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Bulletin de la Dialyse à Domicile

Mineral and bone disorders in peritoneal dialysis in Rabat (Morocco)

(Troubles minéraux et osseux en dialyse péritonéale à Rabat (Maroc))

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Résumé

Introduction
Les troubles du métabolisme minéral et osseux sont fréquents chez les patients en dialyse. Le but de notre travail est de décrire le profil minéral et osseux des patients en dialyse péritonéale au Maroc dans notre centre, de déterminer la prévalence de l'hyperparathyroïdie dans cette population et de relever les facteurs de risques qui y sont associés.
Matériel et méthode
Il s'agit d'une étude transversale incluant tous nos patients en DP chez qui nous avons analysé les différentes données cliniques, biologiques, radiologiques et thérapeutiques en rapport avec le métabolisme minéral et osseux.
Nous avons défini l'hyperparathyroïdie par une parathormone (PTH) ≥ 600pg/ml et nous en avons déterminé les facteurs de risque en comparant deux groupes : avec et sans hyperparathyroïdie.
Résultats
Nous avons recensé 85 patients dont l'âge moyen était de 49.18±17.28 ans et le sexe ratio de 0.77. L'ancienneté en dialyse était de 33.31±26.68 mois. La médiane de PTH était à 668 pg/ml [34-3800] avec une calcémie à 2,18±0,18 mmol/l, une phosphatémie à 1,74±0,51 mmol/l et une vitamine D à 23.74±11.56 ng/ml. L'hyperparathyroïdie a été retrouvée chez 60% des patients.
Les facteurs de risque d'hyperparathyroïdie relevés dans notre étude sont : l'ancienneté en DP, une PTH élevée avant début de dialyse, et l'hyperphosphatémie. Le court suivi médical avant la dialyse semble jouer un rôle important dans le développement de l'hyperparathyroïdie secondaire.
Conclusion
L'hyperparathyroïdie est le trouble minéral et osseux le plus fréquent dans notre série. Les facteurs corrélés à l'hyperparathyroïdie sont l'ancienneté en dialyse, l'hyperphosphorémie et un taux de parathormone élevé avant le début de la dialyse.

Mots clés : osteodystrophie, dialyse péritonéale, hyperparathyroidie, osteopathie andynamique.

Summary

Introduction
Mineral and bone disorders are common in dialysis patients and are responsible for a higher risk of fractures, cardiovascular events, and mortality. The most common mineral and bone disorder in peritoneal dialysis (PD) is adynamic osteopathy. The aim of our work is to describe the mineral and bone profile of our PD patients, assess the frequency of hyperparathyroidism in this population, and identify the risk factors associated to it.
Material and methods
This is a cross-sectional study that included all our PD patients. We analyzed their clinical, biological, radiological ,and therapeutic data related to mineral and bone metabolism.
We defined hyperparathyroidism by a parathyroid hormone (PTH) ≥ 600pg/ml, and we determined the risk factors of hyperparathyroidism by comparing two groups: with and without hyperparathyroidism.
Results
We collected data from 85 patients with a mean age of 49.18±17.28 years and a sex ratio of 0.77. The dialysis vintage was of 33.31±26.68 months. The median PTH level was 668 pg/ml [34-3800] with a mean calcemia level of 2.18±0.18 mmol/l, a mean phosphatemia level of 1.74±0.51 mmol/l, and a vitamin D level of 23.74±11.56 ng/ml. Hyperparathyroidism was diagnosed in 60% of our patients.
The risk factors of hyperparathyroidism in our study were dialysis vintage, a high PTH level before dialysis, and hyperphosphatemia. The short medical follow-up before dialysis seems to play an important role in the development of secondary hyperparathyroidism.
Conclusion
Hyperparathyroidism was the dominant mineral and bone disorder in our study. Its risk factors were dialysis vintage, hyperphosphatemia, and a high PTH level before the start of dialysis.

Key words : Mineral and bone disorder, Peritoneal dialysis, Secondary hyperparathyroidism, adunamic osteopathy

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INTRODUCTION

Peritoneal Dialysis (PD) is a substitute technique for chronic end-stage renal failure, complementary to hemodialysis and renal transplantation.

