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Biochemical Profile Of Mineral Bone Disease In Chronic Kidney Disease Patients And Correlation With Different Stages Of CKD – A Cross-Sectional Study

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Abstract
Objective *In the 21st century, Chronic kidney disease (CKD) has emerged as one of the most important causes of fatality and suffering, a progressive condition that involves nearly 80 crore individuals of the general population universally. The worldwide median prevalence of CKD is 9.5%. This study aims to study the patterns of various bone mineral abnormalities in different stages of CKD patients.*
Methods *Patients of more than 18 years of age with CKD admitted in wards in SCB Medical College and Hospital, Cuttack, under the Department of Medicine and Department of Nephrology from June 2021 to December 2022 will be included after satisfying the inclusion and exclusion criteria for the cross-sectional study. Inclusion criteria: Pre-dialytic CKD patients of more than 18 years of age are included. Exclusion criteria: Patients already on dialysis and transplant patients, patients with known parathyroid abnormalities, patients who are taking NSAIDS, anti-epileptics, known liver diseases, rickets, osteomalacia patients, and patients with known disease of the thyroid and adrenal gland are excluded from this study.*
Results *The prevalence of hyperparathyroidism in stages 3, 4 & 5 CKD is 52%, 58.82% & 87.5%, respectively. The prevalence of hyperphosphatemia in stage 3, 4 & 5 CKD is 4%, 17.65% & 50%, respectively.*
Conclusion *This study concluded that the vitamin D deficiency pattern was more marked in early CKD than in late stages. Serum bone markers should be done during the screening of CKD-MBD patients, as the serum bone markers have a definite correlation with CKD-MBD patients.*
Keywords: *Chronic kidney disease, hyperparathyroidism, hyperphosphatemia, Adynamic bone disease, hypocalcemia, Bone specific Alkaline Phosphatase*

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I. Introduction

In the 21st century, Chronic kidney disease (CKD) has emerged as one of the most important causes of fatality and suffering; a progressive condition that involves nearly 80 crore individuals of the general population universally.[1] The worldwide median prevalence of CKD is 9•5%.[2] In 2017, almost 247 million CKD cases were reported by India (115 million) and China, contributing one-third globally.[3] A 2021 systematic review noted the CKD prevalence from 12% to 21% in different areas of India.[4] From community-based studies, the collective prevalence of CKD in India was 13.24%.[5]

Kidneys and bones are our body's major metabolic buffer systems that help us maintain the internal milieu. Disease in one naturally is going to involve the other in the long term. The relationship between the two has long been identified but not brought to the attention till the recent decades. This increase in the significance being given to the mineral abnormalities is due to its high association with cardiovascular disease and death due to cardiovascular disease.

CKD is defined as kidney damage or a glomerular filtration rate (GFR) below 60ml/ min per 1.73m2 for 3 months or more, irrespective of the cause.[6] The metabolic abnormalities taking place in CKD commence early in the course of the disease. But the term Renal Osteodystrophy is not adequate to describe all the changes that occur in the body with CKD; it only describes the changes taking place in the bone. Hence, the term CKD-Mineral Bone Disease (CKD-MBD) was coined in September 2005 by KDIGO (Kidney Disease Improving Global Outcomes). [6] It defines CKD-MBD as a "systemic disorder of mineral and bone metabolism due to CKD, manifested by one or a combination of the following: Abnormal metabolism of calcium, phosphorus,

PTH & Vitamin D; Abnormalities in Bone turnover, bone mineralization, bone growth & strength; and Vascular or extra-skeletal calcification. [6]

In the early stages of CKD, there is a drop in the serum total calcium levels due to a fall in the production of renal 1, 25-0H Vitamin D, phosphate retention, and reduced GI absorption of calcium. Persistent hypocalcemia is the potent stimulator for PTH secretion; leads to secondary Hyperparathyroidism & tends to maintain the serum calcium levels, by replacing it from the bones.

