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Metabolism of the phosphate and calcium balance in chronic kidney disease: Focus in peritoneal dialysis

(Métabolisme du bilan phosphocalcique dans la maladie rénale chronique : Focus en dialyse péritonéale)

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Summary

Patients with chronic kidney disease (CKD) have abnormal phosphocalcic metabolism. Biologically, this translates into hypocalcemia, hyperphosphatemia, secondary or even tertiary hyperparathyroidism, and increased total and bone alkaline phosphatases. The clinical consequences are an increase in morbidity and mortality, with cardiovascular disease in particular, and bone complications secondary to abnormalities in bone remodeling, with a consequent risk of fracture. Only 25% to 50% of peritoneal dialysis (PD) patients have a phosphocalcium balance within the recommended targets. It is essential to correct this. Particular attention must be paid to the choice of calcium concentration in glucose solutions, taking into account the clinical context and favoring low-calcium solutions (concentration at 1.25 mmol/L). Diet and dietary monitoring are recommended as first-line treatment, with calcium-free phosphate binders if necessary. The aim is to avoid hypercalcemia and hyperphosphatemia. Finally, phosphate extraction is more important in continuous ambulatory PD (CAPD) than in automated PD (APD).

Mots-clés : peritoneal dialysis, phosphocalcium metabolism, low-calcium glucose dialysate

Résumé

Les patients atteints de MRC ont une anomalie du métabolisme phosphocalcique. Elle se traduit biologiquement par l'apparition d'une hypocalcémie, d'une hyperphosphatémie, d'une hyperparathyroïdie secondaire voire tertiaire et d'une augmentation des phosphatases alcalines totales et osseuses. Les conséquences cliniques sont une augmentation de la morbi mortalité en favorisant notamment les maladies cardiovasculaires et les complications osseuses secondaires aux anomalies du remodelage osseux favorisant le risque fracturaire. Seulement 25 à 50% des patients en dialyse péritonéale ont un bilan phosphocalcique dans les cibles recommandées. Il est essentiel de le corriger. Une attention particulière doit être portée sur le choix de la concentration en calcium des solutions glucosées en tenant compte du contexte clinique et en favorisant les solutions de faible teneur en calcium (concentration à 1,25 mmol/L). Un régime alimentaire et un suivi diététique sont recommandés en première intention avec au besoin des chélateurs de phosphate sans calcium. Le but étant d'éviter l'hypercalcémie et l'hyperphosphatémie. Enfin l'extraction du phosphate est plus importante en DPCA qu'en DPA.

Keywords : dialyse péritonéale, métabolisme phosphocalcique, dialysat glucosé à faible teneur en calcium

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Introduction

Biological abnormalities of the phosphorus–calcium balance are a constant feature of chronic kidney disease (CKD), appearing as early as stage 2 and leading to parathyroid gland hyperplasia, bone complications (specific and aspecific), and cardiovascular complications. Together, these are known as the mineral and bone disorders of CKD. The aim of this article is to describe these abnormalities in patients with stage 5 CKD (CKD5) treated with peritoneal dialysis (PD). We begin with a brief review of phosphocalcic metabolism in CKD, its clinical and biological consequences, and a brief reminder of a few recommendations. We will then discuss the clinical consequences of these abnormalities in PD patients and the impact of dialysate bag calcium concentration on these abnormalities.

1) Phosphocalcium metabolism in chronic kidney disease

1-a) Pathophysiology: A reminder

• Hyperphosphatemia

At the steady state, inorganic phosphate is filtered by the glomeruli. Around 70% to 80% is reabsorbed by the cotransporter (Npt2a) in the proximal tubules and 20% to 30% in the distal tubules. This reabsorption depends not only on phosphatemia but also on the serum levels of the parathyroid hormone (PTH), FGF23 (fibroblast growth factor 23), and vitamin D. In CKD, from stage 2 onward, phosphate homeostasis is impaired. When the glomerular filtration rate (GFR) falls below 30 ml/min, compensatory phenomena are overwhelmed, and hyperphosphatemia develops. Hyperphosphatemia, secondary to the drop in the GFR, leads to hyperplasia of the parathyroid glands (i.e., secondary hyperparathyroidism, either through direct action on parathyroid cells or indirectly via hypocalcemia induced by the formation of a phospho-calcium complex and the drop in calcitriol). The stimulation of PTH via its action on increased bone turnover aggravates hyperphosphatemia.