Disorders of mineral and bone metabolism are common in dialysis patients and are responsible for an increased risk of fracture, cardiovascular risk, and mortality. Indeed, numerous studies have demonstrated these disorders, and more specifically those linked to hyperparathyroidism, are responsible for extra-skeletal calcifications, in particular vascular and valvular [1-3], and are implicated in the pathogenesis of left ventricular hypertrophy [4], thus increasing cardiovascular risk and the risk of morbidity and mortality [5].

Unlike hemodialysis, the mineral and bone disorder most frequently found in PD is adynamic osteopathy rather than secondary hyperparathyroidism. The absence of data concerning mineral and bone disorders in the Moroccan population on PD encouraged us to study this subset in order to compare our results with data from the literature.

The aim of our work is to:
- Describe the mineral and bone profile of our patients on PD.
- Determine the prevalence of different mineral and bone disorders in our population.
- Identify the risk factors associated with hyperparathyroidism in our patients on PD.

MATERIAL AND METHODS

We conducted a descriptive and analytical monocentric cross-sectional study in the PD unit of the nephrology department of the Academic Hospital Ibn Sina in Rabat (Morocco). We included all prevalent patients on peritoneal dialysis with a duration of PD greater than 3 months.

An operating sheet has been drawn up to study the following parameters:

1 – Demographic and clinical characteristics:

• Age and sex.
• Comorbidities (arterial hypertension (HTA), diabetes).
• Initial nephropathy.
• The duration of medical follow-up at the stage of chronic renal failure (CRI) stages 4 and 5 before starting dialysis.
• Seniority in dialysis.
• The modality of PD.
• The quality of purification (Kt/v and weekly creatinine clearance).
• Clinical symptoms, namely bone pain, arthralgia, and pathological fractures.

2 – Biological parameters related to mineral and bone disorders:

These parameters were recorded on the basis of an average of the last 3 months:
• Calcemia, phosphatemia, vitamin D2-D3 levels, alkaline reserve, alkaline phosphatase.
• The parathormone level (PTH 1-84) before the start of dialysis, 3 months after the start of it, at 1 year, and the current PTH level. The parathormone assay is a micro-particle immunoassay by chemiluminescence on a blood sample in a dry tube.

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3 – Radiological parameters:

• X-rays of the skeleton—namely X-rays of the skull in profile, of the hands, and of the pelvis—in search of the impact of mineral and bone disorders.
• X-rays of the lateral chest or pelvis looking for aortic or iliac vascular calcifications.
• Cervical ultrasound to look for parathyroid nodules or parathyroid hyperplasia.
• Transthoracic echocardiography looking for valve calcifications.

4 – Therapeutic and evolutionary parameters:

This medical treatment consists of calcium chelators of phosphorus, the most widely used of which is calcium carbonate, non-calcium chelators of phosphorus and vitamin D in its two native and active forms, and calcimimetics. Vitamin D3 (cholecalciferol) is administered according to the protocol of our service, which consists of an attack treatment of 3 doses of 100,000 IU spaced one week apart in the event of a vitamin-D deficiency with a level below 15 ng/ml or spaced 2 weeks apart in the event of a vitamin-D deficiency with a rate between 15 and 30 ng/ml. The maintenance treatment is 25,000 IU per week. Our therapeutic target for vitamin D3 is 40 ng/ml. Active vitamin D (alfacalcidol) is prescribed in cases of secondary hyperparathyroidism without hypercalcemia or hyperphosphatemia. Surgical treatment consists of a 7th/8th parathyroidectomy indicated in the presence of symptomatic hyperparathyroidism with hypercalcemia and/or hyperhposphatemia.

We defined hyperparathyroidism by a parathyroid hormone (PTH) greater than or equal to 600 pg/ml, i.e., 9 times the normal PTH value, according to the Kidney Disease Improving Global Outcomes (KDIGO) recommendations. To determine the risk factors for hyperparathyroidism, we defined two groups: group A having a parathormone greater than or equal to 600 pg/ml (PTH ≥ 600 pg/ml) and group B whose parathormone is strictly lower, at 600 pg/ml (PTH < 600 pg/ml).

Hypoparathyroidism was defined by a PTH less than 60 pg/ml.

The various data collected were analyzed using the statistical analysis software SPSS version 20 with the help of a statistician. The results were expressed in numbers and percentages for the qualitative variables. For the quantitative variables, the results were expressed either as a mean and standard deviation for the variables with a symmetrical distribution or as a median for the variables with an asymmetrical distribution. The comparison of our two groups of patients, that with PTH ≥ 600 pg/ml and that with PTH < 600 pg/ml, was made using the one-factor Anova test, and the variables were checked using the tests of Student and Chi-square. Multivariate analysis was done using the Manova test. A value of $p < 0.05$ was considered significant.

