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Original Article

Correlation and prognostic significance of serum levels of 25 hydroxyvitamin D and parathyroid hormone with secondary osteoporosis in male patients with chronic kidney disease

Shouyong Li[1], Xiaojuan Sha[2], Mengxue Xu[3], Liang Li[3], Fumei Li[4]

[1]Department of Clinical Laboratory, Dongying People's Hospital, Dongying 257091, Shandong, China; [2]Medical Record Department, Dongying People's Hospital, Dongying 257091, Shandong, China; [3]Department of Traumatic Orthopedics, Dongying People's Hospital, Dongying 257091, Shandong, China; [4]Department of Pharmacy, Dongying People's Hospital, Dongying 257091, Shandong, China

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Abstract: Objective: To detect serum levels of 25-hydroxyvitamin D (25-(OH)D) and intact parathyroid hormone (iPTH) in male patients with non-dialysis chronic kidney disease (CKD) complicated with secondary osteoporosis, and to explore their predictive value for long-term fracture outcomes. Methods: A total of 312 male patients with non-dialysis CKD were enrolled and divided into three groups according to bone mineral density (BMD): normal bone mass (n = 98), low bone mass (n = 124), and osteoporosis (n = 90) groups. Serum 25-(OH)D, iPTH and BMD were measured. Correlation analysis, receiver operating characteristic curve analysis, Kaplan-Meier survival analysis and Cox regression analysis were performed. Results: The osteoporosis group had significantly lower serum 25-(OH)D levels (14.27±4.13 ng/mL) and higher iPTH levels (158.34±42.17 pg/mL) than the normal bone mass group (all P<0.001). With the progression of CKD stages, serum 25-(OH)D levels gradually decreased, iPTH levels gradually increased, and BMD values gradually declined (all P<0.001). 25-(OH)D and iPTH were independent influencing factors of BMD. The combined detection of 25-(OH)D and iPTH yielded superior diagnostic efficiency, with an area under the curve of 0.915. Low serum 25-(OH)D and high serum iPTH were independent risk factors for fracture events in patients. Conclusion: Combined detection of serum 25-(OH)D and iPTH can assist the early identification of secondary osteoporosis and fracture risk stratification in male patients with non-dialysis CKD, which provides evidence for targeted clinical intervention.

Keywords: Chronic kidney disease, osteoporosis, 25-hydroxyvitamin D, intact parathyroid hormone, male, fracture, prognosis

Introduction

Chronic kidney disease (CKD) has become a major global public health problem requiring urgent intervention. Relevant epidemiological data show that the prevalence of CKD among Chinese adults is approximately 10.8% [1, 2]. CKD not only seriously endangers public physical health but also imposes a heavy economic and medical burden on the national healthcare system. With the progressive decline of renal function, most CKD patients develop CKD-mineral bone disorder (bone mineral density CKD-MBD), a systemic metabolic abnormality. CKD-MBD causes a variety of bone-related complications, and it is closely associated with vascular calcification. It directly affects the long-term survival and prognosis of CKD patients and serves as a key determinant of patients' long-term rehabilitation quality [3].

Renal disease academic institutions have formally proposed the concept of "CKD-related osteoporosis", clarifying that osteoporosis is the core skeletal manifestation of CKD-MBD and requires independent, targeted evaluation methods and clinical intervention strategies. Different from primary osteoporosis in the general population, CKD-related osteoporosis has more complex pathological mechanisms. Its

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pathogenesis involves multiple interactive pathophysiological links, far exceeding the risk factors of traditional osteoporosis.

Epidemiological data indicate that the incidence of osteoporosis in CKD patients ranges from 30% to 45% [4]. These patients have significantly increased fracture susceptibility, long-term disability risk and all-cause mortality [5]. Despite these clear clinical risks, the clinical detection rate and timely intervention rate of CKD-related osteoporosis remain low in routine clinical practice. Notably, male patients are frequently neglected in relevant screening and management programs.

Serum 25-hydroxyvitamin D (25-(OH)D is a core biomarker for evaluating human vitamin D nutritional status, and vitamin D insufficiency or deficiency is extremely prevalent in CKD patients. Intact parathyroid hormone (iPTH) is the key hormone regulating calcium and phosphorus homeostasis. In the early stage of CKD, iPTH presents compensatory elevation. With the continuous deterioration of renal function, persistent hyperparathyroidism occurs, eventually leading to high-turnover bone disease.

