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Khaled Ibrahim Rabea
Department of Internal
Medicine and Nephrology,
Faculty of Medicine, Tanta
University, Tanta, Egypt

Hanaa Ibrahim Okda
Department of Internal
Medicine and Nephrology,
Faculty of Medicine, Tanta
University, Tanta, Egypt

Mohamed Attia Saad
Department of Clinical
Pathology, Faculty of
Medicine, Tanta University,
Tanta, Egypt

Gamal Fathy ELNaggar
Department of Internal
Medicine and Nephrology,
Faculty of Medicine, Tanta
University, Tanta, Egypt

Corresponding Author:
Khaled Ibrahim Rabea
Department of Internal
Medicine and Nephrology,
Faculty of Medicine, Tanta
University, Tanta, Egypt

Prevalence of mineral bone disorders in chronic kidney disease patients in Gharbia governorate

Khaled Ibrahim Rabea, Hanaa Ibrahim Okda, Mohamed Attia Saad and Gamal Fathy ELNaggar

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Abstract

Background: As kidney function deteriorates, there is a gradual breakdown in the balance of minerals and hormones in the body. The mineral and endocrine functions play a crucial role in regulating of bones. Chronic kidney disease (CKD) - mineral bone disorders (MBD) refer to a wide-ranging clinical condition that arises as a result of CKD, affecting the body's mineral and bone metabolism. The objective of this study was to assess the frequency of mineral bone disorder (MBD) in patients with CKD in Gharbia governorate.

Methods: This cross-sectional and comparative study was carried out from January 2018 to December 2019 at Tanta University Hospitals and Gharbia governmental and central hospitals on 1400 patients aged more than 18 years old, both sexes. Patients are categorized into two equal groups: Group 1: (n=700) non-hemodialysis-dependent CKD patients and Group 2: (n=700) hemodialysis-dependent CKD patients.

Results: The frequency of elevated bone turnover was substantially greater in the individuals with ESRD compared to those with CKD (42.9% vs 1.4%, $p < 0.001$). Conversely, the occurrence of poor bone turnover was notably greater in the CKD patients compared to the ESRD patients (78.6% vs 35.7%, $p < 0.001$). A strong positive connection was seen between serum PTH levels and age ($r = 0.122$; $p < 0.001$), blood urea ($r = 0.402$; $p < 0.001$), serum creatinine ($r = 0.467$; $p < 0.001$), serum phosphorus ($r = 0.549$; $p < 0.001$), and ALP ($r = 0.595$; $p < 0.001$). A strong inverse relationship was seen between blood PTH levels and eGFR ($r = -0.558$; $p < 0.001$), as well as serum calcium levels ($r = -0.646$; $p < 0.001$). A strong correlation was seen between several categories of bone mineral density (BMD) and the features and etiology of CKD ($p < 0.001$).

Conclusions: In Gharbia Governorate, BMD is prevalent in CKD. 80% of the patients with non-dialyzable CKD and ESRD had low and high bone turnover.

Keywords: Prevalence, MBD, CKD, hemodialysis, Gharbia governorate

Introduction

Chronic kidney disease (CKD) is defined by structural and functional abnormalities in the kidney that continue for more than three months, regardless of whether there is a decline in glomerular filtration rate (GFR). These abnormalities arise because the kidneys continue to operate abnormally. It may also be characterized by the detection of urine albumin with an excretion rate over 300 mg per 24 hours ^[1]. CKD impacts around 5-10% of the global population, with an estimated annual incidence rate of around 5-8% ^[2].

Chronic kidney disease-mineral bone disorder (CKD-MBD) is a systemic disease that affects the whole body. The expanded notion of CKD-MBD encompasses disturbances in mineral metabolism, deviations in bone structure, and the presence of calcifications beyond the skeletal system ^[3].

