



Vitamin D deficiency & Coronary Artery Disease

Coronary artery disease (CAD) is one of the most public health problems throughout the world. Now it is considered as the leading cause of mortality and morbidity worldwide.¹ Like other South Asians, Bangladeshi population are unduly prone to develop CAD, which is often premature in onset, rapidly progressive and angiographically more severe.² The underlying pathophysiology of CAD is poorly understood. The 'classic' risk factors like diabetes mellitus, hypertension, dyslipidemia, obesity, smoking and excessive alcohol consumption undoubtedly play important role. Poor dietary habits, excess intake of saturated and trans fat, high salt intake, and lack of physical activity also play important role. Beside conventional risk factors and genetic predisposition, some emerging new risk factors like hypovitaminosis D, arsenic contamination, particulate matter air pollution may play vital role.³

There is a unique role of vitamin D on calcium homeostasis and it has beneficial anti-inflammatory and antiatherosclerotic effects. Vitamin D is also involved in glycemic control, lipid metabolism, regulate insulin secretion and sensitivity. The antihypertensive properties of vitamin D include suppression of the renin-angiotensin-aldosterone system. It has renoprotective effects, having direct effects on endothelial cells, inhibit the growth of vascular smooth muscle cells, prevent secondary hyperparathyroidism and it has also beneficial effects on cardiovascular risk factors.⁴

The exact relationship between hypovitaminosis D and increased cardiovascular risk has not yet been established, multiple hypotheses have been postulated. Vitamin D receptors (VDRs) have been distributed widely throughout the cardiovascular system.⁵ Vitamin D acts via these receptors and reduces cardiac ischemia-reperfusion injury and reactive oxygen species. It also shows favorable effects on inflammation and thrombosis. Atherosclerosis, chronic inflammation, endothelial dysfunction and arterial calcification occur due to hypovitaminosis D. Moreover, vitamin D insufficiency may activate the renin-angiotensin system and increase insulin resistance, endothelial dysfunction, inflammation, platelet function, and blood pressure (BP) regulation.⁶ An experimental trial revealed that vitamin D supplementation suppressed vascular inflammation by inhibiting the Nuclear Factor- κ B (NF- κ B) pathways and decreasing the process of atherosclerosis and hence subsequent Coronary artery disease (CAD).⁷

The association of vitamin D deficiency with coronary artery diseases (CADs) have been investigated in many studies. In a multicenter US cohort study evaluating patients admitted with acute coronary syndrome (ACS), about 95% of patients were found to have low vitamin D levels.⁸ In a case-control study (n = 240), Roy et al. reported that severe vitamin D deficiency was associated with increased risk of acute myocardial infarction after adjusting for risk factors.⁹

A prospective nested case-control study was conducted between 1993 and 1999 of 18,225 US men (Health Professionals Follow-Up Study) and this study revealed that vitamin D deficiency was associated with a higher risk of myocardial infarction in comparison with sufficient 25(OH)D after multivariate adjustment.¹⁰

Vitamin D deficiency is an emerging risk factor for coronary artery disease, in addition to conventional and genetic risk factors. Estimation of serum vitamin D level, genotyping for vitamin D receptor variants, estimation of serum calcium and phosphates level and bone mineral density are mandatory to evaluate the patients with cardiovascular disease. Regular screening, monitoring and treating of vitamin D should be taken to reduce cardiovascular morbidity and mortality.

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