Association between Hypovitaminosis D and Acute Myocardial Infarction

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Abstract: Hypovitaminosis D is defined as a low level of vitamin D ranging from less than 20 ng/ml and insufficient up to 21-29 ng/mL. It is associated with several abnormalities in the body such as osteoporosis, rickets in children and many more. Worldwide, vitamin D deficiency is becoming increasingly prevalent. Among healthy middle-aged to elderly persons, low levels of 25-hydroxy-vitamin D (25-OH D), the main circulating storage form of vitamin D, are found in up to one third to one half of them. Inadequate levels of 25-OH D are primarily triggered by inadequate cutaneous synthesis brought on by insufficient sun exposure or pigmented skin as well as insufficient food intake. Whilst vitamin D deficiency affects the musculoskeletal system, an increasing amount of data suggests that low levels of vitamin D may have an adverse impact on the cardiovascular system. We discussed the function of vitamin D and its cardiovascular protective effects in this article. The incidences of acute myocardial infarction brought on by hypovitaminosis D that increases hospital morbidity.

Keywords: Hypovitaminosis D, Acute Myocardial Infarction, Cardiovascular, Sun exposure

I. Introduction

A prevalent worldwide health issue, vitamin D deficiency known as hypovitaminosis D is often overlooked and untreated, associated with decay of the teeth, rickets, growth retardation, osteomalacia, osteopenia, osteoporosis, lower muscle mass, falls, a greater likelihood of fracture and role in cardiovascular diseases in adults. Insufficient HDL cholesterol, older age, living farther from the equator, having a darker complexion, wintertime season, air pollution, smoking, malabsorption, renal and liver disease, and medication (anticonvulsants, glucocorticoids, antirejection, and HIV therapy) are all linked to vitamin D deficiency. The biologically active form of vitamin D is 1,25 dihydroxyvitamin D. Hypovitaminosis D is defined as the level of calcidiol ranging from less than 20 ng/ml and insufficient up to 21–29 ng/mL. Vitamin D is considered to be potentially dangerous if its concentration is greater than 150 ng/mL and sufficient if it is greater than 30 ng/mL.

[2] Inflammatory and fibrotic pathways, vascular cell development, and renin-angiotensin-aldosterone system (and subsequently blood pressure) are all regulated by vitamin D. Hypovitaminosis D is linked to hyperlipidemia, left ventricular hypertrophy, arterial stiffness, and vascular dysfunction. Due to these factors, vitamin D has been associated with cardiovascular health and CVD risk.

[3] Cardiovascular disease is the leading cause of death worldwide, and coronary artery stenosis decreases myocardial blood flow, which may ultimately result in infarction. The most contentious theory regarding coronary stenosis is atherosclerosis. Inflammation of the body is one of the primary causes, according to researchers. Vitamin D is one of many variables that affect this inflammatory process.

[4] The potential independent link between hypovitaminosis D and in-hospital mortality in patients with coronary artery disease (CAD) was first established by Correia et al. The in-hospital cardiovascular death rate was 24% for patients with vitamin D levels under 10 ng/mL, much higher than the rate seen in the other patients (4.9%, with a relative risk of 4.3).

[5] A growing set of evidence indicates vitamin D deficiency impacts up to 50% of young people and apparently healthy children, which makes it much more common than originally thought. According to the Third National Health and Nutrition Examination Survey (NHANES III), 25% to 57% of individuals in the United States are believed to be vitamin D deficient. Given the increasing atmospheric UVB radiation filtering carried on by higher latitudes' slanted sun rays, the prevalence of vitamin D deficiency rises proportionally to distance from the equator. [6]

II. Incidence Of AMI and hypovitaminosis D

The incidence of coronary artery disease, diabetes, hypertension, and hypovitaminosis D increases linearly with distance from the equator. Furthermore, during seasons with less sunlight exposure, such as the winter months, the risk of cardiac mortality and the incidence of vitamin D deficiency are higher. [7] Current research suggests that a lack of vitamin D may lead to the development of various cardiovascular risk factors, including hypertension, the metabolic syndrome, and diabetes mellitus (DM). Subsequently those with chronic kidney disease and primary hyperparathyroidism, both of which have been associated with vitamin D deficiency, are more likely to die from cardiovascular causes than from disorders related to their underlying disease. [8] Notably, the relative risk of AMI decreased as vitamin D quartiles increased, indicating an inverse link between vitamin D status and AMI risk. Furthermore, more following cohorts have supported similar figures. Among 1739 healthy Framingham Offspring Study participants, the incidence of major cardiovascular events was 50% and 80% higher in those with vitamin D deficiency and insufficiency, respectively. [9] Melamed et al
analysed all cause mortality by quartile of 25(OH)D and concluded that people who were in the lowest quartile of 25(OH)D had significantly higher adjusted mortality rate ratios (MRR (95% CI) 1.28 (1.11-1.48) compared to individuals in the highest quartile of 25(OH)D20). There was an association towards increased mortality rate attributable to cardiovascular mortality in the lowest quartile of 25(OH)D; however, this did not achieve statistical significance (MRR (95% CI) 1.22 (0.90 - 1.65)). As a result, multiple epidemiologic findings appear to imply that low vitamin D status is linked to poor cardiovascular outcomes.[10]