Patients with CKD have significant metabolic alterations that strongly impact mineral and bone metabolism. CKD leads to changes in the levels of serum phosphorus, calcium, vitamin D, PTH, and fibroblast growth factor-23 (FGF-23) ^[4]. Starting with stage 3 of CKD, the kidneys' capacity to eliminate phosphorus is reduced, resulting in increased levels of phosphorus and PTH, as well as decreased levels of 1,25 (OH) 2D, which are related with increasing levels of FGF-23. Impairment of the conversion of 25-hydroxyvitamin D to 1, 25-dihydroxyvitamin D leads to a decrease in the absorption of calcium in the intestines ^[3].

CKD patients may have asymptomatic bone and mineral disturbances for an extended period.

The first presentation of CKD-MBD is mostly characterized by biochemical abnormalities. In advanced stages of the illness, individuals may have bone and muscular pain, weakness, fractures, and even avascular necrosis [5]. CKD patients had a significantly elevated prevalence of bone fractures. The height of people with CKD is twice that of those without CKD. Osteoblastoma, also known as brown tumors, may be seen in severe cases of CKD-MBD due to increased osteoclastic activity driven by PTH [6].

Severe secondary hyperparathyroidism may cause calcific uremic arteriolopathy, a potentially fatal illness characterized by the calcification of tiny cutaneous blood arteries [7].

Quantifying MBD as a consequence of CKD is crucial, both before to and during renal replacement therapy (RRT). Treatments are focused on managing excessive levels of phosphates in the blood, overactive parathyroid glands, and insufficient vitamin D levels, among other conditions. These interventions include the use of phosphate binders, administration of vitamin D analogues, use of calcimimetics, performing parathyroidectomy, and in some cases, kidney transplantation [8].

This research aimed to evaluate the prevalence of mineral and bone disorder (MBD) in patients with CKD in Gharbia governorate.

Patients and Methods

This research was conducted on a sample of 1400 patients, aged 18 years or older, of both genders. Group 1 consisted of patients diagnosed with CKD who did not need hemodialysis. Group 2 consisted of patients diagnosed with ESRD who were undergoing maintenance hemodialysis (3 sessions per week for at least 3 months). The research was done from January 2018 to December 2019 with clearance from the Ethical Committee at Tanta University Hospitals and Gharbia governmental and central hospitals. The patients provided their informed written permission.

Exclusion criteria were patients with chronic liver disease, primary hyperparathyroidism, patients with previous history of parathyroidectomy, patients with malignancies and patients with granulomatous disease e.g. sarcoidosis.

Patients are categorized into two equal groups: Group 1: (n=700) non-hemodialysis-dependent CKD patients and Group 2: (n=700) Hemodialysis-dependent CKD patients.

All patients were subjected to: history taking, history of comorbidities, cause and duration of CKD, hemodialysis history, general and local examination and laboratory investigations [blood urea and serum creatinine, serum calcium and phosphorus, serum 25-OH cholecalciferol level: Two ml blood was drawn on serum tube and they were measured by calorimetric and enzymatic assay (biomajesty) [9], total alkaline phosphatase, Na and K levels].

Serum intact parathyroid hormone (PTH) estimations

It was performed using electrochemiluminescence immunoassay (ECLIA) on the fully automated vidas (Biomerieux). The samples underwent centrifugation, followed by freezing the serum at a temperature of -40° till evaluation. The PTH molecule has a half-life of about 2 - 4 minutes [10]. Currently, the circulating PTH undergoes degradation into several peptide fragments, each with distinct lengths and biological functions [11]. Various types of PTH may be quantified in the blood serum, including

intact PTH, N-terminal PTH, mid-molecule PTH, and 75 terminal PTH. Currently, widely used PTH tests, referred to as "intact," rely on the ELISA double-sandwich technique. Despite the widespread use of intact PTH assays, there is substantial evidence of considerable heterogeneity across tests. The antibodies used in this iteration of experiments were initially designed to specifically identify the C-terminal and N-terminal regions. Subsequently, it was shown that such antibodies also attach to other portions of the PTH molecule, most of which are not active, but some may possess inhibitory characteristics [12].

