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Relationship between Vitamin D and Myocardial Infarction – Clinical Significance and Therapeutic Implications

Mehak Sharma[1], Amit Kumar Sharma[2]*

[1]*Teerthanker Mahaveer University (TMU), Uttar Pradesh (UP), India, Department- MBBS (Bachelor of Medicine and Bachelor of Surgery)*

[2]*Park university, Parkville, MO USA 64152, Department-Master of Healthcare Administration – Management Information Systems*

Address for correspondence –

Dr. Amit Sharma,

Park university, Parkville,MO USA 64152,Department-Master of Healthcare Administration – Management Information Systems

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ABSTRACT

A major risk factor for the development of coronary artery disease (CAD), vitamin D insufficiency is a very common disorder worldwide. Its relevance in the development of myocardial infarction (MI) is still unclear, despite its well-established role in the control of phospho-calcic metabolism. Since the patient has an acute myocardial infarction (AMI), vitamin D insufficiency is a treatable illness that requires prompt assessment and therapy. The several ways that vitamin D may prevent MI are covered in this article, along with its cardioprotective benefits and involvement in the formation of atherosclerosis. The vitamin D receptor (VDR) has been shown to be present on the surface of cardiomyocytes and vascular smooth muscle. Additionally, vitamin D deficiency is essential for activating the renin-angiotensin-aldosterone system (RAAS) and upregulating inflammatory cytokines. This review evaluates several observational studies and randomized controlled trials to investigate the effects of vitamin D insufficiency on MI. Based on observational research, the results indicate a favorable relationship between the incidence of MI and low blood vitamin D levels. Furthermore, research indicates that hypovitaminosis D puts AMI patients at risk for cardiac remodeling and post-infarction consequences. On the other hand, interventional trials show that the effect of vitamin D administration on reducing the risk of MI is questionable. Future studies are thus necessary to highlight the potential of vitamin D supplementation in reducing the incidence of MI, as well as to look into the best dosage and assess long-term effects on cardiovascular morbidity and mortality.

Keywords: cardiovascular diseases, hypovitaminosis d, myocardial infarction, non-st segment elevation myocardial infarction (nstemi)

I. INTRODUCTION

One of the most hotly contested risk factors for myocardial infarction (MI) is vitamin D, also referred to as the sunshine vitamin. Its participation in the development of cardiovascular diseases (CVDs) is still unclear, despite its well-established role in the control of phospho-calcic metabolism. The active form, calcitriol (1,25 dihydroxycholecalciferol), is produced by a sequence of hydroxylation processes of 7-dehydrocholesterol, which is created using UV light from cholesterol found in the human epidermis. More than 200 genes' transcription is altered when calcitriol interacts to the vitamin D receptor (VDR), which is found inside the cell nucleus [1-4].

Lipophilic compounds the size of cholesterol preferentially activate the nuclear receptor VDR [5]. Because vitamin D is lipophilic, it doesn't require additional signal transduction steps to regulate genes. The extensive presence of VDR makes the majority of human body tissues and various cell types responsive to calcitriol [6]. Enterocytes, osteoblasts, parathyroid glands, and distal renal tubule cells all express the VDR, which controls phospho-calcic metabolism. According to recent research, endothelial cells, lymphocytes, macrophages, smooth vascular muscle cells, beta-pancreatic cells, and cardiomyocytes are also home to VDRs, which are responsible for the cardiovascular effects of vitamin D3 [7-9].

Despite the use of successful preventative and therapeutic measures, CVDs, including MI, are significant contributors to the worldwide burden of death and morbidity. Over 30 million people had ischemic heart disease in 2021, and over nine million of them passed away. 9.5% of those over 60 have MI [10,11]. In addition to the well-known risk factors linked to the development of acute myocardial infarction (AMI), the medical faculty's attention has been piqued by the recent discovery of possibly treatable risk factors [12,13].

According to the Third National Health and Nutrition Examination Survey (NHANES III), vitamin D insufficiency is common throughout the United States, and since it is readily treated, it is critical to comprehend the relationship between vitamin D deficiency and MI [14,15]. Higher latitudes are associated with a discernible rise in the risk of cardiovascular death in most of the populations examined. This tendency usually becomes more pronounced during the colder winter months, but it diminishes at higher elevations. This trend has been linked in the past to the negative effects of hypovitaminosis D, which is more common in lower altitudes, colder winters, and higher latitudes [16–18].