The phosphate retention that occurs is not apparent until stage 4 CKD due to increased circulating levels of Fibroblast Growth Factor 23 (FGF-23) as well as compensatory increase in PTH levels, which maintains the serum phosphate level by increased phosphate excretion in urine.[7] After that, the ability to excrete phosphate is reduced, causing hyperphosphatemia. This leads to a decrease in Vitamin D level by inhibiting the renal enzyme 1-alpha-hydroxylase and also worsens the hyperparathyroidism by inducing PTH resistance in bone.[8] Phosphate is also an independent factor in affecting PTH secretion and growth.[9, 10, 11, 12] This vicious cycle of hypocalcemia, hyperparathyroidism, hyperphosphatemia, and again hyperparathyroidism goes on till structural and functional abnormalities arise in the parathyroid gland or the bone pool of calcium is exhausted.

In CKD, in bones, there develops a resistance to the calcemic actions of PTH.[13] This resistance is due to decreased calcitriol levels, phosphate retention, and N-terminal truncated PTH fragments, which have antagonistic actions to PTH.[14, 15] Hence, for mobilizing the bone calcium, the parathyroid gland over secretes PTH.

More than 40% of stage 5 CKD patients have Adynamic bone disease.[16] Adynamic bone disease occurs because of a very low iPTH (< 100 pg/ml) that decreases both osteoblast and osteoclast activities. Bone-specific Alkaline Phosphatase levels fall in Adynamic bone disease. It is a low turnover, normal mineralization state of bone with a low bone volume. In CKD, Adynamic bones have twice the incidence of fracture as compared with high turnover bone states. "It is also highly associated with cardiovascular calcification and mortality.[17, 18, 19]

BMD cannot differentiate the types of renal osteodystrophy.[20, 21, 22] PTH is a marker of parathyroid activity rather than bone turnover.[23] And also, BAP might predict high or low bone turnover.[24, 25, 26] Markers like deoxypyridinoline, Tumor necrosis factor alpha, osteocalcin, and osteoprotegrin have insufficient evidence to be used as markers. All these facts make the bone biopsy the gold standard tool in assessing bone health in CKD. It is invasive and also needs special types of equipment and expertise to carry out. Bone biopsy is also not possible in all clinical settings.

However, despite a high prevalence of MBDs in CKD patients, there are limited data on CKD MBD from India.[27, 28, 29, 30] Even the Indian CKD MBD guidelines of 2011 were based on Western data and expert suggestions, due to a paucity of data from the country, and an urgent need for studies in this area was felt to fill the knowledge gap.[31] Therefore, a prospective study to investigate the prevalence and severity of MBD in CKD patients at our center was performed. This study aims to contribute some data about the patterns of various bone mineral abnormalities in different stages of CKD in the Indian population, where data about the disease is insufficient.

II. Materials And Methods

Study Design: Prospective observational study

Study Location: This was a tertiary care teaching hospital-based study done in Department of General Medicine, at SCB Medical College and Hospital, Cuttack, Odisha.

Study Duration: June 2021 to December 2022.

Sample size: 83 patients.

Subjects & selection method: Patients of more than 18 years of age with CKD admitted in wards in SCB Medical College and Hospital, Cuttack, under the Department of Medicine and Department of Nephrology from June 2021 to December 2022 will be incorporated after fulfilling the inclusion and exclusion criteria for the cross-sectional study.

Inclusion criteria: Pre-dialytic CKD patients of more than 18 years of age are incorporated.

Exclusion criteria: Patients already on dialysis and transplant patients, patients who are taking NSAIDS, anti-epileptics, known liver diseases, patients with known parathyroid abnormalities, rickets, osteomalacia patients, and patients with known disease of the thyroid and adrenal gland are excluded from this study.

Procedure methodology

After obtaining consent from the patient, with aseptic precautions, about 8 ml of venous blood is obtained from the median cubital vein without applying a tourniquet, between 10 am and 12 pm. 3 mL of blood is collected in a K2 EDTA tube for separating plasma. 5 mL of blood is collected in a red-topped standard serum separating tube. After allowing the sample to clot at room temperature for about 1 hour, the samples are centrifuged, and serum & plasma are separately aspirated using Pasteur pipettes. Samples are separately stored in 1 mL Eppendorf tubes. Thus, 4 aliquots are allotted for every patient. Plasma samples are analyzed for 25-OH Vitamin D and iPTH. Serum samples are analyzed for all other biochemical parameters.