Previous studies have separately explored the correlations of 25-(OH)D and iPTH with CKD-MBD. However, few systematic studies have focused on the combined diagnostic value of the two indicators and their efficacy in predicting fracture risk in male patients with non-dialysis CKD [6]. Based on 2024 Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD conceptual framework, this study enrolled male patients with non-dialysis CKD as research subjects [7]. We detected serum 25-(OH)D and iPTH levels, analyzed their correlations with secondary osteoporosis, and explored their predictive value for long-term fracture events. This study aims to provide evidence-based references for early identification, risk stratification and targeted clinical intervention of CKD-related osteoporosis in male patients.

Materials and methods

Research subjects

Case sources and grouping: A total of 312 male patients diagnosed with non-dialysis CKD who received inpatient or outpatient treatment in the Nephrology Department of Dongying People's Hospital from January 2022 to June 2024 were enrolled in this study. Among them, 98 (31.41%) were assigned to the normal bone mass group, 124 (39.74%) to the low bone mass group, and 90 (28.85%) to the osteoporosis group. All patients underwent two independent subgroup analyses: grouping by body mineral density (BMD) T-score and grouping by CKD stage. All patients were further divided into G3a-G5 stages according to CKD staging for subgroup analysis, and the two grouping systems did not overlap or exclude each other. In accordance with the 2024 KDIGO CKD-MBD clinical criteria, osteoporosis was defined as the core skeletal phenotype of CKD-MBD.

The age of enrolled patients ranged from 35 to 75 years. This study complied with the principles of the Declaration of Helsinki. The research protocol was approved by the Ethics Committee of Dongying People's Hospital.

Inclusion criteria: (1) Male gender, aged ≥18 years old; (2) Complete clinical data, including serum 25-(OH)D, iPTH detection results and complete follow-up records.

Exclusion criteria: (1) A history of primary hyperparathyroidism, thyroid diseases, hypogonadism and other endocrine diseases affecting bone metabolism; (2) Use of glucocorticoids, anti-osteoporosis drugs (bisphosphonates, denosumab, teriparatide), active vitamin D and its analogs, or calcium-sensing receptor modulators within 6 months before enrollment; (3) A history of malignant tumors, multiple myeloma or solid organ transplantation; (4) Severe hepatic dysfunction or chronic gastrointestinal malabsorption diseases affecting nutrient and vitamin absorption; (5) Patients receiving hemodialysis or peritoneal dialysis. All enrolled subjects were restricted to non-dialysis CKD patients to eliminate the interference of renal replacement therapy on mineral metabolism.

Research methods

Clinical data collection: Basic clinical data of all subjects were collected in detail, including age, body mass index (BMI), smoking history, drinking history, comorbidities (hypertension, type 2 diabetes mellitus) and medication history (phosphate binders, antihypertensive drugs, hypoglycemic drugs, etc.). CKD staging was

Table 1. Comparison of baseline clinical data among the three groups of patients [$\bar{x} \pm s$, n (%)]

Indicator	Normal Bone Mass (n = 98)	Reduced Bone Mass (n = 124)	Osteoporosis (n = 90)	F/χ^2	P
Age (years)	53.25±8.14	58.67±9.23	64.58±10.12	8.254	<0.001
BMI (kg/m^2)	24.56±2.87	23.11±2.65	21.03±2.41	7.892	<0.001
CKD Stage [n (%)]				15.647	<0.001
G3a	38 (38.78)	24 (19.35)	6 (6.67)		
G3b	32 (32.65)	42 (33.87)	18 (20.00)		
G4	20 (20.41)	40 (32.26)	37 (41.11)		
G5	8 (8.16)	18 (14.52)	29 (32.22)		
Hypertension [n (%)]	70 (71.43)	95 (76.61)	70 (77.78)	1.245	0.537
Diabetes [n (%)]	32 (32.65)	48 (38.71)	40 (44.44)	2.871	0.238

BMI: Body Mass Index, CKD: chronic kidney disease.

performed according to estimated glomerular filtration rate (eGFR): G3a stage (45-59 mL/min/1.73 m^2), G3b stage (30-44 mL/min/1.73 m^2), G4 stage (15-29 mL/min/1.73 m^2), and G5 stage (<15 mL/min/1.73 m^2).

