Vitamin D and Chronic Kidney Disease

Vitamin D is a hormone involved in bone mineral metabolism.[1] The hormone has many extraskeletal effects as well.[2,3] It has multiple immunomodulatory properties,[2] it is involved in glucose metabolism, as it has been proved that it improves glucose metabolism in diabetes mellitus[3] and has a protective role against cardiovascular disease.[4] In this issue of the Journal, in an article the relationship of vitamin D with kidney disease is meticulously reviewed.

Vitamin D is a hormone which belongs to the secosteroid family. Vitamin D is partially ingested with the diet and mainly produced in the skin by the action of ultraviolet irradiation when the skin is exposed to the sun. Therefore, in the current world less exposure to sunlight due to the modern way of life as well as to habits related to covering the body with clothes to avoid sun exposure may aggravate the problem of vitamin D deficiency. In addition, the amount of melanin present within the skin may be related to the action of the sunlight to produce vitamin D in the skin. All these factors have contributed to the development of vitamin D deficiency in the worldwide population.

Vitamin D deficiency is also observed in patients with chronic kidney disease (CKD).[5] Vitamin D deficiency has been observed in more than 80% of renal transplant recipients and non-renal transplant CKD patients. Multiple factors are involved in the pathogenesis of impaired calcium metabolism and vitamin D deficiency in CKD patients.[6] In CKD, 1a-hydroxylase in the kidney is reduced and is further inhibited by the action of hyperphosphaturic osteocyte-derived hormone FGF-23, which increases to compensate for phosphate retention. In addition, FGF-23 induces the expression of 24-hydroxylase responsible for the degradation of $1,25(OH)_2D_3$. These mechanisms lead to secondary hyperparathyroidism, hypocalcemia, and osteoporosis leading to fractures. Skin synthesis of vitamin D is reduced in CKD. Secondary hyperparathyroidism has been hypothesized to decrease CYP450 isoforms within the liver, thus reducing the synthesis of $25(OH)D_3$. The risk for falls and fractures is four times greater in female dialysis patients than in the general population.[7] Furthermore, secondary hyperparathyroidism may progress to tertiary hyperparathyroidism necessitating the removal of the hyperfunctioning parathyroid glands.

Vitamin D deficiency or insufficiency are related to multiple outcomes for CKD and dialysis populations, such as secondary hyperparathyroidism and high bone turnover markers, low bone mineral density, muscle weakness, risk of falls, metabolic syndrome and obesity, insulin resistance, left ventricular hypertrophy and atherosclerosis, vascular calcification and arterial stiffness, cognitive impairment, progression of kidney disease and mortality.

CKD is related to obesity and the metabolic syndrome.[8] Excess accumulation of adipose tissue leads to obesity, which is a main cause of metabolic disorders and kidney disease. In obesity vitamin D metabolism is impaired, vitamin D deficiency being observed.[9] However, the exact relationship of obesity with vitamin D metabolism is currently investigated and not yet fully clarified.

In CKD supplementation with vitamin D should be performed, so that vitamin D levels should be maintained at a level 30 ng/ml or higher.[10] Supplementation with vitamin D may reduce mortality and progression to dialysis in CKD patients.

In conclusion, vitamin D deficiency is observed in CKD and it may be related to morbidity, mortality, and progression to dialysis. Treatment with vitamin D may reduce mortality and morbidity in CKD.

Ifigenia Kostoglou-Athanassiou

Department of Endocrinology, Asclepeion Hospital, Voula, Athens, Greece

Address for correspondence:
Dr. Ifigenia Kostoglou-Athanassiou,
Korinthias 7, 115 26 Athens, Greece.
E-mail: ikostoglouathanassiou@yahoo.gr

Received: 11 Mar 19 **Accepted:** 23 Jan 20
Published: 24 Feb 21

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Quick Response Code:

Website: www.ijpvmjournal.net/www.ijpm.ir

DOI: 10.4103/ijpvm.IJPVM_97_19

How to cite this article: Kostoglou-Athanassiou I. Vitamin D and chronic kidney disease. Int J Prev Med 2021;12:15.