

## Hypercalcaemia and Hypervitaminosis A in Chronic Renal Failure

The Editor,

Sir,

Chronic toxicity by vitamin A may occur with long-term ingestion at lower doses in patients with chronic renal failure, but hypercalcaemia is rarely observed in this setting and is probably due to effects of the vitamin A on bone osteoclastic or osteoblastic phenomena (1). Although the first case report was in 1953, this association has been very rarely described (1).

I read the interesting case study by Hammoud *et al* about a 67-year-old woman with arterial hypertension, chronic kidney disease stage IV and hypercalcaemia around 14 mg/dL. Her blood levels of parathyroid hormone (PTH) and of vitamin D were within the normal ranges, and hypotheses for hypercalcaemia including secondary hyperparathyroidism, hypercalcaemic hypocalciuric syndrome, and occult malignancy were ruled out (1). After establishing the diagnosis of hypervitaminosis A, the vitamin supplement was stopped, and vitamin A and serum calcium levels returned to normal few months later (1). The authors pedagogically described the sources and metabolic steps of vitamin A. Risks of toxicity by excessive ingestion were emphasized in people with renal failure because kidneys are the major excretory route of retinol and retinol-binding protein. Caution with vitamin A is highlighted in this scenario (1).

The report by Hammoud *et al* is useful to clear mechanisms of vitamin disorders related or not to chronic renal disease; but I would like to add some Brazilian data (2–4). Santos *et al* reported a 69-year-old woman with neurological disturbances and xanthoderma associated with long-standing excessive ingestion of green vegetables, papaw and tomato (2). Such dietary behaviour yields higher serum levels of vitamin A, carotenoids and metabolites, which produce a cutaneous yellow to orange discolouration, with the mucosa of normal colour. The patient had neither renal failure nor hypercalcaemia, as blood determinations showed (2). Cutaneous discolouration improved in 2 months by reduction of the excessive ingestion (2). Worthy of note, the variable intensity of xanthoderma observed in individuals with

chronic kidney disease may be due to the slow conversion of carotenoids to vitamin A in the skin (2).

Costa *et al* (2013) retrospectively studied the relationship between PTH levels and carotid thickness in people with chronic renal failure and secondary hyperparathyroidism (3). The purpose was to evaluate the eventual role of PTH serum levels; hypocalcaemia and hyperphosphatemia; vitamin D and calcitriol deficiency; and traditional cardiovascular risk factors in the carotid changes observed in patients with dialytic chronic kidney failure (3). The authors found a significant correlation between the PTH levels and carotid thickness (3).

Costa *et al* (2014) reviewed epidemiological features of vitamin D including calcium and phosphorus homeostasis, arterial hypertension, PTH levels, and chronic renal failure (4). The 14 evaluated patients were distributed in two groups in accordance with their PTH serum levels: 200 pg/mL, or lower, and above 500 pg/mL. The authors emphasized the role of dialysis, and the normal levels of PTH, calcium and phosphate in the prevention of atherosclerosis in patients undergoing haemodialysis. Furthermore, they concluded that the serum levels of PTH were not predictive to infer the respective serum levels of vitamin D (4).

The commented articles may contribute to enhance the understanding of mechanisms of vitamins, minerals and PTH disturbances associated or not with chronic kidney disease.

**Keywords:** Calcium, chronic kidney disease, vitamin A, vitamin D.

*V Modesto dos Santos*

*From: Internal Medicine Department of Armed Forces Hospital and Catholic University of Brasília, Brasília, Brazil.*

*Correspondence: Professor V Modesto dos Santos, Internal Medicine Department of Armed Forces Hospital and Catholic University of Brasília, Estrada do Contorno do Bosque s/n, Cruzeiro Novo. CEP 70630-900 Brasília, Brazil.*

*Email: vitorinomodesto@gmail.com*

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