SRYAHWA PUBLICATIONS

Archives of Nephrology
ISSN: 2639-3573 | Volume 7, Issue 2, 2025
https://doi.org/10.22259/2639-3573.0702003

RESEARCH ARTICLE

Mineral and Bone Disorders during Chronic Kidney Disease

Hamat IBRAHIM[1*], Charfadine SENOUSSI[2], Mahamat Abderamane ZALBA[2], Youssouf DJIBRIL[1], Nassima HISSEIN[1], Djibrine MAHAMAT DJIBRINE[1] , Haoua YOUSSOUF SEID[1] , Zahra ELTIGANI AHMED[1]

[1]Nephrology Department, CHU la Référence Nationale, Ndjamena, Chad.
[2]Nephrology Department, CHU "la Renaissance", Ndjamena, Chad.

Received: 10 July 2025 Accepted: 21 July 2025 Published: 04 August 2025
Corresponding Author: Hamat IBRAHIM, Nephrology Department, CHU la Référence Nationale, Ndjamena, Chad.

Abstract

Background: The objectives of our work were to describe mineral and bone disorders in patients with chronic kidney disease not yet at the dialysis stage and to assess the compliance of phosphocalcium balance indicators according to international KDIGO recommendations.

Methods: This was a cross-sectional, descriptive and analytical study conducted over a 10-month period in CHU la Renaissance in N'Djamena. We included all patients followed in the service for non-dialyzed CKD, aged over 18 years.

Results: 387 patient files were collated, 82 of which were retained. The mean age was 52.7 years (± 15.8), with a male predominance (67.1%). Comorbidities were arterial hypertension (71.9%) and diabetes (24.4%). Clinical signs were diffuse bone pain (37.8%), pruritus (8.5%), muscle pain (7.3%) and insomnia (8.5%). CKD predominated in stage V (55%), followed by stage III (23%). Hypocalcemia and hyperphosphatemia were noted in stage IV (11.9% and 10.7%) and stage V (40.5% and 34.5%). Hyperparathyroidism and 25 OH vitamin D deficiency were 28.8% and 40.5%. Compared with international KDIGO recommendations, compliance was 59.7%, 53.7% and 37% respectively for calcium, phosphorus and parathyroid hormone levels. Radiologically, pathological fracture features were observed in 4.9%. Management was based on calcium carbonate (52.4%), native vitamin D (64.6%) and Cinacalcet (6.1%).

Conclusion: The balance of mineral and bone disorders is far from being achieved for the majority of patients, due to lack of resources and the exorbitant cost of treatment. Prevention of BMD is a fundamental pillar of CKD treatment for our patients.

Keywords: Mineral and Bone Disorders, CKD, CHU- la Renaissance, Chad.

1. Introduction

Mineral and bone disorders (MBSD) according to KDIGO is a systemic disorder of mineral and bone metabolism secondary to chronic kidney disease (CKD) manifested by one or more of the following: it may be an abnormality in calcium, phosphorus or vitamin D metabolism, or an abnormality in bone turnover, mineralization, bone volume, growth and strength, vascular or soft tissue calcifications [1]. All these disorders have been grouped together by KDIGO (Kidney Disease Improving Global Outcome) under the term "mineral and bone disorders of chronic kidney disease" (TMO-MRC) [2]. These disorders are constant and occur early in stage 3 chronic kidney disease (CKD).

Apart from these biological and endocrine abnormalities, complications are mainly bone-related, with an increased risk of fracture linked to specific bone pathologies such as secondary hyperparathyroidism (HPT II) and adynamic osteopathy (AO), which represent a major cause of morbidity, reduced quality of life, and vascular and tissue calcifications that have a pejorative impact on survival [3].

Citation: Hamat IBRAHIM, Charfadine SENOUSSI, Mahamat Abderamane ZALBA, *et al*. Mineral and Bone Disorders during Chronic Kidney Disease. Archives of Nephrology. 2025; 7(2):16-20.

From the above, phosphocalcic disorders therefore logically accompany any advanced and untreated renal failure, but they can occur in the absence of renal failure [4]. Their metabolic, bone and, above all, cardiovascular consequences justify prevention and treatment based on pathophysiological knowledge and international recommendations [5].

In Chad, no specific study of the phosphocalcic profile of CKD patients not on dialysis has been carried out. In view of the TMO problem, the objectives of our work were to describe the mineral and bone disorders of patients with chronic kidney disease (CKD) not yet at the dialysis stage, and to assess the compliance of phosphocalcium balance indicators with international KDIGO recommendations.

2. Patients and Methods

This was a cross-sectional, descriptive and analytical study carried out over a 10-month period (01 November 2018 to 31 August 2019) in the nephrology and hemodialysis department of CHU la Renaissance in Ndjamena. We included all patients followed up in the service for non-dialyzed stage 3,4, and 5 CKD, aged over 18 years who had performed a phosphocalcic assessment, and consented to participate in this study. For each patient included, sociodemographic variables (age, sex, level of education, profession), comorbidities or medical history, clinical, paraclinical and therapeutic variables were collected.