• 25 OH vitamin D

Native vitamin D (25 OH vitamin D) comes from plants, vitamin D2 and the UV-induced transformation of 7-dehydrocholesterol (a cholesterol derivative) into vitamin D3 (cholecalciferol) in the skin. It is hydroxylated in the liver to 25 OH D2 and D3. Under the regulation of PTH and FGF23, it is hydroxylated to 1 α in the kidney. Native vitamin D deficiency is common, affecting 80% of CKD patients. It alone is associated with the progression of CKD, the occurrence of cardiovascular events, and increased arterial stiffness [1] [2]. It is therefore essential to correct it, all the more so as it is necessary for the formation of 1,25 0H2 vitamin D, which is consistently deficient in CKD patients. Circulating native vitamin D is largely bound to a carrier protein (vitamin D binding protein). Only the free 1% and albumin-bound 10% are bioavailable. The usual dosage includes all 25 OH vitamin D. Classically, deficiency is defined as a concentration below 10 ng/ml, and insufficiency as a concentration between 10 and 30 ng/ml.

• 1,25-OH2 vitamin D deficiency

Deficiency of an alfa hydroxylase, secondary to CKD, enabling the hydroxylation of 25 OH

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vitamin D (calcidiol), leads to the reduced production of 1,25-OH2 vitamin D (calcitriol). This hydroxylation is promoted by PTH and inhibited by FGF23. The result is the reduced absorption of calcium and phosphate from the digestive tract as well as hyperplasia of the parathyroid glands.

• Secondary hyperparathyroidism

Stimulation of the parathyroid glands by hypocalcemia, hyperphosphatemia, and calcitriol deficiency leads to parathyroid hyperplasia, increasing the blood secretion of PTH. Initially, this is secondary hyperparathyroidism. It is then partly controlled by correction of the factors that promote it (i.e., hypocalcemia, hyperphosphatemia, and/or calcitriol deficiency). At an advanced stage, correction of these factors no longer enables it to be controlled, and it becomes tertiary hyperparathyroidism, associated with at least the appearance of a parathyroid adenoma. In this case, it is associated with hypercalcemia and is no longer medically controlled. PTH influences bone remodeling, with the increased release of calcium from bone into the blood and the increased production of FGF23 by fibroblasts.

• FGF23 and klotho

FGF23 is a hormone secreted by osteoblasts and osteocytes, inhibiting phosphate reabsorption in the proximal tubule and suppressing calcitriol synthesis. FGF23 is linked to a co-receptor called klotho, which is deficient in CKD. In stage 5 CKD, this deficiency leads to an increase in serum phosphate despite elevated FGF23.

These mechanisms are summarized in *figure 1*.

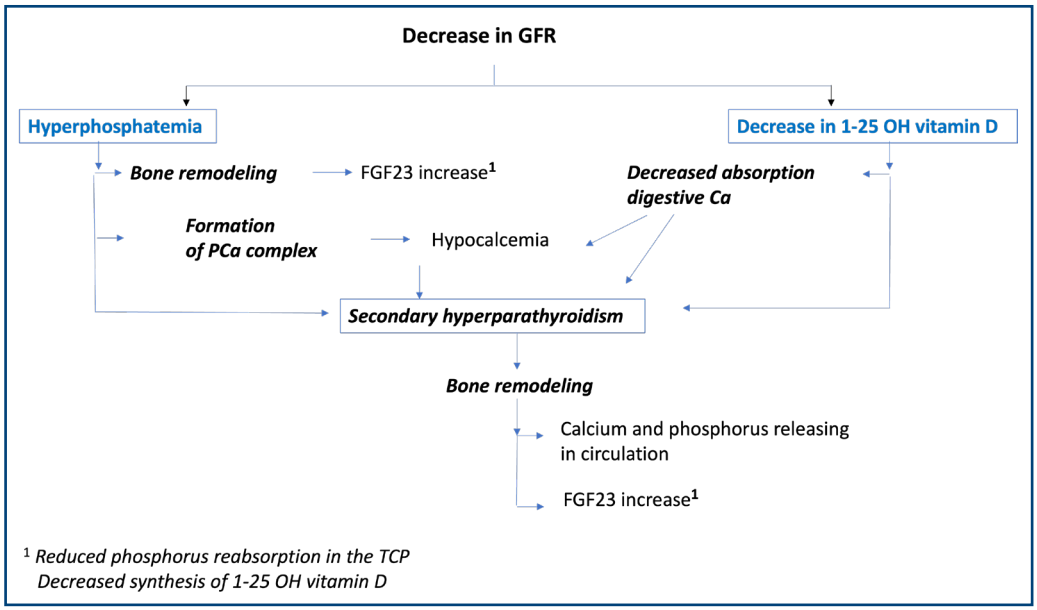

Figure 1. Phospho-calcium balance abnormalities in CKD

1-b) Clinical and biological consequences

The impact of disturbances in phosphocalcium balance during CKD on morbidity and mortality is severe. It is associated with increased mortality, cardiovascular events, and bone complications.