Biochemical index detection: Fasting venous blood samples (5 mL) were collected from all subjects at enrollment, and serum was separated and stored at -80°C for subsequent detection. Serum 25-(OH)D and iPTH levels were quantitatively detected by electrochemiluminescence immunoassay using a Roche Cobas e602 automatic biochemical analyzer. Vitamin D status was classified as deficiency (25-(OH)D <20 ng/mL) and insufficiency (20-30 ng/mL).

Other serum biochemical indicators, including calcium (Ca), alkaline phosphatase (ALP), creatinine (Scr), phosphorus (P), and albumin (Alb), were detected simultaneously. Corrected serum calcium was calculated via the following formula: Corrected calcium (mmol/L) = measured calcium (mmol/L) + 0.02 × (40 - albumin [g/L]). The calcium-phosphorus product was calculated for subsequent analysis.

BMD detection: Dual-energy X-ray absorptiometry (DXA, Discovery A, Hologic, USA) was used to measure the BMD of the lumbar spine (L1-L4), left femoral neck and total hip. All scanning operations were completed by professionally trained technicians with unified standardized operating procedures. Daily quality control calibration was performed to ensure detection accuracy. BMD results were expressed as T-scores, and osteoporosis diagnosis was based on the World Health Organization's diagnostic criteria for male osteoporosis.

The intra-class correlation coefficient (ICC) of repeated lumbar spine and hip BMD measurements was 0.97 (95% CI: 0.95-0.98), indicating excellent test-retest reliability.

Follow-up and prognostic assessment: All subjects received regular follow-up every 6 months after enrollment via outpatient reexamination or telephone interview. The primary endpoint was new-onset fragility fracture, including clinically diagnosed spinal and non-spinal fractures. The secondary endpoints included maintenance dialysis initiation, all-cause mortality, and the study cutoff time (June 2025).

The primary prognostic indicator was fracture-free survival rate. Secondary prognostic indicators included renal function deterioration (eGFR decreased by ≥40% compared with baseline or dialysis initiation) and all-cause mortality. All fracture events were confirmed by imaging examination, and the location, occurrence time and inducement of each fracture were recorded in detail.

Statistical analysis

SPSS 26.0 and MedCalc 20.0 software were used for data statistical analysis. Normally distributed continuous data were expressed as mean ± standard deviation, and one-way analysis of variance (ANOVA) was used for inter-group comparison. Skewed distribution data (such as ALP) were expressed as median (interquartile range, IQR), and the Kruskal-Wallis H test was adopted for inter-group comparison. Categorical

Table 2. Serological and bone density indicators among three groups of patients [$\bar{x} \pm s$, M (P25, P75)]

Indicator	Normal Bone Mass Group (n = 98)	Reduced Bone Mass Group (n = 124)	Osteoporosis Group (n = 90)	F/H	P
25-(OH)D (ng/mL)	27.54±5.68	20.35±4.97	14.27±4.13	12.354	<0.001
iPTH (pg/mL)	65.28±20.45	98.61±31.52	158.34±42.17	15.678	<0.001
Corrective calcium (mmol/L)	2.31±0.15	2.28±0.18	2.25±0.21	1.245	0.291
Blood phosphorus (mmol/L)	1.12±0.21	1.28±0.25	1.49±0.31	8.547	<0.001
Calcium-phosphorus product (mg^2/dL^2)	32.15±5.21	35.87±6.34	41.26±7.85	9.214	<0.001
ALP (U/L)	78.50 (65.00, 92.00)	95.00 (80.00, 115.00)	135.00 (110.00, 178.00)	18.632	<0.001
lumbar vertebra L1-L4 T	-0.35±0.62	-1.68±0.51	-3.12±0.48	20.547	<0.001
Femoral neck T	-0.12±0.55	-1.45±0.48	-2.87±0.52	22.134	<0.001

25-(OH)D: 25-hydroxyvitamin D, iPTH: intact parathyroid hormone, ALP: alkaline phosphatase.

