Vitamin deficiencies in chronic kidney disease

Vitorino Modesto Santos

Received: 9 August 2015  Accepted: 19 October 2015  Published: 7 November 2015


To the Editor

Amini et al. carried out an interesting systematic review about the effects of vitamin B12 supplementation in hyperhomocysteinemia in individuals with end stage renal diseases, and highlighted the lack of expressive number of studies with randomized controlled design (1). Vitamin B12 was administrated either by injection or orally, and some patients received folate as well. It is noteworthy to mention that isolated folate supplementation also reduce the level of hyperhomocysteinemia; in fact, both vitamins play an essential role during the remethylation of homocysteine to methionine (1). The authors mentioned the relationship between hyperhomocysteinemia and atherosclerosis, which have been associated with risk of death due to vascular disorders in this group of patients (1,2).

Costa et al. reviewed numerous epidemiological studies conducted about vitamin D and found a more frequent negative correlation between concentrations of 25(OH) D and parathyroid hormone (3). Low levels of vitamin D [25(OH)D] have been described in association with chronic renal diseases; also, this secosteroid hormone has activity against diabetes mellitus and arterial hypertension, which are major traditional cardiovascular risk factors in the general population (2,3). Another Brazilian study involving retrospective analysis of traditional and non-traditional risk factors in dialitic individuals with cardiovascular disease detected a significant relationship between the carotid intima-media thickness and serum determinations of parathyroid hormone (2). Moreover, secondary hyperparathyroidism is common in patients with chronic kidney disease, and vitamin D supplements may have a positive effect on the mortality rate of this group (2). Involved mechanisms include hypovitaminosis D, hypocalcemia, and hyperphosphatemia, metabolic arterial calcifications, left ventricular hypertrophy, myocardial infarction and cardiac arrest (2).

The mentioned articles can contribute to better understanding the challenging phenomena of the conundrum represented by multivariate aspects of end stage renal diseases. However, I would like to pose additional findings that have been described in hypovitaminosis B12 and D. Schiller et al. prospectively studied 600 dialytic patients and also examined diabetes mellitus, vitamin D deficiency, coronary artery disease, peripheral vascular disease, and stroke in these patients (4). Patients with diabetes had lower blood levels of vitamin D and higher mortality rate than non-diabetic controls, and hypovitaminosis D was associated with increased all-cause of mortality (4). Mishra et al. reported fever, hemolytic anemia, thrombocytopenia, hyperhomocysteinemia, and hyperphosphatemia caused by vitamin B12 and vitamin D deficiencies in a young girl (5). Her renal function was
normal, and her condition improved after receiving vitamin B12 injection and oral vitamin D, which were later changed to oral vitamin B12 and pyridoxine to control the hyperhomocysteinemia (5).

References