Phosphocalcic tests included: calcemia, phosphatemia, parathyroid hormone and vitamin D. Analyses were performed at the CHU la Renaissance laboratory, using UV colorimetry for calcemia (normal: 2.20 - 2.60 mmol/l) and phosphatemia (normal: 0.80 - 1.60 mmol/l). For Parathyroid hormone (PTH), the VIDAS PTH (1-84) method is a third-generation automated quantitative test for the measurement of the biologically active parathyroid hormone PTH (1-84) in human serum or plasma, the result being given according to KDIGO recommendations: a multiplication factor of two to nine times the upper reference limit of serum PTH concentration for a third-generation kit (normal parathyroid hormone 15-585 ng/l; hyperparathyroidism: > 585 ng/l). 25 OH vitamin D was determined using the VIDAS 25 OH vitamin D (Vit D) method, an automated quantitative test for the determination of total 25 OH vitamin D in human serum or plasma (normal: 30 - 100 µg/l). Creatinemia was determined by the enzymatic method (normal: 70 - 115 µmol/l**).** Renal function clearance was calculated using the MDRD (Modification of Diet in Renal Disease) formula.

Data were collected and analyzed using SPSS version 18.0 (Statistique Package for Social Sciences 18.0) and presented in the form of tables and figures. Quantitative data are expressed as mean ± standard deviation and qualitative variables as percentages. The Chi-square test was used to compare qualitative variables, and the Student's t-test to compare quantitative variables.

3. Results

The study included 387 records of patients with chronic renal failure, 82 of whom were retained. The mean age of patients was 52.7 years (± 15.8), with a male predominance (67.1%). The 41-60 age group accounted for 51.2% (42/80) of cases. Comorbidities were dominated by arterial hypertension (71.9%), diabetes (24.4%) and gout (37.80%), and the association of hypertension and diabetes was 19.5% (16/82). Clinical signs were dominated by diffuse bone pain (37.8%), pruritus (8.5%), muscle pain (7.3%), and insomnia (8.5%). Arterial hypertension was found in 80.48% (66/82) of patients. Mean serum creatinine was 824.7 µmol/l (± 599.5). CKD predominated in stage V (55%). Mean hemoglobin (Hb) was 9.3 g/dl (± 2.4), with anemia present in 98.78% of patients.

Hypocalcemia was moderate to severe in 59.7% of cases, with a mean calcemia of 2.0 mmol/l (± 0.4), while mean phosphatemia was 2.1 mmol/l (± 1.2). Hypocalcemia and hyperphosphatemia were noted in stage IV (11.9% and 10.7%) and stage V (40.5% and 34.5%) (Table I). Hyperparathyroidism and 25 OH vitamin D deficiency were 28.8% and 40.5% in stage V CKD. Hypocalcemia (p=0.005) and hyperphosphatemia (p=0.0001) predominated in stage V patients with a significant difference.

Table I. *Distribution of patients by CKD stage and TMO*

Parameter	Stage IIIa	Stage IIIb	Stage IV	Stage V	p value
Calcemia (< 2.20 mmol/L)	2	3	10	34	0,005
Phosphatemia(> 1.60 mmol/L)	1	5	9	29	0,000
PTH (> 585 ng/L)	1	0	5	24	0,15
Vitamin D (< 20ng/L)	5	7	15	34	0,610

Mean parathyroid hormone was 692.9ng/l (± 478.0), 25 OH vitamin deficiency was moderate to severe in 74.39% (61/82) patients, with a mean of 17.9µg/l (± 7.9). Compared with international KDIGO recommendations, compliance was 59.7%, 53.7% and 37% respectively for calcium, phosphorus and parathyroid hormone levels. Radiological findings (Table II). Pathological fracture features were observed in 4.9% of patients.

Table II. *Biological abnormalities of mineral and bone disorders*

Biological Abnormality	n	%	Mean and Standard Deviation
Calcemia (< 2.20 mmol/L)	49	59,8	2,0 ± 0,4
Phosphatemia (> 1.60 mmol/L)	44	53,65	2,1 ± 1,2
PTH (> 585 ng/L)	30	36,58	692,9 ± 478,0
Vitamin D (< 30ng/L)	61	74,39	17,9 ± 7,9

Management was based on calcium carbonate (52.4%), native vitamin D (64.6%) and Cinacalcet (6.1%). In addition to phosphocalcium supplementation, patients received erythropoietin (47.6%), iron supplementation (73.2%), antihypertensive drugs (87.8%), and antidiabetic drugs (24.4%).