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In fact, this metabolic disorder favors the appearance of calcifications, particularly within the vascular wall, affecting either the intima, favoring vessel obstruction, or the media, resulting in increased pulse wave velocity, increased arterial stiffness, and left ventricular hypertrophy. The intensity of calcification and its impact on morbidity and mortality can be assessed using calcium scores, such as the Framingham or Kauppila scores. Calcifications can also reach the vessels of the hypodermis and induce a severe pathology known as calciphylaxis, which is also favored by the use of antivitamin K. In this case, painful, deep, digging ulcerations, usually proximal, appear. They are usually complicated by superinfections and malnutrition. The mortality rate is over 50%, depending on the series.

Bone complications are also frequent and severe. They include lesions associated with secondary hyperparathyroidism, adynamic osteopathy, and/or osteomalacia. Lesions associated with osteoporosis or aluminic osteopathy have also been described. Together, these lesions quadruple the risk of fracture in MRC5D patients. Trabecular bone biopsy of the iliac crest, which was used to describe and classify bone lesions in renal disease, is no longer routinely recommended because it is invasive. However, it is still recommended in cases of pathological fractures, hypophosphatemia, unexplained hypercalcemia, and suspected aluminic intoxication. Similarly, standard bone X-rays are no longer routinely performed in follow-up [3].

Biological manifestations of abnormal phosphocalcic balance include hypocalcemia, hyperphosphatemia, elevated PTH, and a 1,25-OH2 vitamin D deficiency.

It is recommended to measure total calcium and phosphorus simultaneously at least once a month. PTH levels should be checked with the same kit every three months, more frequently in the case of active treatment of secondary hyperparathyroidism. Serum 25 OH vitamin D should be measured before the start of dialysis and then once a year with an interval of more than two weeks after a dose of native vitamin D. The assay may be repeated every three to six months in the event of elevated PTH or when monitoring native vitamin D therapy. Total alkaline phosphatase should also be measured every three to six months. It should be noted that total alkaline phosphatases are elevated in bone diseases (hyperparathyroidism, liver metastases, osteomalacia) and liver diseases. Serum bone alkaline phosphatase levels depend in part on osteoblast activity and hepatic degradation. BAP levels above 20 g/l are associated with hyperparathyroidism and below 9 g/l with adynamic osteopathy. Other bone markers are bone collagen fragments. The most common are the carboxy-terminal cross-link telopeptide fragments of bone collagen (cross-laps or CTX), released in osteoclastosis. P1NP, osteocalcin, and TRAP5b are also markers of bone remodeling but are not routinely used.

The recommendations were updated in 2017 to correct these anomalies [3]. The biological targets

⬇ *Table I. Biological values of the Phospho-calcium balance recommended by the KDIGO 2017 [1] in MRC5D patients*

Biological parameters	**Dialysis targets**
Total calcium	Laboratory standards (2.1-2.55 mmol/L)
Phosphatemia	"Towards" the standard
Serum PTH concentration	Two to nine times upper laboratory limit
Serum total alkaline phosphatase concentration	Laboratory standards
Serum bone alkaline phosphatase concentration	9-20 µg/L
25 hydroxy vitamin D2 or D3	30-50 µg/L

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to be achieved are in *table 1*. Among others, it can be noted that for patients at the MRC3A to 5D stage, it is recommended to avoid hypercalcemia (2C), to favor calcium-free phosphate binders (2B), and for patients at the MRC5 D stage to choose a calcium-poor dialysate with a calcium concentration of 1.25 mmol/L to 1.5 mmol/Lin hemodialysis (HD) [1].

2) Phosphocalcium balance abnormalities in peritoneal dialysis

2-a) Are the recommended biological targets achieved in peritoneal dialysis?

The impact of dialysis mode on phosphocalcium balance is controversial and poorly described. A recent study carried out in the United States from 2007 to 2011 on 132,523 incident MRC5-D patients compared phosphocalcium balance according to dialysis mode (in-center HD three times a week, PD, long nocturnal HD, and home HD). Patients on long nocturnal HD had significantly lower PTH levels than patients on chronic thrice-weekly in-center HD, while patients on PD and daily home HD had significantly higher levels [4].