Estimation of eGFR: Which was calculated using MDRD formula ($eGFR = 186.3 \times (\text{serum creatinine})^{-1.154} \times (\text{Age}^{-0.203}) \times (0.742 \text{ if female}) \times (1.21 \text{ if African American})$) [13].

Diagnosis of BMD and study's Outcomes: The product was developed using laboratory markers linked to MBD, such as serum PTH, serum calcium, serum phosphorus, and serum ALP. The subjects under scrutiny were classified into 3 cohorts according to their serum PTH levels.

The patients are categorized into three groups according to their PTH levels. The first cohort comprises individuals with PTH levels < 150 pg/ml, which signifies suboptimal bone remodeling. The second category comprises individuals with PTH levels ranging from 150 to 300 pg/ml, which indicates a typical rate of bone remodeling. The third category consists of individuals with PTH levels over 300 pg/ml, which indicates elevated bone turnover [14].

The primary outcome was the prevalence of BMD among the studied groups, while the secondary outcomes were the association between BMD and different clinical and laboratory parameters.

Sample Size Calculation

In order to find patients who were eligible, we used a non-probability sequential sampling method. All patients were recruited from Tanta University Hospitals and Gharbia governmental and central hospitals. A total of 1400 participants were deemed eligible.

Statistical analysis

Utilizing SPSS v27 (IBM, Chicago, Illinois, United States of America), the statistical analysis was carried out. Histograms and the Shapiro-Wilks test were used in order to determine whether or not the data distribution in question was normal. In order to examine the parametric data, an analysis of variance (ANOVA) test was first performed, and then a post hoc test (Tukey) was performed. The mean and standard deviation (SD) were represented as the parametric data. In order to represent the non-parametric quantitative data, the median and the interquartile range (IQR) were used. The Kruskal-Wallis test was then utilized in order to analyze the data. For the purpose of comparing each group, the Mann Whitney test was used. The Chi-square test was used to analyze the qualitative variables, which were reported in terms of frequency and percentage (%). The equation known as the Pearson moment correlation is used for the purpose of determining the linear connection that exists between variables that are dispersed in a systematic manner. For the purpose of determining the non-linear monotonic connection that exists between variables that do not conform to a normal distribution, the Spearman rank

correlation equation is used. It was determined that a two-tailed P value that was lower than 0.05 was statistically significant.

analyzed groups in terms of sex, etiology, and duration of CKD ($p = 0.024$, $p < 0.001$, and $p < 0.001$, respectively). There were no notable disparities in age across the groups under investigation. Table 1.

Results: Significant differences were seen between the

Table 1: Comparison of the 2 analyzed groups based on demographic characteristics, etiology, duration of CKD, and length of dialysis

		Group I (n = 700)	Group II (n = 700)	P
Age (years)		55.13±8.83	56.30±8.59	0.117
Sex	Male	440 (62.9%)	480 (68.6%)	0.024*
	Female	260 (37.1%)	220 (31.4%)	
Cause of CKD	DM	290 (41.4%)	220 (31.4%)	<0.001*
	HTN	320 (45.7%)	360 (51.4%)	
	Others	90 (12.9%)	120 (17.1%)	
Duration of CKD (years)		3.40±1.27	4.80±1.17	<0.001*
Duration of dialysis (years)		--	2.85±1.14	--

Significant differences were seen between the tested groups in terms of serum creatinine, blood urea, eGFR, PTH,

calcium (Ca), phosphorus, Vitamin D, sodium (Na), potassium (K), and ALP levels ($p < 0.001$). Table 2.

