major osteoporotic fracture or hip fracture risk (all $p > 0.05$). Fracture risk values were comparable between abnormal and normal groups across all parameters.

Table 6: Association Between iPTH Levels and FRAX-Based Fracture Risk

iPTH level	Major osteoporotic fracture (%) (Mean ± SD)	Hip fracture (%) (Mean ± SD)
Abnormal	2.27 ± 1.74	0.46 ± 0.59
Normal	1.72 ± 1.71	0.28 ± 0.56
p-value	0.038	0.047

Patients with abnormal iPTH levels demonstrated significantly higher fracture risk compared to those with normal levels. The mean major osteoporotic fracture risk was 2.27 ± 1.74% in the abnormal group versus 1.72 ± 1.71% in the normal group ($p = 0.038$). Similarly, hip fracture risk was significantly higher in patients with elevated iPTH (0.46 ± 0.59 vs 0.28 ± 0.56; $p = 0.047$).

Table 7: Association Between Vitamin D Status and FRAX-Based Fracture Risk

Vitamin D status	Major osteoporotic fracture (%) (Mean ± SD)	Hip fracture (%) (Mean ± SD)
Deficient	2.38 ± 1.83	0.45 ± 0.58
Insufficient	2.09 ± 1.70	0.44 ± 0.63
Sufficient	1.21 ± 0.55	0.19 ± 0.21
p-value	0.085	0.406

Although fracture risk appeared higher in patients with vitamin D deficiency compared to those with sufficient levels, the association did not reach statistical significance. Major osteoporotic fracture risk ranged from 2.38 ± 1.83% in deficient patients to 1.21 ± 0.55% in sufficient patients ($p = 0.085$), while hip

fracture risk also showed a declining trend with improving vitamin D status but remained non-significant ($p = 0.406$).

DISCUSSION

Disordered mineral metabolism, secondary hyperparathyroidism (SHPT), and vitamin D deficiency are common complications in CKD patients. A high prevalence of biochemical abnormalities of CKD-MBD was observed in this observational study involving CKD stages 2–5D patients, a finding that is consistent with several previous studies, reflecting the well-established and progressive disruption of mineral homeostasis in CKD.

Abnormalities of serum calcium (53.4%), phosphate (68.9%), vitamin D (95.0%), and iPTH (88.2%) were highly prevalent among the study subjects. Most patients demonstrated hypocalcaemia, hyperphosphataemia, vitamin D deficiency or insufficiency, and SHPT. Specifically, hypocalcaemia was observed in 43.5%, hypercalcaemia in 9.9%, hypophosphataemia in 22.4%, and hyperphosphataemia in 46.6% of patients. Vitamin D deficiency and insufficiency were present in 48.4% and 46.6% of patients, respectively, while elevated iPTH levels (secondary hyperparathyroidism) were found in 85.1% of cases. Similar findings were reported in an Indian tertiary care hospital-based study by Vikrant *et al.,* [13] A high prevalence of mineral metabolism disorders has also been reported in Western populations [14]. In the present study of 161 CKD patients, both the prevalence and severity of CKD-MBD were comparable to those reported in other populations, indicating a consistently high global burden of these biochemical abnormalities regardless of geographic variation.

Osteoporotic fractures are associated with increased morbidity and mortality. Low bone mineral density (BMD) is a key determinant of osteoporosis and fracture risk. However, fracture risk is also influenced by several clinical factors independent of BMD, including age, sex, prior fragility fractures, parental history of hip fracture, corticosteroid use, excessive alcohol intake, rheumatoid arthritis, and other conditions such as liver disease and early menopause before 45 years, emphasizing that fracture risk in CKD is multifactorial rather than solely dependent on bone density.

The FRAX tool integrates these clinical risk factors to estimate the 10-year probability of major osteoporotic and hip fractures, with or without BMD. In this study, FRAX scores were calculated using the WHO guideline-based Indian FRAX calculator without BMD. Patients were assessed for relevant clinical risk factors included in the FRAX algorithm, allowing standardized estimation of fracture risk based purely on clinical parameters in the absence of BMD data.