The percentage of dialysis patients whose phosphocalcium status is within recommended targets is poorly studied in HD and PD. Two studies have attempted to answer this question based on the 2009 KDIGOs. The first is a prospective multicenter study based on the NECOSAD cohort (1997–2004). Its main objective was to assess the adequacy of phosphocalcic status at three months in incident PD and HD patients. A total of 586 PD patients and 1,043 HD patients were included. The percentage of patients within the PD targets was low: 29% for calcemia, 50% for phosphate, 58% for phosphocalcium product, and around 25% for PTH levels. The same applies to HD [5].

Another study confirms this result. This time, it comes from the BRAZPD II cohort of PD patients, which includes 65%–70% of prevalent patients in Brazil. The aim of this prospective observational study, which ran from 2004 to 2011, was to assess phosphocalcium levels one year after the start of PD treatment . At one year, 50.4% of the patients were within calcium targets, 54.7% within phosphatemia targets, and 28.5% within PTH targets [6].

Finally, recently, the prospective multicenter observational pDOPPS study, including adult PD patients from 2014 to 2022, reported that only 25% to 33% of patients had PTH levels within recommended targets, 38% to 62% for serum calcium [7], and 37% of patients had phosphatemia above 5.5 mg/dl [8] with significant international variability.

We can conclude that the recommended targets are achieved in only 25% to 50% of cases. Correcting this disorder is difficult. This can be partly explained by the heterogeneity of clinical practices. This was highlighted in an Italian multicenter survey of 107 nephrologists in 2022 [9]. On the other hand, non-compliance can also be attributed to frequently reported drug intolerance.

2-b) What is the impact of phosphocalcium metabolism on morbidity and mortality in PD?

- Impact on mortality

The impact of abnormal calcium–phosphate balance on mortality has been demonstrated mainly in four studies.

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The leading study is by Rhee et al. [10], from the American registry of 9,244 incident patients. Patients were included from 2001 to 2006 and followed up until 2019. There is a linearly increased risk of all-cause mortality with alkaline phosphatase levels above 150 U/L. The odds ratios and confidence intervals are 1.18 [1.03–1.36], 1.27 [1.08–1.50], 1.49 [1.23–1.79], and 1.35 [1.19–1.53] for alkaline phosphatase concentrations of 150–170 U/L, 170–190 U/L, 190–210 U/L, and over 210 U/L, respectively [10]. The risk of mortality according to PTH level describes a U-shaped curve, with an excess risk when PTH levels are below 200 pg/ml and above 700 pg/ml. The odds ratios and confidence intervals are 1.25 [1.12–1.41], 1.12 [1.02–1.23], 1.06 [0.96–1.18], 1.09 [0.97–1.24], 1.12 [0.97–1.29], 1.18 [0.99–1.40], and 1.23 [1.09–1.3] for PTH concentration levels of less than 100 pg/mL, 100–200 pg/mL, 300–400 pg/mL, 400–500 pg/mL, 500–600 pg/mL, 600–700 pg/mL, and more than 700 pg/mL, respectively [10].

These results were confirmed in the following three studies.

The first is a Chinese retrospective single-center study including 1,662 incident continuous ambulatory PD (CAPD) patients from 2006 to 2013. The results show an increased risk of all-cause and cardiovascular mortality when patients have phosphatemia below 1.13 mmol/L or well above 1.78 mmol/L (OR: 1.818, 95% CI [1.379–2.396] and 2.069, 95% CI [1.428–2.998]), a phosphocalcic product greater than 55 mg /dl22 (OR 1.735, 95% CI = [1.261–2.386] and 2.175, 95% CI [1.450–3.262]), and a calcemia lower than 2.1 mmol/L. Each mmol/L of albumin-corrected serum calcium reduced the risk of all-cause mortality by 14.3% [0.749–0.981] [11].

In the second study, the aim was not only to assess the impact of phosphocalcic status on cardiovascular and all-cause mortality but also on diuresis and transfer to HD. The results show that hypercalcemia, hyperphosphatemia, and hyperparathyroidism are associated with an increased risk of mortality. Hypocalcemia is associated with a more rapid decline in residual renal function. Transfer to HD is less frequent when phosphatemia is normal or low and serum PTH levels are low (<200 pg/ml) [12].