Haemoglobin, total leukocyte count, differential count, total platelet count, urine-routine microscopy, fasting blood sugar, 2hour post prandial blood sugar, HBA1C, liver function test, serum protein, serum albumin, serum urea & creatinine, serum sodium, serum potassium, serum calcium (range is 0.8-20.1 mg/dl), serum phosphate, serum magnesium (normal range is 1.6 to 2.8 mg/dl.), serum vitamin D (measuring range is 3.00-70.0 ng/ml), serum parathormone (measuring range is 1.20 - 5000 pg/ml), and bone specific alkaline phoshatase were done.

A questionnaire regarding symptoms of CKD-MBD is given to all the patients in our study. “Bone pain, proximal muscle weakness, and fragile fractures” are asked about. Those who have one or more of the symptoms are considered symptomatic. Ophthalmic fundus examination is carried out in eligible patients who don’t have cataract or other ocular abnormalities.

Statistical analysis:

The data were analyzed using SPSS (Statistical Package for Social Science) Version 16.01. The data collected were scored and analyzed & categorical variables were presented as frequencies and percentages. Student t-test was used for testing the significance of all the variables’ means and standard deviations in groups. The chi-square test was used to compare proportions. The Pearson Correlation test was used to find out the relationship between the variables. All the statistical results were considered significant at a P value ≤ 0.05.

III. Results

This study was conducted with 83 CKD patients. Out of them, 42 were females and 41 males, leading to a slight female predominance; the M: F ratio is 0.9:1. In the present study, the average age of CKD patients is 54.3 + 12.67 years.

Table no 1 shows the prevalence of hypocalcemia in stages 3, 4 & 5 CKD is 12%, 11.6% & 37.5%, respectively. The overall prevalence of hypocalcemia, normocalcemia & hypercalcemia in stage 3-5 CKD is 19.28%, 72.29% & 8.43%, respectively. This prevalence is not statistically significant as per our study (p value – 0.11). The prevalence of hyperparathyroidism in stages 3, 4 & 5 CKD is 52%, 58.82% & 87.5%, respectively. The overall prevalence of hypoparathyroidism, normoparathyroidism & hyperparathyroidism in stage 3-5 CKD is 6.02%, 28.92% & 65.06%, respectively. This prevalence is statistically significant as per our study (p value – 0.04). The prevalence of Vitamin D deficiency in stage 3, 4 & 5 CKD is 4%, 20.59% & 0%, respectively. The overall prevalence of vitamin D deficiency and no deficiency in stage 3-5 CKD is 9.64% & 90.36%, respectively. This prevalence is statistically significant as per our study (p value – 0.02).

Table no 1: Calcium vs. e-GFR, iPTH vs. e-GFR, Vitamin D vs. e-GFR

Calcium	**e-GFR**							
	Stage 3		**Stage 4**		**Stage 5**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Below normal	3	12.00%	4	11.76%	9	37.50%	16	19.28%
Normal	19	76.00%	27	79.41%	14	58.33%	60	72.29%
Above normal	3	12.00%	3	8.82%	1	4.17%	7	8.43%
Total	25	100%	34	100%	24	100%	83	100%
Chi- square	7.65						Not Significant	
P-value	0.11							
iPTH	**e-GFR**							
	Stage 3		**Stage 4**		**Stage 5**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Below normal	3	12.00%	1	2.94%	1	4.17%	5	6.02%
Normal	9	36.00%	13	38.24%	2	8.33%	24	28.92%
Above normal	13	52.00%	20	58.82%	21	87.50%	54	65.06%
Total	25	100%	34	100%	24	100%	83	100%
Chi- square	9.84						Significant	
P-value	0.04							
Vitamin D	**e-GFR**							
	Stage 3		**Stage 4**		**Stage 5**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Not deficiency	24	96.00%	27	79.41%	24	100%	75	90.36%

Deficiency	1	4.00%	7	20.59%	0	0%	8	9.64%
Total	25	100%	34	100%	24	100%	83	100%
Chi- square	8.15						Significant	
P-value	0.02							