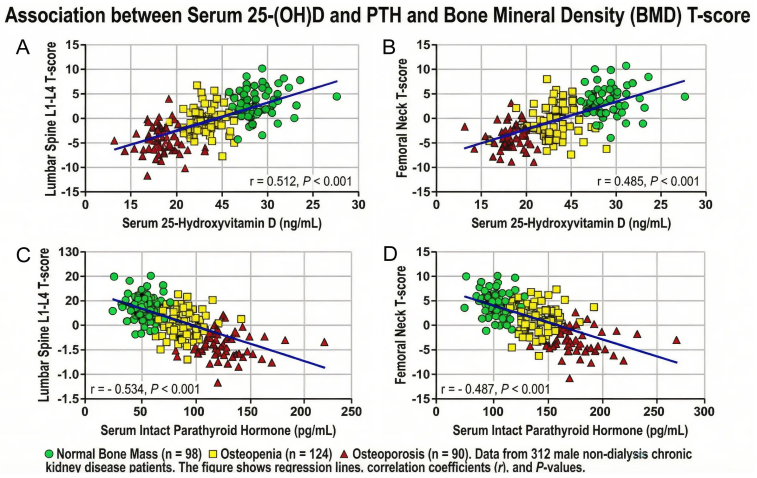


Figure 1. Association of serum 25-(OH)D, iPTH and BMD T-score. A. 25-(OH)D vs. lumbar spine L1-L4 T-score (r = 0.512, P<0.001); B. 25-(OH)D vs. femoral spine T-score (r = 0.485, P<0.001); C. iPTH vs. lumbar spine L1-L4 T-score (r = -0.534, P<0.001); D. iPTH vs. femoral neck T-score (r = -0.487, P<0.001).

data were expressed as case number (percentage), and compared by the χ^2 test or Fisher's exact test.

Pearson or Spearman correlation analysis was used to analyze the correlation between indicators. Multiple linear regression analysis was performed to identify independent influencing factors of BMD. Receiver operating characteristic (ROC) curves were plotted to assess the diagnostic efficacy of 25-(OH)D and iPTH for CKD-related osteoporosis. Cox proportional hazards regression model was used to analyze independent risk factors for new-onset fragility fractures.

This study only completed internal cohort validation, without external independent cohort validation, which may limit the extrapolation of the diagnostic model. The Cox model's proportional hazards assumption was not formally tested in this study. A two-tailed P<0.05 was considered statistically significant.

Quality control

(1) Subject enrollment strictly followed the preset inclusion and exclusion criteria to minimize selection bias; (2) All biochemical indicators were detected with the same batch of reagents and the same instrument, and all BMD tests were completed by a fixed professional technician to ensure test stability and consistency; (3) Two independent researchers conducted double data entry and logical verification to ensure data authenticity and accuracy; (4) Standardized follow-up procedures were implemented, and the overall study dropout rate was controlled within 10%.

Results

Comparison of baseline clinical characteristics

There were significant inter-group differences in age, CKD stage distribution and BMI among the three BMD groups (all P<0.05). Compared with the normal bone mass group, the osteoporosis group had older age, higher proportion of G4-G5 stage CKD and lower BMI. The prevalence of hypertension and type 2 diabetes showed no significant difference among the three groups (all P>0.05) (**Table 1**).

Table 3. Serum 25-(OH)D, iPTH, and lumbar spine t-scores stratified by CKD stage (Same 312 Patients as in BMD Grouping) ($\bar{x} \pm s$)

CKD	n	25-(OH)D (ng/mL)	iPTH (pg/mL)	Lumbar vertebra T-score (SD)
G3a	68	25.32±5.21	72.15±18.34	-0.68±0.54
G3b	92	21.47±4.89	95.47±24.56	-1.34±0.61
G4	97	17.63±4.12	124.38±35.12	-2.01±0.58
G5	55	13.21±3.87	168.72±44.78	-2.78±0.63
F		10.254	14.367	18.452
P		<0.001	<0.001	<0.001

CKD: chronic kidney disease, 25-(OH)D: 25-hydroxyvitamin D, iPTH: intact parathyroid hormone.