Finally, the strength of the third study (pDOPPS) is that it is international. The increased risk of cardiovascular and all-cause mortality in cases of hyperphosphatemia, PTH levels below 300 pg/ml and above 599 pg/ml, and hypercalcemia was also found [8] [7]. The extent of vascular calcifications impacts mortality not only in the general population but also in PD patients. A European prospective observational multicenter study from 2009 to 2013 including 269 PD patients showed increased mortality risk in patients with a severe Kauppila score [13].

FGF23 levels are increased in CKD and are associated with excess mortality [14]. FGF23 levels are lower in PD than in chronic in-center HD [15]. A retrospective single-center study of patients treated with CAPD from 2005 to 2011 assessed the impact of FGF23 levels on mortality. Two hundred and five patients were included. The risk of mortality was higher in patients with high levels (FGF23 > 119 RU/ml), with an OR 2.87 95% CI [1.06–7.76] [16]. In a more recent study, FGF23 was an independent risk factor for cardiovascular events in 270 CAPD patients [17].

• Impact on bone complications

Abnormalities in the phosphocalcium balance have an impact on bone remodeling, fracture risk, and mortality. The risk of fracture is lower in PD than in HD [18]. Few studies have described

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bone damage in PD patients. However, the first showed that in 57 patients treated with PD, most had adynamic osteopathy (63.2%) and that plasma PTH levels had a good positive predictive value for adynamic osteopathy at less than 150 pg/ml and for bone hyper-remodeling at more than 450 pg/ml. In the same article, PTH stimulation by hypocalcemia was greater in adynamic bone than in hyper-remodeling bone (166.4% ± 134% vs. 83.5% ± 73.6%) [19].

Furthermore, after one year of dialysis, bone demineralization was greater in incidental HD patients (n = 104) than in PD patients (n = 138). This bone demineralization is associated with an increased risk of mortality [20]. It is now recommended to perform biphoton X-ray absorptiometry in patients with suspected bone demineralization and fracture risk so as to initiate preventive treatment. A bone biopsy may be necessary to assess bone turnover and determine the appropriate treatment. Such management requires close collaboration with rheumatologists [3].

- Impact on peritoneal infections

Abnormalities in phosphocalcic balance have an impact on the occurrence of infections.
In general, age, level of inflammation, and nutritional status influence PTH levels in patients with CKD. PTH levels are implicated in the dysregulation of the immune response to infection. Thus, a PTH level below 150 pg/ml is associated with an independent risk of mortality from infectious causes in HD and PD patients [21].

In this Chinese single-center retrospective study with a seven-year follow-up, 270 incident PD patients were included from 2012 to 2018 and grouped according to their PTH level (below 150 pg/ml, between 150 and 300 pg/ml, and above 300 pg/ml). The aim was to investigate the association between risk of peritoneal infection occurrence and PTH level. A PTH level below 150 pg/ml is an independent risk for peritoneal infection (1.643 [1.01–2.6]). Gram-negative infections are significantly more frequent in patients with a PTH level below 150 pg/ml, while gram-positive and germ-free cocci infections are significantly more frequent in patients with a PTH level above 300 pg/ml [22].

Taken together, these studies demonstrate the impact on morbidity and mortality of abnormal calcium-phosphate balance in PD, along with the difficulty of achieving the recommended targets. As this data mainly comes from the American and Asian continents, it would be interesting to conduct European or French studies to confirm these results in our population.

2-c) What is the impact of calcium concentration in dialysate solutions on phosphocalcic metabolism?

The impact of the choice of dialysate calcium concentration on phosphocalcic balance has been well demonstrated in HD [23].

In PD, two calcium concentrations are available for glucose solutions, irrespective of the glucose concentration (i.e., 1.25 mmol/L or 1.75 mmol/L). For solutions containing icodextrin or amino acids as osmotic agents, the calcium concentration is fixed at 1.75 mmol/L and 1.25 mmol/L, respectively. The impact of the choice of calcium concentration on phosphocalcic balance in PD therefore concerns glucose solutions only.

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Glucose solutions with a calcium concentration of 1.75 mmol/L are most frequently used. However, the choice of glucose solutions with a calcium concentration of 1.25 mmol/L is recommended to avoid a positive calcium balance or hypercalcemia (2C) [24].

A recent meta-analysis of four randomized studies and three observational studies (439 patients) shows, despite the heterogeneity of the data, that the use of dialysate solutions with a calcium concentration of 1.25 mmol/L results in a greater reduction in blood calcium levels than solutions with a calcium concentration of 1.75 mmol/L and a greater increase in PTH, with no impact on blood phosphorus levels or on the occurrence of peritoneal infection. Conversely, glucose solutions with a calcium concentration of 1.75 mmol/L induce a greater decrease in PTH levels [25].