Table no 2 shows the overall prevalence of hypomagnesemia, normomagnesemia & hypermagnesemia in stage 3-5 CKD is 18.07%, 39.76% & 42.17%, respectively. This prevalence is statistically not significant as per our study (p value = 0.53). The prevalence of hyperphosphatemia in stage 3, 4 & 5 CKD is 4%, 17.65% & 50%, respectively. The overall prevalence of hypophosphatemia, normophosphatemia & hyperphosphatemia in stage 3-5 CKD is 9.64%, 67.47% & 22.89%, respectively. This prevalence is statistically significant as per our study (p value = 0.001) [Table: - 2]. The overall prevalence of low BAP, normal BAP & high BAP in stage 3-5 CKD is 3.61%, 74.70% & 21.69%, respectively. This prevalence is statistically not significant as per our study (p value = 0.18).

Table no 2: Magnesium vs. e-GFR, Phosphorus vs. e-GFR, BAP vs. e-GFR

Magnesium	**e-GFR**							
	Stage 3		**Stage 4**		**Stage 5**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Below normal	4	16%	6	17.65%	5	20.83%	15	18.07%
Normal	7	28%	16	47.06%	10	41.67%	33	39.76%
Above normal	14	56%	12	35.29%	9	37.50%	35	42.17%
Total	25	100%	34	100%	24	100%	83	100%
Chi- square	3.1504						Not Significant	
P-value	0.53							
Phosphorus	**e-GFR**							
	Stage 3		**Stage 4**		**Stage 5**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Below normal	4	20.00%	3	8.82%	0	0%	7	9.64%
Normal	19	76.00%	25	73.53%	12	50.00%	56	67.47%
Above normal	1	4.00%	6	17.65%	12	50.00%	19	22.89%
Total	25	100%	34	100%	24	100%	83	100%
Chi- square	17.28						Significant	
P-value	0.001							
BAP	**e-GFR**							
	Stage 3		**Stage 4**		**Stage 5**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Below normal	2	8.00%	0	0%	1	4.17%	3	3.61%
Normal	21	84.00%	24	70.59%	17	70.83%	62	74.70%
Above normal	2	8.00%	10	29.41%	6	25.00%	18	21.69%
Total	25	100%	34	100%	24	100%	83	100%
Chi- square	6.21						Not Significant	
P-value	0.18							

Table no 3 shows the prevalence of low BAP in asymptomatic and symptomatic MBD in CKD stage 3-5 is 4.08% & 2.94%, respectively. The prevalence of high BAP in asymptomatic and symptomatic MBD in CKD stage 3-5 is 22.45% & 20.59%, respectively. The difference in Bone turnover markers in stage 3-5 CKD in asymptomatic and symptomatic MBD patients is statistically not significant (p value = 0.94). So we infer that symptomatic MBD patients need not have abnormal BAP and thus abnormal bone turnover. The prevalence of hypomagnesemia in asymptomatic and symptomatic MBD in CKD stage 3-5 is 18.37% & 17.65%, respectively. The prevalence of hypermagnesemia in asymptomatic and symptomatic MBD in CKD stage 3-5 is 44.90% & 38.23%respectively. The prevalence of magnesium abnormalities in stage 3-5 CKD with symptomatic MBD and asymptomatic MBD is not statistically significant (p value – 0.78). Hence, clinical symptomatology of CKD-MBD need not predict the bone turnover rate.

Table no 3: Symptoms vs. BAP, Symptoms vs. Magnesium

BAP	**Symptom Status**					
	Asymptomatic		**Symptomatic**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**
Below normal	2	4.08%	1	2.94%	3	3.61%
Normal	36	73.47%	26	76.47%	62	74.70%
Above normal	11	22.45%	7	20.59%	18	21.69%
Total	49	100%	34	100%	83	100%
Chi- square	0.13				Not Significant	
P-value	0.94					
Magnesium	**Symptom Status**					
	Asymptomatic		**Symptomatic**		**Total**	

	N	%	N	%	N	%
Below normal	9	18.37%	6	17.65%	15	18.07%
Normal	18	36.73%	15	44.12%	33	39.76%
Above normal	22	44.90%	13	38.23%	35	42.17%
Total	49	100%	34	100%	83	100%
Chi- square	0.4922				Not Significant	
P-value	0.78					

Table no 4 shows ophthalmic fundus examination is carried out in 38 patients of the total of 83. The prevalence of abnormal BAP is not statistically significant (p value – 0.06) in any type of retinopathy encountered.