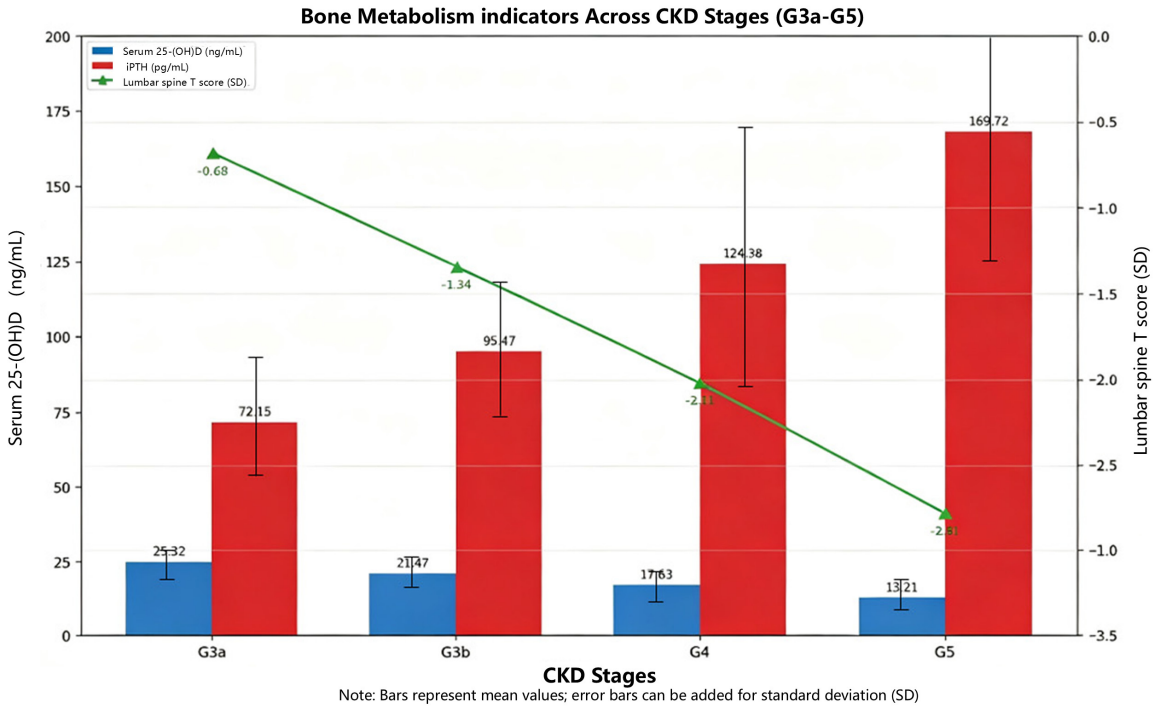


Figure 2. Trends in serum 25-(OH)D, iPTH, and lumbar spine bone mineral density T-scores in patients with different CKD stages.

Comparison of serum biochemical and bone metabolism indicators

With the aggravation of bone loss and osteoporosis, serum 25-(OH)D levels decreased continuously, while iPTH levels increased gradually. The osteoporosis group had significantly lower 25-(OH)D levels (14.27±4.13 ng/mL vs 27.54±5.68 ng/mL) and higher iPTH levels (158.34±42.17 pg/mL vs 65.28±20.45 pg/mL) than the normal bone mass group (all P<0.001).

In addition, serum phosphorus and ALP levels in the osteoporosis group were significantly higher than those in the other two groups. ALP data conformed to skewed distribution and was expressed as median (IQR), with inter-group differences analyzed by the Kruskal-Wallis H test (**Table 2**).

Correlation analysis of serum 25-(OH)D, iPTH and BMD

Stepwise multiple linear regression analysis indicated that after adjusting for age, BMI and eGFR, serum 25-(OH)D (β = 0.324, t = 5.287, P<0.001) and iPTH (β = -0.385, t = -6.014, P<0.001) were independent influencing factors of lumbar BMD (**Figure 1**). The adjusted R^2 of the model was 0.473, indicating that the two