However, these changes, linked to variations in the calcium concentration of glucose solutions, were not found in this recent prospective single-center study, in which 20 prevalent PD patients usually treated with solutions containing a calcium concentration of 1.75 mmol/L received treatment with 1.25 mmol/L solutions. On the other hand, there was an increase in the prescription and consumption of calcitriol, which promotes calcium absorption and thus limits hypocalcemia [26]. Finally, the calcium concentration of glucose solutions has no impact on the calcium score at two years [27].

In light of these studies and recommendations, various authors suggest choosing the calcium concentration of the dialysate according to the clinical context. A low-calcium solution (1.25 mmol/L) should be considered in cases of hypercalcemia and/or low PTH and a high-calcium solution (1.75 mmol/L) in cases of hyperparathyroidism. The calcium concentration of the solution should first be chosen according to the biological parameters; then a second step is to prescribe an oral treatment aimed at correcting abnormalities in the phosphocalcic balance, which will limit digestive intolerance and non-compliance.

2-d) What is the impact of peritoneal properties and dialysis mode in PD (CAPD versus APD) on phosphocalcium balance?

Few studies on this subject are available. A single-center observational study to investigate phosphate clearance according to peritoneal properties and dialysis mode shows that phosphate and creatinine have different peritoneal clearances and diffusion. Moreover, phosphate elimination is better in CAPD than in automated PD (APD), whatever the type of peritoneum (fast, slow, medium-slow, or medium-fast transporter), even though hyperphosphatemia is more frequent in the case of slow peritoneum and peritoneal phosphate clearance is poorer. CAPD would therefore be the most suitable dialysis modality in PD in cases of hyperphosphatemia [28].

In conclusion, phosphocalcium balance abnormalities in PD are frequent and associated with significant morbidity and mortality. Unfortunately, few patients achieve the recommended targets. To achieve this, patients need to have an appropriate calcium, phosphate, and protein diet as well as regular dietary monitoring. It is vital to preserve residual renal function. The choice of glucose dialysate solutions according to calcium concentration is essential, as is the type of PD modality (CAPD versus APD) depending on the patient's profile. Finally, this is difficult to do without drug treatments, but their prescription must be preceded or at least accompanied by the preceding measures. Hyperphosphatemia must be corrected, avoiding calcium phosphate

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binders, hypocalcemia must be corrected (without inducing hypercalcemia), and vitamin D intake and PTH levels must be controlled via calcimimetics.

Conflicts of interest

The author declares no conflict of interest.

Références

1 - Jean G, Charra B, Chazot C. Vitamin D deficiency and associated factors in hemodialysis patients. J Ren Nutr. 2008 Sep;18(5):395-9. doi: 10.1053/j.

2 - Holick MF. Vitamin D and the kidney. Kidney Int. 1987 Dec;32(6):912-29. doi: 10.1038/ki.1987.295.

3 - Ketteler M, Block GA, Evenepoel P, Fukagawa M, Herzog CA, McCann L, Moe SM, Shroff R, Tonelli MA, Toussaint ND, Vervloet MG, Leonard MB. Executive summary of the 2017 KDIGO Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD) Guideline Update: what's changed and why it matters. Kidney Int 2017, 1:26-36. doi: 10.1016/j.kint.2017.04.006.

4 - Soohoo M, Obi Y, Rivara MB, Adams SV, Lau WL, Rhee CM, Kovesdy CP, Kalantar-Zadeh K, Arah OA, Mehrotra R, Streja E. Comparative Effectiveness of Dialysis Modality on Laboratory Parameters of Mineral Metabolism. Am J Nephrol, 2022;53(2-3):157-168. doi: 10.1159/000521508.

5 - Noordzij M, Korevaar JC,Bos WJ, Boeschoten EW, Dekker FW, Bossuyt PM, Krediet RT. Mineral metabolism and cardiovascular morbidity and mortality risk: peritoneal dialysis patients compared with haemodialysis patients. NDT 2006, Sep;21(9):2513-20. doi: 10.1093/ndt/gfl257.

6 - Weissheimer R, Gardano S, Bucharles E, Madid Truyts CA, Jorgetti V, Figueiredo AE, Barrett P, Olandoski M, Pecoits-Filho R, de Moraes TP. High prevalence of biochemical disturbances of chronic kidney disease - mineral and bone disorders (CKD-MBD) in a nation-wide peritoneal dialysis cohort: are guideline goals too hard to achieve? J Bras Nefrol 2021, Apr-Jun;43(2):173-181. doi: 10.1590/2175-8239-JBN-2020-0147.

