Editorial

Vitamin D and Cardiovascular disease

Cardiovascular Disease (CVD) is one of the major causes of morbidity and mortality throughout the world. A number of risk factors have been conventionally associated with the occurrence of CVDs with deficiency of vitamin D in recent times being emphasized to be one of the emerging key ingredients of increased cardiovascular risk. Even though vitamin D is classically known for its crucial role in calcium and bone metabolism, evolving data indicate that vitamin D deficiency plays an important role in the genesis of CVDs like hypertension, diabetes, left ventricular hypertrophy and congestive heart failure and major cardiovascular events like myocardial infarction. Nearly 95% of the body's requirements for vitamin D are derived from cutaneous synthesis from sun exposure with the remainder ingested from dietary source. The serum 25 (OH)D level is considered as the standard clinical measure of vitamin D levels in body and for purposes of monitoring vitamin D therapy[1,2]

Studies in normotensive and hypertensive subjects reveal an inverse relationship vitamin D metabolites and plasma renin activity regardless of baseline renin levels or salt intake. Increased plasma renin in turn upregulates renin-angiotensin-aldosterone (RAAS). Vitamin D influences glycemic control through modulation of pancreatic RAS activity and regulation of calcium ion traffic across beta cells that directly affect insulin synthesis and secretion. Experimental studies have shown that calcitriol dose dependently suppresses the release of the proinflammatory cytokines-tumor necrosis factor –œ (TNF- œ) and interleukin-6 (IL-6).[3] In addition, the regulatory role of vitamin D on monocyte or macrophage differentiation may determine monocyte infiltration and cholesterol capture in the vascular wall and this may be the underlying reason behind increased plaque instability and incident myocardial infarctions in vitamin D deficient patients. Vitamin D deficiency causes stimulation of parathormone release(PTH). High PTH is associated with cellular apoptosis, fibrosis, vascular and myocardial smooth muscle cell hypertrophy ultimately predisposing to increase in arterial pressure and myocardial dysfunction.[4,5]

Vitamin D deficiency can be prevented and corrected easily by sunlight and / or vitamin D supplementation, though the benefit accrued after replenishment requires further studies, and definitive randomized controlled trials are needed to determine whether vitamin D therapy will live up to its hype.[5]

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