In the present study, no significant association was found between CKD stage and either major osteoporotic fracture risk or hip fracture risk. The mean 10-year probability of major osteoporotic fracture across CKD stages G2, G3a, G3b, G4, and G5 was 1.50 ± 0.89%, 1.37 ± 0.98%, 2.42 ± 2.16%, 2.54 ± 2.05%, and 2.18 ± 1.58%, respectively. Similarly, the mean hip fracture risk across these stages was 0.31 ± 0.39%, 0.51 ± 1.02%, 0.45 ± 0.68%, 0.49 ± 0.57%, and 0.41 ± 0.51%, respectively. There was no statistically significant difference among or between CKD stages for either outcome, suggesting that CKD stage alone may not independently determine fracture risk when assessed by FRAX.

In contrast, Manda *et al.,*[15], using the Indian FRAX calculator, reported significantly higher fracture risk in CKD stage 4 compared to stage 3. The mean 10-year probability of major osteoporotic fracture was 9.47 ± 2.62% in stage 4 versus 1.92 ± 0.8% in stage 3 ($p < 0.0001$). Similarly, hip fracture risk was 4.61 ± 1.45% in stage 4 compared to 0.75 ± 0.49% in stage 3 ($p < 0.0001$), and this discrepancy with the present study may be attributed to differences in patient characteristics, disease severity distribution, and study settings.

In the present study, most CKD patients were above 40 years of age (77.7%). The mean age was 49.9 ± 11.4 years, which is lower than that reported by Manda *et al.,*[15] (57.7 ± 11.3 years) and Levin *et al.,*[14] (71.1 years). Males slightly predominated in the present study (52.8%), with a male-to-female ratio of approximately 1.1:1, which is consistent with the findings of Ghosh *et al.,*[16], reflecting a relatively younger CKD cohort with mild male predominance.

Most patients had a normal BMI (60.9%), with a mean BMI of 23.5 ± 3.4 kg/m², which is comparable to the findings of Manda *et al.,*[15]. Regarding primary renal disease, diabetes mellitus was the most common cause of CKD (42.2%), followed by hypertension (25.5%) and glomerulonephritis (10.6%), similar to observations reported by Ghosh *et al.,*[16], indicating that metabolic and vascular disorders remain the leading etiologies of CKD in this population.

Vitamin D deficiency or insufficiency was highly prevalent in this study, with 48.4% and 46.6% of patients affected, respectively. Similar high prevalence rates have been reported in previous studies, including Manda *et al.,*[15], who reported vitamin D deficiency in 93.3% of patients, and Bansal *et al.,*[17], who found deficiency in 88.9% of cases. The mean vitamin D level in this study was 21.43 ± 9.73, which is higher than the mean level reported by Manda *et al.,*[15] (14 ± 4.19), suggesting persistent but relatively less severe deficiency in this cohort.

In this study, no significant association was observed between major osteoporotic fracture risk and

serum calcium, phosphate, vitamin D, alkaline phosphatase, uric acid, total protein, or albumin levels in CKD patients. Similarly, no significant association was found between hip fracture risk and these biochemical parameters, indicating that individual biochemical markers alone may not adequately predict fracture risk in CKD.

However, a statistically significant association was observed between elevated iPTH levels and fracture risk. Major osteoporotic fracture risk was significantly higher in patients with abnormal iPTH levels (2.27 ± 1.74% vs 1.72 ± 1.71%; p = 0.038). Similarly, hip fracture risk was also significantly higher in the abnormal iPTH group (0.46 ± 0.59% vs 0.28 ± 0.56%; p = 0.047). A similar significant correlation between iPTH levels and FRAX scores was also reported in the study by Manda *et al.,*[15], highlighting the central role of PTH dysregulation in CKD-related skeletal fragility.

Limitations of the study

The study had several limitations:

- The desired sample size was not achieved due to time constraints and the COVID-19 pandemic situation.
- This study was conducted on a relatively small, purposively selected sample, which may not be representative of the general population.
- This was a cross-sectional study without a control group; therefore, causal relationships between variables could not be established.
- The study was conducted over a limited period of time; a longer study duration would have provided more robust findings.

CONCLUSION

Disordered mineral metabolism was highly prevalent among CKD patients, particularly vitamin D abnormality, hypocalcaemia, hyperphosphataemia, and hyperparathyroidism. Although no significant association was observed between CKD stage and FRAX-based major osteoporotic or hip fracture risk, abnormal iPTH levels were significantly associated with increased fracture risk. These findings highlight the importance of monitoring mineral metabolism abnormalities, especially iPTH, in patients with chronic kidney disease.

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