RESULTS

I. Characteristics of our population of peritoneal dialysis patients

We identified 85 patients meeting our inclusion criteria whose mean age was 49.18 ± 17.28 years, with extremes of 13 and 86 years. The sex ratio was 0.77, with a female predominance of 56.5% (n=48).

Chronic renal failure is third to diabetes in 17.6% of cases and to nephro-angiosclerosis in 11.8% of cases. The initial nephropathy remains undetermined in 28 patients (32.9%). Other etiologies

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of renal failure are summarized in Figure 1.

The average seniority in PD of our patients was 33.31 ± 26.68 months, with extremes of 5 and

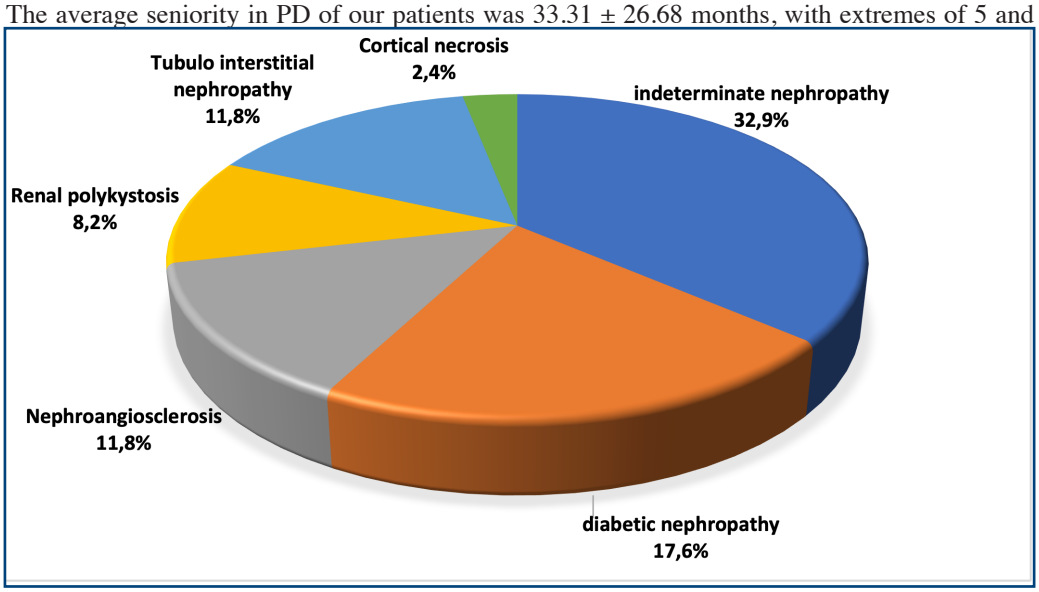

Figure 1. Distribution of patients on PD according to their initial nephropathy

124 months (10.33 years). All our peritoneal exchange solutes were based on glucose at 1.25 mmol/l in calcium and 0.25 mmol/l in magnesium.

The number of patients with a PTH ≥ 600 pg/ml was 51, i.e., a prevalence of 60%, versus 34 patients with a PTH < 600 pg/ml. Two patients (2.35%) had hypoparathyroidism, defined by a PTH lower than 60 pg/ml.

II. Particularities of patients with secondary hyperparathyroidism

The prevalence of hyperparathyroidism in our patients was 60% (n=51). The mean age of the subgroup of patients with a PTH ≥ 600 pg/ml was 47.22 ± 16.78 years, with extremes of 13 and 86 years. The sex ratio was 0.54, with a female predominance of 33 women (64.7%) compared to 18 men (35.3%). The initial nephropathy was dominated in this subgroup of patients by nephroangiosclerosis (15.7%) and diabetic nephropathy (7.8%). Tubulointerstitial nephritis represented 15.7% of cases and polycystic kidney disease 11.8%. Like the initial population, indeterminate nephropathy represented a third of patients (33.3%). The average duration of medical follow-up before the start of PD and during stages 4 and 5 of chronic kidney disease was 46.08 months. The seniority in PD was 39.51 ± 28.81 months, with extremes at 5 and 124 months.