7 - Nitta K, Bieber B, Karaboyas A, Johnson DW, Kanjanabuch T, Kim YL, Lambie M, Hartman J, Shen JI, Naljayan M, Pecoits-Filho R, Robinson BM, Pisoni RL, Perl J, Kawanishi H. International variations in serum PTH and calcium levels and their mortality associations in peritoneal dialysis patients: Results from PDOPPS. Perit Dial Int. 2024 Mar 19:8968608241235516. doi: 10.1177/08968608241235516.

8 - Lopes MB, Karaboyas A, Zhao J, Johnson DW, Kanjanabuch T, Wilkie M, Nitta K, Kawanishi H, Perl J, Pisoni RL; PDOPPS Steering Committee. Association of single and serial measures of serum phosphorus with adverse outcomes in patients on peritoneal dialysis: results from the international PDOPPS. Nephrol Dial Transplant. 2023 Jan 23;38(1):193-202. doi: 10.1093/ndt/gfac249.

9 - Fusaro M, Barbuto S,Gallieni M, Cossettini A, Re Sartò GV, Cosmai L, Cianciolo G, La Manna G, Nickolas T, Ferrari S, Bover J, Haarhaus M, Marino C, Mereu MC, Ravera M, Plebani M, Zaninotto M, Cozzolino M, Bianchi S, Messa P, Gregorini M, Gasperoni L, Agosto C, Aghi A, Tripepi G. Real-world usage of Chronic Kidney Disease - Mineral Bone Disorder (CKD-MBD) biomarkers in nephrology practices. Clin Kidney J 2023, Nov 29;17(1):sfad290. doi: 10.1093/ckj/sfad290.

10 - Rhee CM, Molnar MZ, Lau WL, Ravel V, Kovesdy CP, Mehrotra R, Kalantar–Zadeh K. Comparative mortality – predictability using alkaline phosphatase and parathyroid hormone in patients on peritoneal dialysis and hemodialysis. Peritoneal Dialysis International 2013, Vol. 34, pp. 732–748 doi: 10.3747/pdi.2013.00110.

11 - Wu M, Wu H, Huang X, Ye H, Huang F, Yu X, Yang X. Associations between serum mineral metabolism parameters and mortality in patients on peritoneal dialysis. Nephrology (Carlton) 2019, Nov;24(11):1148-1156. doi: 10.1111/nep.13535.

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https://doi.org/10.25796/bdd.v7i3.83573
ISSN 2607-9917

12 - Murashima M, Fujii N, Goto S, Hasegawa T, Abe M, Hanafusa N, Fukagawa M, Hamano T. Associations of calcium, phosphate and intact parathyroid hormone levels with mortality, residual kidney function and technical failure among patients on peritoneal dialysis. Clin Kidney J 2023, Sep 7;16(11):1957-1964. doi: 10.1093/ckj/sfad223.

13 - Mäkelä S, Asola M, Hadimeri H, Heaf J, Heiro M, Kauppila L, Ljungman S, Ots-Rosenberg M, Povlsen JV, Rogland B, Roessel P, Uhlinova J, Vainiotalo M, Svensson MK, Huhtala H, Saha H. Abdominal Aortic Calcifications Predict Survival in Peritoneal Dialysis Patients. Perit Dial Int 2018, Sep-Oct;38(5):366-373. doi: 10.3747/pdi.2017.00043.

14 - Lu, X, Hu, M.C. Klotho/FGF23 Axis in Chronic Kidney Disease and Cardiovascular Disease. Kidney Dis. 2017, 3: 15–23.

15 - Bi S, Liang Y, Cheng L, Wang Y, Wang T, Han Q, Zhang A. Hemodialysis is associated with higher serum FGF23 level when compared with peritoneal dialysis. Int Urol Nephrol. 2017 Sep;49(9):1653-1659. doi: 10.1007/s11255-017-1605-z. Epub 2017 Apr 28. PMID: 28455658.

16 - Kim HJ, Park M, Park HC, Jeong JC, Kim DK, Joo KW, Hwang YH, Yang J, Ahn C, Oh KH. Baseline FGF23 is Associated with Cardiovascular Outcome in Incident PD Patients. Perit Dial Int. 2016 Jan-Feb;36(1):26-32. doi: 10.3747/pdi.2013.00343. Epub 2014 Sep 2. PMID: 25185018; PMCID: PMC4737562.