Table no 4: BAP with Fundus

Fundus	BAP							
	Below normal		Normal		Above normal		Total	
	N	%	N	%	N	%	N	%
CRVO	0	0%	1	3.57%	0	0%	1	2.63%
HTR-1	0	0%	1	3.57%	0	0%	1	2.63%
HTR-2	0	0%	0	0%	1	12.50%	1	2.63%
HTR-3	1	50.00%	0	0%	0	0%	1	2.63%
IRMA	0	0%	1	3.57%	0	0%	1	2.63%
NPDR	0	0%	13	46.44%	3	37.50%	16	42.11%
PDR	0	0%	1	3.57%	1	12.50%	2	5.26%
RP	0	0%	1	3.57%	0	0%	1	2.63%
Normal	1	50.00%	10	35.71%	3	37.50%	14	36.84%
Total	2	100%	28	100%	8	100%	38	100%
Chi- square	25.34						Not Significant	
P-value	0.06							

Table no 5 shows there is a positive correlation between BAP and iPTH, with a Pearson correlation of 0.34, which is statistically extremely significant (p value < 0.001). Very high values of BAP are associated with very high values of iPTH, indicating that those patients might be in high-turnover bone disease. A Positive correlation is seen between BAP and Vitamin D, and it is statistically significant (p value – 0.01). The iPTH and vitamin D are inversely correlated, but this correlation is not statistically significant.

Table no 5: Correlation between BAP, iPTH, Vitamin D

		BAP	iPTH	Vitamin D
BAP	Pearson correlation	1	0.34	0.28
	Sig. (2 tailed)	-	0.001**	0.01*
	N	83	83	83
iPTH	Pearson correlation	0.34	1	-0.10
	Sig. (2 tailed)	0.001**	-	0.36
	N	83	83	83
Vitamin D	Pearson correlation	0.28	-0.10	1
	Sig. (2 tailed)	0.01*	0.36	-
	N	83	83	83
***Significant, **Extremely Significant**				

Table no 6 shows iPTH and calcium are found to be negatively correlated, i.e., when calcium falls, iPTH rises, and the reverse occurs in hypercalcemia. The parathormone is a minute-to-minute compensatory response of the body to a fall in serum ionized calcium levels. Hence, this correlation is strong (p value – 0.01).

Table no 6: Correlation between Calcium, Vitamin D and iPTH

Variable		Calcium	Vitamin D
iPTH	Pearson correlation	-0.30	-0.10
	Sig. (2 tailed)	0.01*	0.36
	N	83	83
***Significant**			

IV. Discussion

We studied 83 CKD stage 3-5 patients with an average age of 54.3 +/- 12.67 years. The study has a slight female predominance, with the M: F ratio of 0.9:1. The symptomatic CKD-MBD individuals, according to our study, comprised 40.96%. The remaining 59.03% didn't have any symptoms related to CKD-MBD. This further reveals how disguising the clinical picture of MBD is in our setup.

The prevalence of hypoparathyroidism is 6.02%, indicating the possible load of low turnover bone disease, whereas hyperparathyroidism, with a cut-off of iPTH > 65 pg/ml, is noted in 52% in stage 3, 58.82% in stage 4, and 87.5% in stage 5, with an overall 65.06% indicating high turnover bone disease. Reeta et al. study noted hyperparathyroidism in 55.5% in stage 3, 68.6% in stage 4, and 83.8% in stage 5, with an overall 71.12%.[32] Vikrant et al. study noted hyperparathyroidism in 72.7% in stage 3, 85.8% in stage 4, and 92.5% in stage 5, with an overall 82.7%.[33] Other studies done by Z. Jabbar et al. showed hyperparathyroidism overall to be 73%, A. T. Valson et al. showed 57.3% in stage 4 CKD and 89.5% in stage 5 CKD, with an overall 79.3% hyperparathyroidism, and B. Ghosh et al. showed 84.62% in stage 4 and 88.29% in stage 5. [34, 354, 29] High turnover state assessed by Z. Jabbar et al. is 60% with a cut-off of iPTH as > 300 pg/ml.[34] In our study, 39.7% of patients have an iPTH value of more than two to nine times the normal upper limit, out of which 57.6% have high BAP, indicating that the truly high turnover state exists in less than what is estimated by iPTH. The overall prevalence of hypoparathyroidism, normoparathyroidism & hyperparathyroidism in stage 3-5 CKD is statistically significant (p value = 0.04) as per our study.