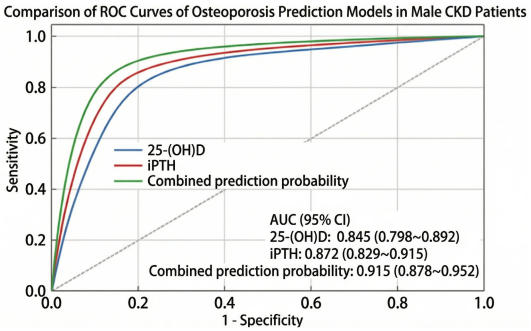


Figure 3. Comparison of ROC curves of osteoporosis prediction models in male CKD patients. Comparison of ROC curves of osteoporosis prediction models in male CKD patients. The blue curve represents 25-(OH)D, the red curve represents iPTH, and the green curve represents combined prediction probability. AUC (95% CI): 25-(OH)D: 0.845 (0.798-0.892); iPTH: 0.872 (0.829-0.915); Combined prediction probability: 0.915 (0.878-0.952).

indicators could explain 47.3% of the total variation in BMD.

With the progression of CKD stages, serum 25-(OH)D levels decreased progressively, iPTH levels increased gradually, and lumbar BMD T-scores decreased significantly. Compared with G3a stage patients, G5 stage patients had a 50% reduction in 25-(OH)D levels, more than doubled iPTH levels, and a mean lumbar T-score of -2.78, indicating severe bone loss (**Table 3**; **Figure 2**).

Diagnostic value of 25-(OH)D and iPTH for CKD-related osteoporosis

ROC curve analysis showed that the AUC of serum 25-(OH)D for diagnosing osteoporosis (T-score ≤-2.5) in male CKD patients was 0.845 (95% CI: 0.798-0.892). At the optimal cutoff value of 16.25 ng/mL, the sensitivity and specificity were 82.50% and 78.90%, respectively. The AUC of iPTH for osteoporosis diagnosis was 0.872 (95% CI: 0.829-0.915), with a sensitivity of 80.00% and specificity of 85.40% at the optimal cutoff value of 112.30 pg/mL.

The combined detection of 25-(OH)D and iPTH had an AUC of 0.915 (95% CI: 0.878-0.952), which was significantly higher than that of single indicator detection (Z = 2.547, P = 0.011) (**Figure 3**). The optimal cutoff values of the two indicators were verified internally and applied to subsequent Kaplan-Meier survival stratification and Cox regression analysis.

Follow-up and prognostic analysis

By the study cutoff time (June 2025), 9 of the 312 patients were lost to follow-up, with a dropout rate of 2.88%. The median follow-up duration was 28 months (range: 6-42 months). During follow-up, 46 patients (14.74%) developed new fragility fractures, including 22 vertebral fractures and 24 non-vertebral fractures. In addition, 88 patients (28.21%) initiated maintenance dialysis, and 15 patients (4.81%) died of all-cause causes.

Baseline comparison between fracture group and non-fracture group: Excluding patients with incomplete follow-up data, the enrolled subjects were divided into fracture group (46 cases) and non-fracture group (257 cases). Baseline data comparison showed that the fracture group had significantly lower serum 25-(OH)D levels (14.27±4.13 ng/mL vs 21.47±6.85 ng/mL, t = 8.647, P<0.001) and significantly higher iPTH levels (176.45±48.23 pg/mL vs 98.34±35.61 pg/mL, t = 10.254, P< 0.001) than the non-fracture group.

Kaplan-Meier survival analysis: Patients were stratified according to the optimal cutoff values of 25-(OH)D (≤16.25 ng/mL vs >16.25 ng/mL) and iPTH (≥112.30 pg/mL vs <112.30 pg/mL). Kaplan-Meier fracture-free survival analysis showed that patients with 25-(OH)D ≤16.25 ng/mL had significantly lower fracture-free survival rate (Log-rank χ^2 = 18.254, P<0.001). Patients with iPTH ≥112.30 pg/mL also presented poorer fracture-free survival (Log-rank χ^2 = 20.136, P<0.001) (**Figure 4**).

Cox regression analysis: Forward likelihood ratio Cox regression analysis showed that decreased serum 25-(OH)D (HR = 0.954, 95% CI: 0.923-0.986, P = 0.006), elevated iPTH (HR = 1.008, 95% CI: 1.004-1.012, P<0.001), advanced age (HR = 1.058, 95% CI: 1.021-1.096, P = 0.002) and reduced eGFR (HR = 0.971, 95% CI: 0.953-0.989, P = 0.002) were independent risk factors for new-onset fractures in male CKD patients (**Table 4**; **Figure 5**).

