Editorial

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Hyperphosphatemia in Chronic Kidney Disease

Hiperfosfatemia en la enfermedad renal crónica: incrementar la excreción renal de fósforo tal vez sea la clave para su tratamiento

As García Ospina and col. explain in their excellent article entitled "Importance of hyperphosphatemia in chronic kidney disease, how to avoid it and treat it by nutritional measures", an adequate diet constitutes one of the fundamental therapeutic pillars against the serum excess of Phosphorus in patients with chronic renal failure. Also, it should be remembered that other complementary strategies for the treatment of hypophosphatemia are:

a) reduce intestinal absorption of phosphorus by the use of chelators (calcium carbonate, etc.); b) reduce the phosphorus passage from the bone to the plasma compartment (bisphosphonates, control of secondary hyperparathyroidism, etc.), c) Body excretion of phosphorus, either by urine or dialysis[1,2].

With respect to the strategy of increasing the urinary excretion of phosphorus, it is precisely the mechanism that generally preserves the chronic renal patient suffering from hyperphosphatemia to about the end of stage IIIb of their disease (glomerular filtration rate: 30 ml/Min/1.73 m²), where, due to the progressive increase in the phosphorus excretion, stimulated in part by the serum rise of the parathormone, the fractional phosphorus excretion can pass from 9 ± 05% in healthy people, to 25 ± 0.9% in patients with chronic renal insufficiency stage III, up to 40 ± 09% in chronic nephrotic stage V[3,4].

However, it usually occurs in advanced stages of chronic renal failure (stages IV and V) that, although the fractional excretion of phosphorus in urine continues to increase, its magnitude is not enough to prevent such levels of glomerular filtration loss (Glomerular filtration rate 30 ml/min/1.73 m²), no hyperphosphatemia occurs. As a result of the poor dialyzation of phosphorus, pharmacological increase in phosphorus tubular excretion in order to combat hyperphosphatemia would be extremely useful not only in advanced chronic renal disease (stages IV and V), but also in the patient in chronic dialysis with significant residual diuresis. In this sense, the concept of preserving the patient's residual diuresis on dialysis is important, not only by avoiding the use of nephrotoxic drugs, but also by continuing the use of angiotensin II receptor blocker (nephroprotection) inhibitors and Applying the concept of incremental dialysis (lower dose of dialysis) both at the onset of peritoneal dialysis and hemodialysis[5]. Among drugs that have been shown to increase urinary phosphorus excretion, blocking proximal tubular reabsorption, indapamide is a diuretic which, in animal studies, has been shown not only to reduce glomerular filtration but also to achieve a significant reduction in serum and histological levels of phosphorus, reaching a dose dependent increment in the fractional excretion of this divalent anion in the order of up to 75 ± 13%[6].

This is the reason why we proposed together with Dr. César Restrepo Valencia from RISRECP (Ibero-American Research Network on Renal Health and Prevalent Chronic Diseases) a protocol of clinical research in order to evaluate the effect of indapamide in the control of hyperphosphatemia in patients with chronic kidney disease, study to which all our colleagues are already invited.

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