Bone pain was the main clinical symptom, being found in 27.5% of patients. Arthralgia was found in 7 patients (13.7%). Four patients (7.8%) presented pathological fractures due to hyperparathyroidism.

The various biological parameters related to calcium phosphate metabolism are summarized in Table 1.

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Table I. Biological parameters related to the phosphocalcic metabolism of our patients on peritoneal dialysis

Biological parameters	Our population (Mean ± standard deviation)	Patients with secondary hyperparathyroidy (Mezne ± standard deviation)
Number of patients	85	51
Calcemia (mmol/l)	2.18 ± 0.18	2.22 ± 0.20
Phosphatemia (mmol/l)	1.74± 0,5	1.8 ± 0.48
Vitamin D (ng/ml)	23.74 ± 11.56	24.23 ± 11.23
Alcaline phosphatases (UI/l)	214.86 ± 255.81	271.86 ± 299.26
Alcaline reserve (mEq/l)	25.45 ± 2.56	25.25 ± 2.59
Actual parathormone (pg/ml)	940 ± 801	1362.76 ± 779.80
PTH before dialysis (pg/ml)	600 ± 504	787.39 ± 526.18
PTH 3 months after start of dialysis (pg/ml)	540 ± 380	699.40 ± 362.27
PTH 1 year after start of dialysis (pg/ml)	664 ± 451	865.04 ± 421.59

Bone radiological signs related to hyperparathyroidism were dominated by diffuse bone demineralization in 40% of cases, a worm-eaten appearance of the skull in 20% of cases, and resorption of the phalangeal tufts in 17% of cases.

Cervical ultrasound results were normal in 25.5% of patients with hyperparathyroidism. The results also showed a parathyroid nodule in 23.5% of patients and hyperplasia of the parathyroid glands in 5.9%. Transthoracic echocardiography was performed in 56.9% of patients and noted valve calcifications in 2 patients (3.9%). Iliac or aortic vascular calcifications were found in 9 patients with hyperparathyroidism (17.9%).

Therapeutically, calcium supplementation was prescribed for 53% of patients to correct hypocalcemia, calcium phosphorus sequestrants in 61%, and non-calcium chelators in 30% of patients to correct hyperphosphatemia. Native vitamin D was prescribed to 80.4% of patients and active vitamin D to 17.6%. Calcimimetics were used in only one patient in our series (2%), and 7th/8th parathyroidectomy was performed in 9 patients (17.6%). Two other patients are awaiting surgery.

RISK FACTORS ASSOCIATED WITH SECONDARY HYPERPARATHYROIDISM

The comparison between the two groups of patients, group A having a PTH ≥ 600 pg/ml and group B with a PTH < 600 pg/ml, made it possible to identify the factors associated with hyperparathyroidism (table 3).

The main factor associated with hyperparathyroidism in PD was a pathological parathyroid hormone level at the IRCT stage before the start of dialysis (p: 0.001). Seniority in dialysis was also

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an important risk factor (p: 0.015), as well as hyperphosphatemia (p: 0.046). The high level of PAL was significantly associated with hyperparathyroidism (p: 0.007). In a multivariate analysis, only the high PTH level before the start of PD was an independent risk factor for hyperparathyroidism in PD (p: 0.016).

Table II. Factors associated with hyperparathyroidism in peritoneal PD in a univariate analysis

VARIABLES	PTH ≥ 600 pg/ml	PTH < 600 pg/ml	P
Number of patients	51 (60 %)	34 (40 %)	-
Age (years)	47,22 ± 16,78	52,18 ± 17,84	0.081
Sex	18 M / 33 F	19 M / 15 F	0.667
Duration of medical follow-up during the IRC stage	46.08 months	64.68 months	0.17
Parathormone before PD start (pg/ml)	767.59 ± 519.96	260.95 ± 192.47	0.001*
Seniority in DP in months	39.51 ± 28.81	24.00 ± 20.14	0.015*
Phosphoremia (mmol/l)	1.80 ± 0.48	1.61± 0.54	0.046*
Calcemia (mmol/l)	2.22 ± 0.20	2.14 ± 0.14	0.053
Alcaline reserve (mEq/l)	25.25 ± 2.59	25.74 ± 2.52	0.781
Alcaline phosphatase (UI/l)	271.86 ± 299.26	122.94 ± 118.65	0.007*
Vitamin D2-D3 (ng/ml)	24.23 ± 11.23	23.30 ± 12.08	0.759
Number of peritonitis	0.78 ± 1.064	0.59 ± 0.867	0.403