4. Discussion

TMO is associated with a high risk of morbidity and mortality. In our series, the subjects concerned were mainly young adults. The mean age of our patients was 52.7 years (± 15.8), with extremes of 18 and 95 years. The 41-60 age group was the most represented, at 51.2%. In the Democratic Republic of Congo, the mean age was 51.38 (±13.47), and 55 in Morocco [6, 7]. In Africa in general, and in Chad in particular, CKD affects the socially active segment of the population. In the West, however, it mainly affects people over the age of 72 [8]. In our study, men predominated (67.1%). This predominance was 56.14% and 60% respectively in Côte d'Ivoire and Mali [9]. This confirms literature data that CKD affects more men than women [10, 11, 12]. The high proportion of men can be explained by the lower economic level of the female population in our country. Low income is a factor in the loss of hospital follow-up for CKD patients.

Manifestations related to mineral and bone disorders were: diffuse bone pain (37.8%), pruritus (8.5%), muscle pain (7.3%), and insomnia (8.5%). In Mali, Traoré et al reported bone pain in 7.5% of cases, muscle pain in 5.3% and pruritus in 2.6%[13]. This would be the consequence of TMO-MRC, due to the quantitative and qualitative alteration of bone tissue, but also to the increased risk of falls linked to the myopathy, hypoglycemia and peripheral neuropathies observed during uremia, and to orthostatic hypotension.

Stage 5 chronic renal failure in our patients accounted for 55%, these results are similar to data observed by other authors in developing countries [6, 9, 11, 14]. Unlike in developed and emerging countries, where less than 50% of CKD patients were seen at moderate stage [12, 15, 16].

Phosphocalcic disorders are frequently observed in CKD, manifested by abnormalities in serum concentrations of phosphorus, calcium and regulatory hormones. These abnormalities are constant and constitute a cardiovascular risk factor [4]. Hypocalcemia was generally a function of disease severity, with a statistically significant correlation (p=0.005) between these values and CKD stages. This is related to decreased intestinal calcium and phosphate absorption, which increases PTH gene transcription and contributes to secondary hyperparathyroidism [3, 4]. Calcitriol synthesis declines in renal failure as soon as glomerular filtration rate falls below 60 mL/min [3, 4, 17]. Several mechanisms may contribute to the development of hyperparathyroidism in these patients, including: lower plasma calcitriol concentration, which directly stimulates parathyroid hormone synthesis and secretion; hypocalcemia and hyperphosphatemia, which promote parathyroid hormone secretion independently of calcitriol action; and reduced parathyroid hormone secretion as a result of calcemia [17].

In our series, plasma parathyroid hormone concentration increased as CKD worsened, and by the terminal stage of the disease, hyperparathyroidism was observed in 29.26% of patients, although there was no correlation between hyperparathyroidism and bone pain (p=0.470), and pathological fracture (p=0.147). The KDIGO recommends assessing bone turnover, excess or deficiency of which can have consequences for bone (with risk of fracture), mineral metabolism (hypercalcemia, hyperphosphatemia) and cardiovascular calcifications [1]. 25 OH vitamin D deficiency in CKD occurs early, in between 80% and

96% of cases, due to the high serum concentration of FGF-23 [18, 19]. It is associated with the progression of chronic kidney disease, cardiovascular calcifications, arterial stiffness, cardiovascular events and mortality [4]. In our study, 25 OH vitamin D deficiency was moderate to severe in 23.2% and 51.2% of cases. Traoré, D et al had reported 58.1% of cases [13].

Therapeutically, calcium carbonate supplementation was used in 52.4% of cases, with an average dosage of 1.5g per day, and native vitamin D in 64.6% of patients. Five patients (6.1%) received Cinacalcet (anti-hyperparathyroid). Vitamin D supplementation aims to reduce the risk of pathologies associated with deficiency, such as osteomalacia and fractures. In Mali, 17.2% of patients were treated with calcium carbonate and 12.9% with vitamin D [13]. In France, 42.4% received Calcium, 55.4% Vit D, and 19.7% Sevelamer (non-calcium chelator) [12]. The molecules used in our study should be associated with non-calcium binders and calcimimetics in cases where phosphatemia and PTH were highly elevated. Given their availability and cost, our prescription was limited to calcium and vitamin D. Unlike in emerging countries [12, 20], the use of calcium carbonate is falling at the expense of Sevelamer and others.

5. Conclusion

The balance of mineral and bone disorders is far from being achieved for the majority of patients, due to lack of resources and the exorbitant cost of treatment. The subjects concerned are mainly young adult males (67.1%), who in most cases consult us at the terminal stage (55%) of the disease. The functional and vital complications associated with TMO are formidable and disabling. Prevention of TMO is a fundamental pillar of CKD treatment for our patients.

Conflict of Interest

we have no conflict of interest, and the study was not funded

6. References

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