III. Role of Vitamin D in AMI

Many hypotheses have been put out, but the exact mechanism underlying the elevated cardiovascular risk in patients with hypovitaminosis D is still unknown. The cardiovascular system is filled with many vitamin D receptors (VDRs). Vitamin D has beneficial effects on thrombosis and inflammation while lowering cardiac ischemia-reperfusion injury and reactive oxygen species. Low 25(OH)D levels additionally contribute to arterial calcification, endothelial dysfunction, chronic inflammation, and atherosclerosis. In addition, vitamin D deficiency can increase insulin resistance, endothelial dysfunction, inflammation, platelet function, and blood pressure (BP) via triggering the renin-angiotensin system. An experimental study found that vitamin D administration reduced atherosclerosis and, consequently, CAD by inhibiting the Nuclear Factor-B (NF-B) pathways and reducing vascular inflammation.[11] The relationship between vitamin D and the prognosis for HF is the subject of several theories. Due to a Ca2+ ion excess in myocardial cells, HF affects cardiac contraction and relaxation. The absence of vitamin D may affect how Ca2+ functions in cardiac cells, resulting in fibrosis, intra-organizational inflammation, and cardiomyocyte hypertrophy. Low vitamin D levels may lead to endothelial dysfunction, inflammation, and renin-angiotensin system activation. The review revealed that patients without ACEi/ARB had more favourable vitamin D effects on in-hospital mortality. The effect of vitamin D was greater in patients who did not use ACEi/ARBs, suggesting that these patients had an active renin-angiotensin system.[12] Indications of the adverse cardiovascular effects of vitamin D deficiency in animal models and epidemiological research reveals an increase in cardiovascular events throughout the winter and at increasing latitudes from the equator led to the discovery of vitamin D's role in MI. In patients who are recovering from a myocardial infarction, it's critical to monitor their vitamin D levels and supplement any deficiencies. After myocardial infarction, vitamin D signalling has significant cardioprotective effects via anti-inflammatory, antifibrotic, and anti-apoptotic pathways.[13] An association between low vitamin D levels and a family history of cardiovascular disease is considered a risk factor. Since genes can affect a person's vitamin D status, this can mainly be explained. Three loci which are involved in cholesterol production, hydroxylation, and vitamin D transport have genetic variants that affect vitamin D levels and can be utilised for determining individuals who have a significantly increased risk of vitamin D deficiency. [14] Low vitamin D levels were associated with a higher risk of AMI in 18225 men in the Health Professionals Follow-up Study, even after controlling for other cardiovascular risk factors, and at a 10-year follow-up, subjects with normal vitamin D levels (> 30 ng/mL) had approximately half the risk of AMI. These findings were recently repeated in a comprehensive meta-analysis, which found an adjusted pooled relative risk of 1.52 for total cardiovascular events when comparing the lowest to highest baseline circulating vitamin D concentration categories.[15]

IV. Conclusions

In conclusion, the growing body of evidence presented in this review highlights the significant association between hypovitaminosis D and acute myocardial infarction (AMI). Vitamin D deficiency has been linked to various cardiovascular risk factors, including hypertension, metabolic syndrome, diabetes mellitus, and atherosclerosis. It is evident that low levels of vitamin D impact the cardiovascular system through multiple pathways, such as inflammation, endothelial dysfunction, arterial calcification, and activation of the renin-angiotensin system. Furthermore, studies have shown that patients with normal vitamin D levels have a reduced risk of AMI compared to those with vitamin D deficiency. While observational cohort studies have driven much of the current evidence, the exact mechanisms underlying the increased cardiovascular risk in individuals with hypovitaminosis D remain unclear. Nonetheless, experimental studies have indicated that vitamin D supplementation can have beneficial effects on thrombosis, inflammation, and cardiac ischemia-reperfusion injury, potentially reducing the risk of cardiovascular events. Given the global prevalence of vitamin D deficiency and its impact on cardiovascular health, it is crucial for healthcare professionals to monitor vitamin D levels in patients recovering from myocardial infarction and consider supplementation in cases of deficiency. Additionally, genetic factors may contribute to an individual's vitamin D status, suggesting the need for personalised approaches in identifying those at higher risk for vitamin D deficiency and related cardiovascular complications. Overall, this review underscores the importance of further research to better understand the mechanisms linking hypovitaminosis D to AMI and emphasises the potential benefits of addressing vitamin D deficiency as part of a comprehensive approach to cardiovascular health.

Conflicts of Interest
AUTHORS HAVE NO CONFLICT OF INTEREST.

References