Table 2: Evaluation of the two groups based on renal function and biochemical testing

	Group I (n = 700)	Group II (n = 700)	P
Renal Functions			
Serum creatinine (mg/dL)	3.75±1.32	8.52±2.76	<0.001*
Blood urea (mg/dL)	103.9±25.25	156.7±54.60	<0.001*
eGFR (ml/min/1.73 m ²)	26.83±18.13	6.21±3.08	<0.001*
Biochemical analysis			
PTH (pg/ml)	92.0 (76.0-148.0)	210.0 (74.2-456.0)	<0.001*
T. Calcium (mg/dl)	8.35 (7.80-8.70)	7.90 (7.60-8.50)	<0.001*
Phosphorus (mg/dl)	5.45 (4.50-5.70)	5.60 (5.0-6.50)	<0.001*
Ca*Phosphorus	42.63 (39.56-45.60)	44.86 (40.18-50.32)	<0.001*
25 (OH) Vit.D (ng/ml)	21.50 (13.0-35.0)	19.0 (11.0-27.0)	<0.001*
Na+ (mmol/L)	136.99±2.05	137.57±3.70	<0.001*
K+ (mmol/L)	5.04±0.49	5.15±0.74	<0.001*
ALP (IU/L)	106.44±30.16	161.13±55.90	<0.001*

Data are presented as mean ± SD or median (IQR) or frequency (%), eGFR: estimated glomerular filtration rate, PTH: Parathyroid hormone, ALP, ESRD: End-Stage Renal Disease.

The prevalence of high bone turnover was significantly higher in the ESRD patients than the CKD patients (42.9% versus 1.4%, $p < 0.001$). On the other hand, the prevalence of low bone turnover was significantly higher in the CKD patients than the ESRD patients (78.6% versus 35.7%, $p < 0.001$). Table 3

Table 3: Evaluation of the two groups compared using BMD

	Group I (n = 700)	Group II (n = 700)	P
High turnover	10 (1.4%)	300 (42.9%)	<0.001*
Normal	140 (20.0%)	150 (21.4%)	
Low turnover	550 (78.6%)	250 (35.7%)	

The relation between different categories of BMD, demographic characteristics, serum creatinine, blood urea and eGFR of the included patients, there were a significant difference between different categories of BMD in terms of age and sex ($p < 0.001$). The relation between different categories of BMD and renal functions of the included patients. The relation between different categories of BMD and CKD characteristics of the included patients causes of CKD and duration of CKD ($p < 0.001$), and there was no significant difference between studied groups in terms of duration of dialysis ($p = 0.651$). Table 4.

Table 4: Relation between BMD and demographic data, cause, duration of CKD, duration of dialysis and renal functions

		BMD			Test of Sig.
		High Turnover (n = 310)	Normal (n = 290)	Low Turnover (n = 800)	
Age (years)		58.16±6.16	55.38±6.30	54.89±10.07	H=23.758* (<0.001*)
		P1=0.475, P2<0.001*, P3<0.001*			
Sex	Male	180 (58.1%)	160 (55.2%)	580 (72.5%)	$\chi^2=38.705^*$ (<0.001*)
	Female	130 (41.9%)	130 (44.8%)	220 (27.5%)	
		P1<0.001*, P2=0.007*, P3=0.001*			
Cause of CKD	D.M	90 (29.0%)	130 (44.8%)	290 (36.3%)	$\chi^2=34.049^*$ (<0.001*) H=101.97* (<0.001*) H=0.860 (0.651)
	HTN	190 (61.3%)	120 (41.4%)	370 (46.3%)	
	Others	30 (9.7%)	40 (13.8%)	140 (17.5%)	
		P1>0.05, P2>0.05, P3>0.031*			

