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Original article

Correction of 25-OH-vitamin D deficiency improves control of secondary hyperparathyroidism and reduces the inflammation in stable haemodialysis patients☆

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ABSTRACT

Introduction: Patients on haemodialysis (HD) have a high prevalence of 25-OH-vitamin D (25-OH-D)deficiency. Secondary hyperparathyroidismis a common condition in these patients, which is very important to control. 25-OH-D is involved in regulating calcium homeostasis. As such, appropriate levels of this vitamin could help to control bone mineral metabolism.

Objective: To evaluate the effect 25-OH-D repletion in HD patients with 25-OH-D deficiency (<20 ng/ml) on the control of secondary hyperparathyroidism and microinflammation status.

Patients and methods: Prospective observational study in which stable patients on HD with 25-OH-D deficiency (<20 ng/ml) were treated with oral calcifediol 0.266 mcg/every 2 weeks for three months. Dialysis characteristics, biochemical parameters and drug doses administered were analysed before and after the correction of the deficiency.

Results: Forty-five stable HD patients with a mean age of 74.08 ± 12.49 years completed treatment. Twenty-seven patients (60%) achieved 25-OH-D levels above 20 ng/ml (23 with levels > 30 ng/ml and 4 between 20 and 30 ng/ml). Parathyroid hormone levels decreased in 32 of the 45 patients, 23 of which (51%) achieved a >30% decrease from baseline. In terms of concomitant treatment, we observed a significant reduction in the selective vitamin D receptor activator dose, but no changes in calcimimetic or phosphate binders administration. In terms of malnutrition–inflammation status, a decrease in C-reactive protein was noted, although other microinflammation parameters, such as activated monocytes (CD14+/CD16+ and CD 14++/CD16+) were unchanged. No changes were observed in the levels of FGF-23.

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Conclusions: Correcting 25-OH-D deficiency in HD patients is associated with better secondary hyperparathyroidism control with lower doses of vitamin D analogues, as well as an improvement in inflammatory status. Our results support the recommendation to determine 25-OH-D levels and correct its deficiency in these patients.

La corrección del déficit de 25-OH-vitamina D mejora el control del hiperparatiroidismo secundario y el estado inflamatorio de pacientes estables en hemodiálisis

Palabras clave:
Hipovitaminosis D
Hemodiálisis
Hiperparatiroidismo secundario
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RESUMEN

Introducción: El déficit de 25-OH-vitamina D (25-OH-D) es común en los pacientes en hemodiálisis (HD). Por otra parte, es bien conocida la elevada incidencia de hiperparatiroidismo secundario en este grupo de pacientes, y lo importante que es su adecuado control. La 25-OH-D está implicada en la regulación de la homeostasis del calcio, por lo que tener niveles adecuados puede contribuir en el control del metabolismo óseo-mineral.

Objetivos: Evaluar el efecto de la repleción de 25-OH-D en pacientes en HD con déficit vitamínico (niveles < 20 ng/ml), en el control del hiperparatiroidismo secundario y en el estado de microinflamación.

Pacientes y métodos: Estudio observacional, prospectivo en el que se trataron pacientes estables en HD con déficit de 25-OH-D (<20 ng/ml), con calcifediol 0,266 mcg/15 días vía oral durante 3 meses. Los datos de HD, parámetros bioquímicos y las dosis de fármacos administrados fueron analizados antes y después de la corrección del déficit.

Resultados: Un total de 45 pacientes estables en HD con edad media 74,08 ± 12,49 años completaron el tratamiento. Del total, 27 pacientes (60%) alcanzaron niveles de 25-OH-D > 20 ng/ml (en 23 fueron > 30 ng/ml, y 4 entre 20-30 ng/ml). Las cifras de hormona paratiroidea descendieron en 32 de los 45 pacientes, alcanzando en 23 (51% de tratados) un descenso > 30% respecto al valor basal. En cuanto al tratamiento concomitante, se objetivó un descenso significativo de la dosis de activador selectivo del receptor de vitamina D; sin evidenciarse cambios en la dosis de calcimimético ni de quelantes. Respecto al estado de malnutrición-inflamación, destaca un descenso de la proteína C reactiva, aunque no se modificaron otros parámetros de microinflamación como los monocitos activados (CD14+/CD16+ y CD 14++/CD16+). Tampoco se observaron cambios en los niveles de FGF-23.

Conclusiones: La corrección del déficit de 25-OH-D en pacientes en HD se asocia a un mejor control del hiperparatiroidismo secundario con menores dosis de análogos de vitamina D y a una mejoría en el estado inflamatorio de estos pacientes. Nuestros resultados apoyan la recomendación de determinar niveles de 25-OH-D y corregir el déficit en pacientes en HD.

Introduction

The hormonal system of native vitamin D (25-OH-D) is linked to the regulation of calcium homeostasis and bone metabolism. 25-OH-D deficiency is very common not only in specific groups of patients, but also in the general population.[1] 25-OH-D deficiency is highly prevalent in patients with chronic kidney disease in its various stages, and may be found in up to 90% of the population in CKD stage 5D patients (haemodialysis; HD).[2,3]

Deficiency of 25-OH-D is associated to a greater prevalence of diseases such as cancer[4] and cardiovascular disease.[5,6] This is possibly explained not only by its relation with bone and mineral metabolism, but also by its pleiotropism.[7]

Of the pleiotropic effects of 25-OH-D, its role in the immune system and its possible association to chronic inflammation are notable. HD patients present with chronic microinflammation,[8] which plays an important role in the elevated morbidity and mortality of these patients. The uraemia-related inflammation can be assessed by the measurement of traditional biochemical parameters (albumin, ferritin or C-reactive protein [CRP][9]), however these are not always changed, so it is necessary to use more sensitive methods. Recent studies have shown that the determination of activated monocytes (CD14+/CD16+ and CD 14++/CD16+) in the peripheral blood of patients with chronic kidney disease is more sensitive of inflammation than the conventional methods.[10] Determination of activated monocytes may be used to evaluate inflammation in response to treatments or