Discussion

CKD-related osteoporosis is a core skeletal subtype of CKD-MBD [8]. Early identification and individualized intervention of CKD-related osteoporosis have become a research hotspot in clinical nephrology [9]. Different from post-

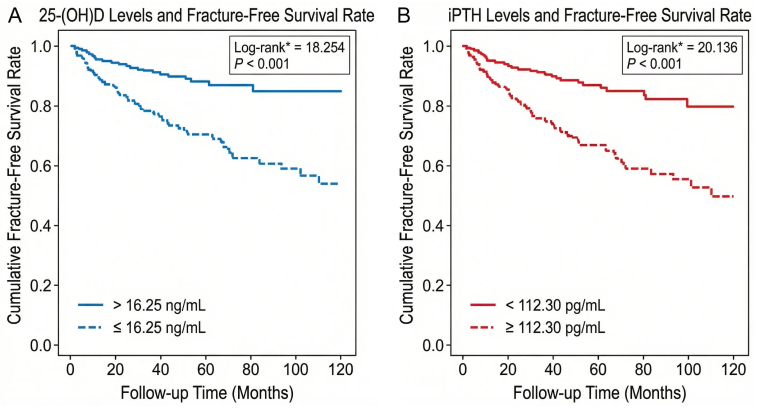


Figure 4. 25-(OH)D concentration and fracture-free survival rate, and iPTH levels and fracture-free survival rate. A. Fracture-free survival stratified by 25-(OH)D: ≤16.25 ng/mL vs. >16.25 ng/mL. Log-rank χ^2 = 18.254, P<0.001. B. Fracture-free survival stratified by iPTH: ≥112.30 pg/mL vs. <112.30 pg/mL. Log-rank χ^2 = 20.136, P<0.001.

menopausal osteoporosis in females, male CKD patients lack obvious early warning signs of bone loss, and most osteoporosis cases are diagnosed only after fracture occurrence [10].

iPTH is a core regulatory hormone of calcium and phosphorus metabolism. Secondary iPTH elevation commonly occurs during CKD progression, which disturbs bone metabolism and induces high-turnover bone disease. Based on the 2024 KDIGO guideline framework, this study systematically analyzed the clinical value of the two indicators in this specific population, aiming to supplement clinical evidence for early risk prediction and intervention of CKD-related osteoporosis [11].

This study confirmed that with the progression of CKD and bone mass loss, serum 25-(OH)D levels decreased significantly while iPTH levels increased markedly, which is consistent with previous domestic and foreign studies [12, 14, 15]. The potential mechanism is explained as follows: impaired renal 1α-hydroxylase activity in CKD patients reduces active vitamin D synthesis. Meanwhile, dietary restriction and reduced sun exposure further aggravate vitamin D deficiency, leading to decreased serum 25-(OH)D levels.

Vitamin D deficiency and hypocalcemia continuously stimulate parathyroid secretion, resulting in compensatory iPTH elevation and secondary hyperparathyroidism [13]. Progressive renal dysfunction impairs phosphorus excretion and causes hyperphosphatemia. Combined with decreased 25-(OH)D, hyperphosphatemia upregulates fibroblast growth factor 23 (FGF23) secretion and downregulates α-klotho expression. The FGF-23/α-klotho axis further inhibits its renal 1α-hydroxylase activity and promotes parathyroid hyperplasia, forming a vicious cycle of mineral and bone metabolism disorder [16-18].

Higher serum phosphorus, calcium-phosphorus product and ALP levels in the osteoporosis group further confirmed the active high-turnover bone metabolic state, which is in line with the pathological characteristics of CKD-MBD [19, 20]. Multiple linear regression analysis verified that 25-(OH)D and iPTH were independent determinants of BMD, independent of age, renal function and BMI. This indicates that vitamin D deficiency and secondary hyperparathyroidism are the core driving factors of bone loss in male non-dialysis CKD patients.

No significant difference in corrected serum calcium was observed among the three groups in this study. This result indicates that PTH-mediated regulatory effect can maintain serum calcium within a normal range in CKD patients. Therefore, single serum calcium detection cannot reflect the real state of bone metabolism, while 25-(OH)D and iPTH are more sensitive biomarkers for evaluating CKD-related bone injury.