Duration of CKD (years)	4.79±1.19	4.02±1.41	3.86±1.40	H=101.97* (<0.001*)
	P1<0.001*, P2<0.001*, P3=0.052			
Duration of dialysis (years)	n=300	n=150	n=250	H=0.860 (0.651)
	4.79±1.19	4.02±1.41	3.86±1.40	
P1>0.05, P2>0.05, P3>0.05				
Renal Functions				
Serum creatinine (mg/dL)	8.0 (6.50-9.60)	6.0 (5.30-8.40)	4.20 (3.0-5.95)	369.71* (<0.001*)
	P1<0.001*, P2<0.001*, P3<0.001*			
Blood urea (mg/dL)	164.0 (123.0-192.0)	136.0 (97.0-160.0)	110.0 (90.0-130.0)	217.76* (<0.001*)
	P1<0.001*, P2<0.001*, P3<0.001*			
eGFR (ml/min/1.73 m ²)	4.0 (3.0-6.0)	8.0 (4.0-16.0)	14.50 (7.0-39.0)	406.52* (<0.001*)
	P1<0.001*, P2<0.001*, P3<0.001*			

Data are presented as mean ± SD or median (IQR) or frequency (%), P1: p value for relation between high turnover and normal, P2: p value for relation between high turnover and low turnover, P3: p value for relation between normal and low turnover, CKD, DM: diabetes mellitus, HTN: hypertension, eGFR.

Age, blood urea, serum creatinine, serum phosphorus, ALP, and serum PTH levels were shown to be strongly positively correlated with one another (r = 0.122; p< 0.001), as were

serum creatinine, serum phosphorus, ALP, and serum phosphorus respectively. Table 5.

Table 5: Correlation between PTH and different parameters in total sample

	N=1400	
	PTH	
	r _s	p
Clinical factors		
Age (years)	0.122	<0.001*
Duration of CKD (years)	0.158	<0.001*
Renal Function		
Blood urea (mg/dL)	0.402	<0.001*
Serum creatinine (mg/dL)	0.467	<0.001*
Laboratory parameters		
Phosphorus (mg/dL)	0.549	<0.001*
ALP (IU/L)	0.595	<0.001*
Na+ (mmol/L)	0.032	0.234
K+ (mmol/L)	0.244	<0.001*

r: Spearman coefficient, *: significant at p≤0.05, eGFR, ALP: Alkaline phosphatase

There was a significant negative correlation between serum PTH and eGFR (r = -0.558; p<0.001) and serum Ca (r = -

0.646; p<0.001). Figure 1.

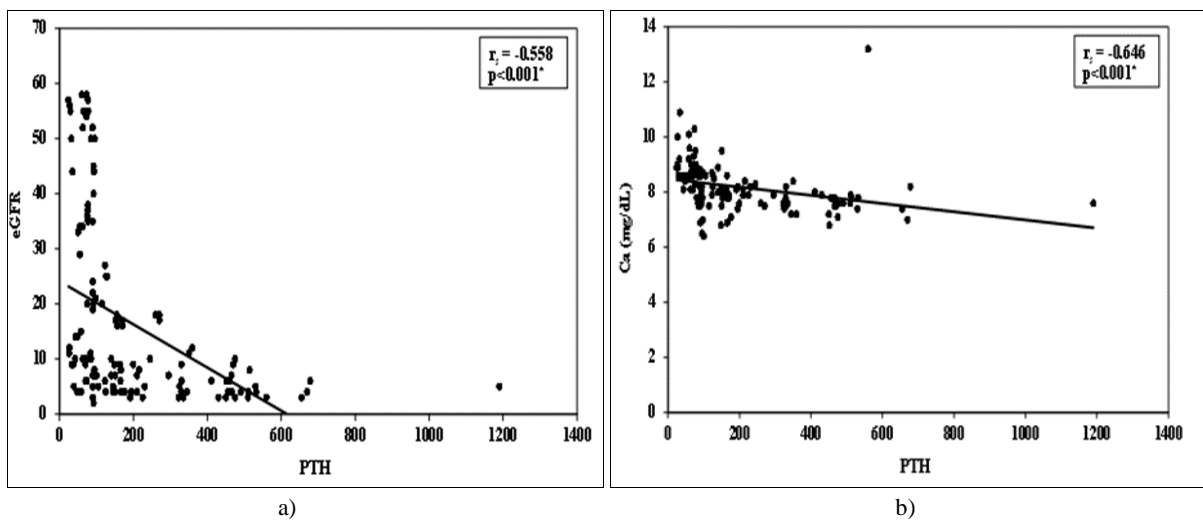


Fig 1: Correlation between PTH and estimated glomerular filtration rate (eGFR) and serum calcium in total sample