The prevalence of hypocalcemia in our study is noted to be 12%, 11.76%, and 37.5% in stages 3, 4 & 5 CKD, respectively, with an overall 19.28%. Reeta et al. study noted hypocalcemia in 12.3%, 18.6%, and 36.1% in stages 3, 4 & 5 CKD, respectively, with an overall 42.85%.[32] Vikrant et al. study noted hypocalcemia in 25.1% in stage 3, 14.2% in stage 4, and 32.1% in stage 5, with an overall 24.41%.[33] A study by Agarwal et al. showed that 29.9% and 49.6% of hypocalcemia are prevalent in stages 4 and 5.[36] The overall prevalence of hypocalcemia, normocalcemia & hypercalcemia in stage 3-5 CKD is not statistically significant as per our study. The cause of a high percentage of normocalcemia may be due to the intake of calcium tablets for CKD.

Hyperphosphatemia is found in 4%, 17.65% & 50% in stages 3, 4 & 5 of CKD, with an overall of 22.89% in our study. It is in concordance with other studies done by Valson et al., where it was 59%, by Ghosh et al., where it was 64.10% in stage 4 and 70.27% in stage 5.[35, 29] Hyperphosphatemia is found in 13.4%, 32.8% & 66.4% in stages 3, 4 & 5 of CKD, with an overall of 41.2% in the Reeta et al. study.[32] Hyperphosphatemia is found in 32.8%, 52.2% & 83.6% in stages 3, 4 & 5 of CKD, with an overall of 53.72% in the Vikrant et al. study.[33] It can be seen that phosphate retention increases with a fall in GFR. These are the potential candidates for phosphate-lowering therapies if clinically feasible. The overall prevalence of hypophosphatemia, normophosphatemia & hyperphosphatemia in stage 3-5 CKD is statistically extremely significant (p value = 0.001) as per our study.

In this present study, Vitamin D deficiency is found only in stages 3 and 4 of CKD, 4% and 20%, respectively. In stage 5 CKD, vitamin D deficiency is not found. Previous studies done by Vikrant et al. and Z. Jabbar et al. showed up to 80% and 90.71% of vitamin D deficiency.[33, 34] The Reason for this is not known. A bone biopsy can explain this. The overall prevalence of vitamin D deficiency and no deficiency in stage 3-4 CKD is statistically significant (p value = 0.02) as per our study.

In our study, the prevalence of an abnormal BAP was not found in a statistically significant percentage among any of the following groups, i.e., symptomatic individuals/ patients with an abnormal fundus examination. Thus, none of the above features in a CKD-MBD patient can predict an abnormal bone turnover. Only bone turnover markers next to bone biopsy can predict bone turnover diseases in CKD. The overall prevalence of low BAP, normal BAP & high BAP in stage 3-5 CKD is statistically not significant as per our study.

In our study, statistically significant correlation, as analyzed by Pearson coefficient, is seen between iPTH and calcium (r = -0.30; p = 0.01); iPTH and BAP (r = 0.34; p = 0.001); BAP and Vitamin D (r = 0.28; p = 0.01). The overall prevalence of hypomagnesemia, normomagnesemia & hypermagnesemia in stage 3-5 CKD is statistically not significant as per our study.

V. Conclusion

This study shows symptomatology of CKD-MBD patients does not predict the bone turnover rate. This study concluded that the vitamin D deficiency pattern was more marked in early CKD than in late stages. Bone turnover rate can be best assessed by turnover markers like iPTH, BAP, serum calcium, serum phosphorus, etc. Serum bone markers should be done during the screening of CKD-MBD patients, as the serum bone markers have a definite correlation with CKD-MBD patients. The screening should begin in the early stage of CKD.

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