DISCUSSION

Prevalence and risk factors of mineral and bone disorders in PD

At the end of our work, hyperparathyroidism was found to be the most common mineral and bone disorder, appearing in 60% of our patients. Its prevalence is rather variable according to the extant literature. Its prevalence is only 5.4% in a Taiwanese national series conducted over 8 years, which included 12,116 patients on PD [6]. A Mexican series carried out in 2013 that included 365 patients on PD found hyperparathyroidism in 20% of CAPD patients and 32% of APD patients [7]. The Italian ATENA series, which included 378 patients on PD for at least a year, found 30% to have secondary hyperparathyroidism [2]. The study carried out in Singapore, which involved 86 incident patients on PD found hyperparathyroidism in 45.3% of patients 4 months from the start of dialysis [3].

In the same Mexican study cited above, hypoparathyroidism was more frequent, appearing in 56.6% of patients on CAPD and 64.2% on APD [7]. Hypoparathyroidism is indeed the mineral and bone disorder most frequently found in the literature pn PD patients [8]. Adynamic osteopathy was histologically proven in 63.2% of patients in a Spanish series of 57 PD patients [9]. Many studies incriminate diabetes as its main risk factor [7, 8, 10]. Other risk factors include age, iatrogenic suppression of PTH by vitamin D, hypercalcemia, calcium phosphorus chelators, or calcium-rich dialysates [8]. In our series, 2 (2.35%) patients developed hypoparathyroidism. One of these patients was diabetic. The literature correlates hypoparathyroidism with a greater risk of mortality than hyperparathyroidism [6, 10].

Mineral and bone disorders start early in chronic renal failure (CRF). We found that a high level

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of PTH before the start of dialysis is the main risk factor for hyperparathyroidism in dialysis (p: 0.001), hence the interest of the control of mineral and bone disorders during stages 4 and 5 of chronic kidney disease. Medical follow-up during the CKD stage is also essential. Patients who come to consultation at the end stage and patients with a short medical follow-up during CRF develop more hyperparathyroidism than those with a longer follow-up and therefore better control, although the difference was not statistically significant in our study (p: 0.17). The later consultations of our patients and their long history of CKD before starting dialysis could explain the higher rate of secondary hyperparathyroidism in our population of PD patients when compared to the results found in the extant literature.

Dialysis seniority is also significantly related to secondary hyperparathyroidism in our series (p: 0.015). The average seniority of our dialysis patients was 33.31 months, which is consistent with the Mexican series, which had an average seniority in PD of 30.8 months [7]. The Taiwanese study's average seniority was 46.8 months [6].

Another important risk factor for hyperparathyroidism found in our study is hyperphosphatemia (p: 0.046). This is explained by the pathophysiology of hyperparathyroidism, as hyperphosphatemia is the first disorder of mineral homeostasis triggering hyperparathyroidism. Alkaline phosphatases, also significantly elevated (p: 0.007), reflect bone remodeling rather than a consequence of hyperparathyroidism.

The Singapore study found a statistically significant link between hyperparathyroidism and the occurrence of peritonitis in patients on PD [3]. This link, however, was not found in our patients (p: 0.403).

Regarding the demographic profile of the patients, our series included young patients with an average age of 49.18 years, meaning this demographic profile equated roughly to the Mexican series (average age of 48.7 years) [7]. The average age was 52 years in the Taiwanese series [6], 68 years in the Singaporean series [3], and 64.7 years in the Italian ATENA series [2]. In our work, age was not found to be a factor associated with secondary hyperparathyroidism during PD. Young age is, on the other hand, associated with a higher concentration of PTH in the Taiwanese study [6], which could explain the high levels of PTH in our series.

Concerning the sex of the patients, we had a female predominance (56.5%, sex ratio: 0.77). This characteristic aligned our work with the Taiwanese series (sex ratio: 0.85) [6] and contrasted it with the other series where the male sex is predominant (a sex ratio of 1.43 in ATENA [2] and 1.32 in the Mexican series [7]). An English study of 282 PD patients found a significantly higher PTH concentration in women [11], but our study did not find any significant difference between men and women (p: 0.667).