Discussion

CKD is a significant public health issue and the most common ailment among older individuals, affecting around 8-16% of the global population [15]. Renal function decline lasting over 3 months is the defining characteristic of this

syndrome, which is categorized based on the extent of kidney damage, with ESRD being the most severe manifestation. Individuals with CKD have much higher rates of illness and death compared to those without CKD. These include higher rates of cardiovascular-related death,

anemia, malnutrition, bone mineral disorders, and cancer [16].

As CKD advances and kidney function declines, a group of compounds termed uremic retention solutes start to build up in the body. Among these molecules, there are those that have harmful effects on the body and are referred to as uremic toxins. These factors are believed to have a role in causing inflammation, immune system dysfunction, vascular disease, platelet dysfunction, and an increased risk of bleeding. They also contribute to an imbalance in the gut's microbial community, leading to an increased movement of bacteria, as well as changes in how drugs are processed by the body. Additionally, they contribute to the development of CKD [17].

On the contrary, Bone Mineral Disturbance (BMD) is a significant public health issue in patients with late-stage renal illness undergoing regular haemodialysis. CKD-MBD is characterized by changes in bone structure in individuals with CKD, those with severe kidney failure, and those on haemodialysis. The blood calcium, phosphorus, ALP levels, and PTH measures are used for the purpose of evaluating, diagnosing, and guiding the therapy [18].

The two primary bone abnormalities in CKD are high turnover hyperparathyroidism and low turnover bone disorders. The serum PTH level is a reliable screening technique for distinguishing between these two illnesses [19].

A bone biopsy is necessary to get a conclusive diagnosis of renal bone disease [20]. The invasiveness, high expense, and general complexity of the treatment have led to its withdrawal from clinical practice. The measurement of serum PTH has traditionally been regarded as the primary biochemical indicator for diagnosing and monitoring the treatment of renal bone disease [21].

The liver plays a crucial role in the process of converting Vitamin D into its active metabolites by hydroxylation. Vitamin D and PTH control the amounts of minerals in the blood and the process of bone restructuring. An imbalance in the endocrine calcium-PTH-Vitamin D axis seems to contribute to the development of osteometabolic disorder [22].

The study included male volunteers with a mean age of around 55 years. Consistent with our findings, Ghonemy *et al.* [23] surveyed 15 dialysis centers at public hospitals in El Sharkia, Egypt, to assess the prevalence and risk factors of chronic kidney disease (CKD). Of the 1,004 patients selected, 62.2% were male and 37.8% were female. The patients had an average age of 52.03 ± 14.67 years. Sixteen percent of end-stage renal disease patients lived in rural regions. Diabetes and hypertension were identified as the predominant etiologies of CKD in the current investigation. Consistent with our research, Lang *et al.* [24] investigated the relationships between serum albumin levels and the progressive decline in renal function as assessed by eGFR using a cohort study in the Health Aging and Body Composition Study. High blood pressure and diabetes were the main causes of CKD.

In this report, we found the relation between studied groups and other biochemical analysis of the included patients. There was a significant difference between studied groups in terms of serum PTH ($p < 0.001$), serum Ca ($p < 0.001$), serum phosphorus ($p < 0.001$), and Vitamin D ($p < 0.001$). Patients with ESRD (on hemodialysis) had higher serum PTH, serum phosphorus, and lower serum Ca and Vitamin D levels than non-hemodialysis dependent CKD patients. In