This study has several limitations. First, the diagnostic and predictive models of 25-(OH)D and iPTH only completed internal cohort validation, without multi-center external verification, which limits the generalizability of the models. Second, the Cox proportional hazards assumption was not tested, which may affect the accuracy of risk factor analysis. Third, the study only included non-dialysis CKD patients, and the conclusion is not applicable to dialysis or renal transplant patients. Fourth, this study only detected baseline indicators, without dynamic monitoring of 25-(OH)D and iPTH changes. Further longitudinal studies are needed to explore the correlation between dynamic indicator fluctuations and fracture risk.

Table 4. Multivariate cox regression analysis of factors affecting new fractures in male CKD patients

Variable	β	SE	Wald χ^2	P	HR (95% CI)
Age (for each additional year)	0.056	0.018	9.679	0.002	1.058 (1.021-1.096)
eGFR (for every 1 mL/min decrease)	-0.029	0.009	10.383	0.002	0.971 (0.953-0.989)
25-(OH)D (for every 1 ng/mL decrease)	-0.047	0.017	7.642	0.006	0.954 (0.923-0.986)
iPTH (for every 1 pg/mL increase)	0.008	0.002	16.000	<0.001	1.008 (1.004-1.012)

eGFR: estimated glomerular filtration rate, 25-(OH)D: 25-hydroxyvitamin D, iPTH: intact parathyroid hormone.

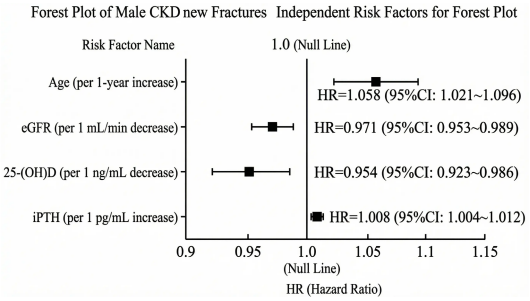


Figure 5. Forest plot of independent risk factors for new fractures in male CKD patients. Forest plot of multivariate Cox regression analysis for factors affecting incident fractures in male non-dialysis CKD patients. Age (HR = 1.058 per 1-year increase, 95% CI: 1.021-1.096), eGFR (HR = 0.971 per 1 mL/min decrease, 95% CI: 0.953-0.989), 25-(OH)D (HR = 0.954 per 1 ng/mL decrease, 95% CI: 0.923-0.986), and iPTH (HR = 1.008 per 1 pg/mL increase, 95% CI: 1.004-1.012) were all independent risk factors for fractures.

In conclusion, this study verified that combined detection of serum 25-(OH)D and iPTH has high diagnostic efficiency for secondary osteoporosis in male non-dialysis CKD patients. Based on the optimal cutoff values obtained from ROC analysis, we established a simple and practical clinical risk stratification system:

High fracture risk: 25-(OH)D ≤16.25 ng/mL combined with iPTH ≥112.30 pg/mL; Moderate fracture risk: single abnormality of 25-(OH)D ≤16.25 ng/mL or iPTH ≥112.30 pg/mL; Low fracture risk: 25-(OH)D >16.25 ng/mL combined with iPTH <112.30 pg/mL.

We recommend targeted intervention thresholds: vitamin D supplementation for patients with 25-(OH)D ≤16.25 ng/mL, and further evaluation and anti-hyperparathyroidism treatment for patients with iPTH ≥112.30 pg/mL.

A standardized clinical workflow is proposed: regular 25-(OH)D and iPTH screening for all male non-dialysis CKD patients; immediate BMD detection and early anti-osteoporosis intervention for high-risk patients; lifestyle intervention and close follow-up for moderate-risk patients; and regular routine monitoring for low-risk patients. This simplified model realizes rapid fracture risk stratification and individualized clinical decision-making, effectively bridging basic biomarker detection and clinical precise intervention.

Disclosure of conflict of interest

None.

Address correspondence to: Fumei Li, Department of Pharmacy, Dongying People's Hospital, 317 South 1st Road, Dongying 257091, Shandong, China. E-mail: lifumeidy@163.com

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