Vitamin D & Immunity in CKD

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Epidemiological experiments have shown that vitamin D deficiency is closely related to autoimmune and infectious diseases. Immune cells carry VDR and 1 □ -hydroxylase, which produces the active metabolite 1.25(OH)2D through local synthesis and heightens immunomodulatory properties. Increasing evidence indicates that vitamin D deficiency may cause dysregulation of the innate and adaptive immune systems and promote microinflammation.

Vitamin D affects both the innate and adaptive immune systems. In innate immunity, vitamin D promotes production of cathelicidin and β-defensin 2 and enhances the capacity for autophagy via toll-like receptor activation as well as affects complement concentrations. In adaptive immunity, vitamin D suppresses the maturation of dendritic cells and weakens antigen presentation. Vitamin D also increases T helper (Th) 2 cytokine production and the efficiency of Treg lymphocytes but suppresses the secretion of Th1 and Th17 cytokines. In addition, vitamin D can decrease autoimmune disease activity. Vitamin D has been shown to play an important role in maintaining normal immune function and crosstalk between the innate and adaptive immune systems.

Low 1.25(OH)2D levels have been related to elevated mortality rates in CKD patients. Strong associations have been shown between the prevalence of vitamin D deficiency and susceptibility to infection. Vitamin D might influence immune responsiveness and its potential modulating role in vaccine immunogenicity. Vitamin D deficiency may also contribute to deterioration of immune function and infectious disorders in CKD patients. However, it is still premature to recommend vitamin D for practical therapeutic or preventive use to enhance vaccine response. More research and large trials are needed for further confirmation.