agreement with our findings. Aggarwal and colleagues [25] enrolled a total of 75 adult patients. According on their GFR, the patients were divided into three groups. Vitamin D, albumin, calcium, phosphate, alkaline phosphatase, and serum creatinine were measured at baseline. The average blood phosphate, ALP, and iPTH values exhibited a consistent upward trend as the progression of CKD occurred. Conversely, CKD patients saw a gradual decline in both mean adjusted blood calcium and Vitamin D levels. Okoye and his colleagues [3] Attempted to determine the prevalence of markers suggesting CKD-MBD in persons who had not yet started dialysis. The researchers evaluated a total of 168 individuals, including 85 persons diagnosed with CKD and 83 healthy controls. The University of Nigeria Teaching Hospital in Enugu had kidney centers and an outpatient medical area where these people were going. We found out the GFR and checked the levels of calcium, phosphorus, ALP, PTH, and 25-hydroxyvitamin D (25 (OH) D) in the blood. The CKD patients had elevated levels of blood PTH and serum phosphorus, as well as decreased levels of serum Ca and Vitamin D compared to the control group. Hyperkalemia, defined as a blood K level of 5.5 mmol/L or more, is often seen in individuals with end-stage renal disease (ESRD). The occurrence of hyperkalemia in individuals undergoing dialysis is believed to be between 5 and 10%. In the present study, patients with ESRD (hemodialysis dependent) had higher serum K and serum ALP than non-hemodialysis dependent CKD patients. Likewise, Nakhoul and colleagues [26] conducted research to examine the connections between K+ problems, mortality, and progression to ESRD in a population with CKD. A total of 36,359 individuals with an eGFR, or eGFR, below 60 ml/min/1.73 m² and K+ levels assessed between January 1, 2005, and September 15, 2009, were identified using the electronic health record-based CKD registry. Those diagnosed with ESRD had elevated levels of serum K+ compared to those in the early stages of CKD. Hsieh and colleagues [27] performed a cross-sectional investigation to examine the blood K + level in patients with CKD in the late stages (stages 3-5) who did not show any clinical signs of hyperkalemia. There were 531 individuals diagnosed with advanced stage CKD. Renal function impairment in the late stages of CKD was accompanied by an increase in the average K+ level ($p < 0.05$). Concerning the primary outcomes of the present study, the prevalence of high bone turnover was significantly higher in the ESRD patients than the CKD patients (42.9% versus 1.4%, respectively; $p = 0.001$). On the other hand, the prevalence of low bone turnover was significantly higher in the CKD patients than the ESRD patients (78.6% versus 35.7%, respectively; $p = 0.001$). In line with our findings, Pan and colleagues [28] reported that the prevalence of decreased BMD was 62.9%. According to the Second annual report of the Egyptian Society of Nephrology in 1997, a countrywide study in Egypt revealed that renal bone disease is present in 33.3% of dialysis patients. In research conducted by Buargub *et al.* [29], it was shown that 28.1% of the patients exhibited laboratory evidence of hyper parathyroid bone disease, whereas 27% of the patients showed poor turnover bone disease. This resulted in a total prevalence of 55.3% for BMD abnormalities. Agarwal [25] it was shown that 39.4% of patients diagnosed with stage 5 CKD had hyperparathyroidism. In a study conducted by Jabbar *et al.* [30] on Indian patients with CKD stages 4 and 5, it was

shown that 60% of the patients had hyperparathyroidism. The researchers used a threshold value of iPTH>300 pg/mL for both stages.

One potential limitation of our investigation was the comparatively tiny sample size. The research was conducted at a single facility. Consequently, we advise that renal bone disorders be anticipated in all patients with chronic renal failure. Regular monitoring of serum concentrations of calcium, phosphate, ALP, vitamin D, and PTH is recommended. Management of hyperparathyroidism should be an integral component for patients with chronic renal failure.

Conclusion

In Gharbia Governorate, BMD is prevalent in CKD. 80% of the patients with non-dialyzable CKD and ESRD had low and high bone turnover.

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Conflict of Interest: Nil.

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