

## RENOPROTECTIVE EFFECTS OF VITAMIN D AND RENIN-ANGIOTENSIN SYSTEM

ALKETA KOROSHI, ALMA IDRIZI

### Abstract

Vitamin D has many physiological functions. First it is a primary regulator of calcium homeostasis. Beyond that, vitamin D and its receptors (VDR) play important role in the immune system, cardiovascular system, reproductive system and insulin resistance. An important aspect of vitamin D pleiotropic effects is the interaction with components of renin-angiotensin system (RAS). It was demonstrated that vitamin D-null mice have a sustained elevation of renin expression. The combination of both the AT<sub>1</sub> blockers and the vitamin D analogues, leads to a marked amelioration of the molecular and clinical markers of diabetic nephropathy.

This combination may protect the kidney through the effects on both the glomerular and the tubulointerstitial compartments. There are different studies that corroborate the renoprotective action of vitamin D in CKD. In fact the renoprotective mechanisms in humans remain to be discovered, but these are realized through reduction of proteinuria, high blood pressure, inflammation as well as hemodynamic effects.

An important mechanism is the role of vitamin D as a potent negative endocrine regulator of renin expression. Actually we can accept that low vitamin D levels are a candidate novel risk factor for the progression of renal disease but it is not demonstrated yet that vitamin D can prolong the time to end-stage renal disease.

This question remains to be answered in other future controlled clinical trials.

### Introduction

Department of Nephrology and Dialysis, University Hospital Center, Tirana, Albania Vitamin D is a primary regulator of calcium homeostasis. Genetic inactivation of either the vitamin D receptor (VDR), a member of the nuclear receptor superfamily that mediates the action of 1,25-dihydroxyvitamin D<sub>3</sub> [1,25(OH)<sub>2</sub>D<sub>3</sub>], or 25-hydroxyvitamin D<sub>3</sub> 1 $\alpha$ -hydroxylase, the rate-limiting enzyme for the biosynthesis of 1,25(OH)<sub>2</sub>D<sub>3</sub>, results in impaired calcium homeostasis, leading to hypocalcemia, secondary hyperparathyroidism, and rickets (1,2,3,4,5). However, the wide tissue distribution of VDR suggests that the vitamin D endocrine system has additional physiological functions beyond calcium homeostasis. There are many experimental and clinical data that corroborate the pleiotropic actions of vitamin D. Indeed, vitamin D and VDR have been shown to play important roles in the immune system, cardiovascular system, reproductive system, and hair growth (1). Many of these actions of vitamin D are realized independently from the effects on PTH and the level of calcium and phosphorus. Although VDR-deficient mice do not have a