17 - Xu L, Hu X, Chen W. Fibroblast growth factor-23 correlates with advanced disease conditions and predicts high risk of major adverse cardiac and cerebral events in end-stage renal disease patients undergoing continuous ambulatory peritoneal dialysis. J Nephrol 2019, Apr;32(2):307-314. doi: 10.1007/s40620-018-0557-4.

18 - Mathew AT, Hazzan A, Jhaveri KD, Block GA, Chidella S, Rosen L, Wagner J, Fishbane S. Increasing hip fractures in patients receiving hemodialysis and peritoneal dialysis. Am J Nephrol. 2014;40(5):451-7. doi: 10.1159/000369039.

19 - Carmen Sánchez M, Auxiliadora Bajo M, Selgas R, Mate A, Millán I, Eugenia Martínez M, López-Barea F. Parathormone secretion in peritoneal dialysis patients with adynamic bone disease. Am J Kidney Dis. 2000 Nov;36(5):953-61. doi: 10.1053/ajkd.2000.19093. PMID: 11054351.

20 - Iseri K, Qureshi AR, Ripsweden J, Heimbürger O, Barany P, Bergström IB, Stenvinkel P, Brismar TB, Lindholm B. Sparing effect of peritoneal dialysis vs hemodialysis on BMD changes and its impact on mortality. J Bone Miner Metab 2021, Mar;39(2):260-269. doi: 10.1007/s00774-020-01144-8.

21 - Hong YA, Kim JH, Kim YK, Chang YK, Park CW, Kim SY, Kim YS, Kang SW, Kim NH, Kim YL, Yang CW. Low parathyroid hormone level predicts infection-related mortality in incident dialysis patients: a prospective cohort study. Korean J Intern Med 2020, Jan;35(1):160-170. doi: 10.3904/kjim.2018.264.

22 - Yang Y, Da J, Jiang Y, Yuan J, Zha Y. Low serum parathyroid hormone is a risk factor for peritonitis episodes in incident peritoneal dialysis patients: a retrospective study. BMC Nephrol 2021, Jan 29;22(1):44. doi: 10.1186/s12882-021-02241-0.

23 - Jean G, Mayor B, Hurot JM, Deleaval P, Lorriaux C, Zaoui E, Chazot C. Biological impact of targeted dialysate calcium changes in haemodialysis patients: the key role of parathyroid hormone. Nephrol Dial Transplant. 2013 Jan;28(1):176-82. doi: 10.1093/ndt/gfs119. Epub 2012 Jul 3.

24 - Wang AY, Brimble KS, Brunier G, Holt SG, Jha V, Johnson DW, Kang SW, Kooman JP, Lambie M, McIntyre C, Mehrotra R, Pecoits-Filho R. ISPD cardiovascular and metabolic guidelines in adult peritoneal Dialysis patients' part II - Management of Various Cardiovascular Complications. Perit Dial Int 2015, 35:388–96.

25 - Jin L, Zhou J, Shao F, Yang F. Long-term effects on PTH and mineral metabolism of 1.25 versus 1.75 mmol/L dialysate calcium in peritoneal dialysis patients: a meta-analysis. BMC Nephrology 2021, 22:44. doi.org/10.1186/s12882-021-02241-0

26 - Piraciaba MCT, Cordeiro L, Guimarães EA, Abensur H, Pereira BJ, Jorgetti V, Moysés RMA, Elias RM. A feasibility study of avoiding positive calcium balance and parathyroid hormone increase in patients

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ISSN 2607-9917

on peritoneal dialysis. Bone Rep 2022, Sep 29;17:101625. doi: 10.1016/j.bonr.2022.101625.
27 - An N, Zhou H, Li X, Yu X, Yang H, Zhai L, Huang Y, Yao C. Effect of low-calcium and standard-calcium dialysate on serum calcium, phosphorus and full-segment parathyroid hormone in patients on peritoneal dialysis: A retrospective observational study. Int J Artif Organs 2023, Oct-Nov;46(10-11):539-546. doi: 10.1177/03913988231206641. Epub 2023 Oct 25. PMID: 37877542.
28 - Courivaud C, Davenport A. Phosphate Removal by Peritoneal Dialysis: The Effect of Transporter Status and Peritoneal Dialysis Prescription. Perit Dial Int 2016, Jan-Feb;36(1):85-93. doi: 10.3747/pdi.2014.00173.

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