This same English study found higher PTH levels in Afro-Caribbean patients compared to Caucasians and Asians and therefore incriminates ethnicity as a determining factor in the severity of hyperparathyroidism [11].

The initial nephropathy most frequently found in our patients was diabetic nephropathy, which is consistent with national epidemiology. The etiology of nephropathy remains undetermined in a third of cases because patients consult at a late stage.

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COMPLICATIONS OF SECONDARY HYPERPARATHYROIDISM

Secondary hyperparathyroidism was the most common bone and mineral disorder in our series and is responsible for multiple bone and extra-bone complications. One of these main complications is osteitis fibrosa. Indeed, PTH binds to the PTH/PTHrP receptor on osteoblasts and thus indirectly stimulates osteoclast formation and bone remodeling [12]. This strong bone remodeling accentuates bone fragility, which explains bone pain and the increased risk of fractures associated with severe hyperparathyroidism [1, 12]. In the Dialysis Outcomes Practice Patterns Study (DOPPS), intact PTH levels greater than 900 pg/ml were independently associated with the risk of developing a new fracture [13]. This risk is increased in patients with a prolonged uremic state, which is responsible for osteopenia. The risk is also higher in elderly patients who have a greater risk of falls, especially if they are on psychoactive drug molecules [1]. In our patients, skeletal radiographs found bone abnormalities in 45.1% of patients with secondary hyperparathyroidism; these abnormalities were dominated by diffuse demineralization (39.9%), the worm-eaten appearance of the skull (19.6%), and resorption of the phalangeal tufts (17.6%). 7.8% of patients had pathological fractures.

Another important complication of secondary hyperparathyroidism is vascular calcifications, which are associated with cardiovascular mortality [2, 3]. The first mechanism is a passive precipitation of calcium phosphate in the vascular system secondary to hypercalcemia and hyperphosphatemia [8, 14]. The second mechanism is active through hyperphosphatemia, which promotes vascular calcifications by inducing the transformation of vascular smooth muscle cells into osteochondrogenic phenotypes and by promoting the mineralization of their cell matrix [3, 8]. At the stage of chronic kidney disease, the uremic state also participates in the formation of vascular calcifications by promoting the elevation of the levels of calcification promoters, such as type-1 collagen or TNFβ, and the reduction of inhibitory factors, such as osteoprotegerin, the Matrix Gla protein (MGP), or fetuin A [1]. Length of time on dialysis, inflammation, and hyperhomocysteinemia are also risk factors for vascular calcifications [8], and vascular calcifications were found in 17.9% of our patients.

In addition to vascular calcifications, insufficient phosphorus control can lead to valvular calcifications, which was found in 3.9% of our patients as well as soft tissue calcifications [1, 3].
Hyperparathyroidism is also implicated in the occurrence of cardiovascular complications and increased cardiovascular mortality [15]. The DOPPS study showed that this risk was greater in patients with a PTH greater than 600 pg/ml [5]. Hyperparathyroidism also plays a role in the pathogenesis of left ventricular hypertrophy. Experimental studies have shown that PTH exerts a direct hypertrophic effect on cardiomyocytes [4]. Several observational studies have shown that the progression of LVH was delayed or even reversed after parathyroidectomy [12].

The increased risk of mortality due to hyperparathyroidism is also due to the high risk of fractures and associated adverse events, such as prolonged immobilization, malnutrition, and infection [12].

Finally, other adverse effects of hyperparathyroidism include emaciation and muscle atrophy [12], worsening of anemia through direct inhibition of erythropoiesis, reduction in red blood cell lifespan [12], and immune dysfunction [12].

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CONCLUSION

At the end of our work we observed that secondary hyperparathyroidism is the most common mineral and bone disorder in our 85 patients (60%).
The factors correlated with hyperparathyroidism in our study are length of time on dialysis, hyperphosphatemia, and a high parathyroid hormone level before the start of dialysis. This reinforces the importance of medical follow-up and early evaluation of phosphocalcium status and treatment of mineral and bone disorders in patients from stages 4 and 5 of chronic kidney disease ; it conditions the subsequent mineral and bone status of patients on dialysis.
In addition to bone complications leading to the risk of fracture, this hyperparathyroidism can be responsible for serious systemic complications, such as extra-skeletal calcifications, including valvular, vascular, and left ventricular hypertrophy involving the vital prognosis of patients.

CONFLICT OF INTEREST

The authors declare no conflict of interest for this article.

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