Normal vitamin D blood levels are defined by the United States Endocrine Society as being more than or equal to 30 ng/mL. values between 21 to 29 ng/mL suggest vitamin D insufficiency, whilst values less than or equal to 20 ng/mL indicate deficiency. Notably, vitamin D insufficiency is a common dietary deficit that affects patients of all ages worldwide, including adults and children [19–21]. Approximately one billion people worldwide suffer from hypovitaminosis D, and one-third of these individuals lack identifiable risk factors [22, 23].

In order to assist researchers and clinicians in clarifying diagnostic and treatment recommendations, we present a thorough summary of the literature that is currently available, outlining the relationship between vitamin D deficiency and MI, suggested mechanisms of action, and therapeutic implications of vitamin D supplementation in lowering the risk of CVD.

Clinical Significance

A number of components are involved in the suggested process that connects vitamin D to cardiovascular health. Cell development, DNA repair, cell differentiation, apoptosis, and oxidative stress are all significantly impacted by vitamin D [24, 25]. Numerous tissues, including vascular smooth muscle and cardiomyocytes, contain the VDR. Vitamin D activates VDR, which influences differentiation, proliferation, and gene expression. Since vitamin D deficiency has been related to increased intima thickness and coronary artery calcification, it is hypothesized that vitamin D levels affect plaque development and stability [26–28]. Additionally, vitamin D prevents thrombosis, the formation of foam cells from macrophages, the uptake of cholesterol by macrophages, the transport of high-density lipoprotein (HDL), the production of nitric oxide, and vascular repair—all of which have been demonstrated to be protective against cardiovascular events like MI [29,30]. Numerous CVDs, including as hypertension, coronary artery disease (CAD), ischemic heart disease, stroke, and heart failure, have been associated with low levels of circulating vitamin D [31, 32].

Because vitamin D regulates inflammation, atherosclerosis, and endothelial dysfunction, it is a key factor in the development of cardiovascular diseases like MI. Increased inflammation and endothelial dysfunction can result from low vitamin D levels. Atherosclerosis, one of the primary risk factors for MI, may result from this [33, 34].

Vitamin D is involved in controlling the production of inflammatory cytokines and preventing the proliferation of pro-inflammatory cells, both of which are critical in blocking the processes that lead to atherosclerosis. Atherosclerosis is caused by suppression of the prostaglandin and cyclooxygenase pathways, downregulation of the renin-angiotensin-aldosterone system (RAAS), amplification of anti-inflammatory cytokines, reduction of cytokine-induced manifestation of adhesion molecules, and matrix metalloproteinase 9 [35, 36]. Through a cis-DNA element in the renin gene's promoter region, vitamin D encourages the downregulation of the RAAS system. The hypertension linked to vitamin D shortage is explained by the negative relationship between vitamin D levels and the RAAS system, which permits endothelial dysfunction, plaque development, and eventually MI [37, 38].

By controlling mitochondrial metabolism and energy delivery, vitamin D is known to exert cardioprotective effects [39]. It can affect insulin sensitivity and increase the risk of heart attacks and other cardiovascular events [40–42]. Through non-genomic processes, vitamin D affects the activity of enzymes and the opening of the L-type calcium channel in calcium homeostasis, which in turn affects smooth muscle tone and contractility, which is important for cardiovascular health [26]. Heart issues including hypertrophy and left ventricular remodeling, which are risk factors for MI, have been linked to low vitamin D [43,44].

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Due to a complex web of interactions between vitamin D and estrogen that affect the onset, duration, and prevention of heart attacks in women, vitamin D insufficiency or deficiency has been linked to the development of cardiovascular disorders, particularly MI. In female populations, estrogen has been shown to have positive effects on the cardiovascular system. A decrease in circulating estrogen, which is hypotensive and causes vascular relaxation, can be brought on by low vitamin D levels. Low levels of circulating estrogen raise the incidence of hypertension and decrease protection against artery damage caused by high blood pressure [45, 46].

Additionally, women's cardiovascular risks might worsen after menopause due to a decrease in estrogen levels, which increases their susceptibility to MI when accompanied with a vitamin D deficit [45,47]. Additionally, vitamin D is crucial for safeguarding cardiomyocytes, particularly in females. It has been demonstrated that estrogen and vitamin D tend to preserve cardiomyocytes by three primary mechanisms: regulating contractility, calcium homeostasis, and mitochondrial function. When vitamin D levels are low, cardiomyocyte hypertrophy, faster matrix turnover, and kinetic irregularities might increase a person's vulnerability to cardiovascular diseases like MI, especially in women [47].

Therapeutic Intervention

A significant level of vitamin D deficiency was found in AMI patients (95.9%) compared to the control group (78.4%) in a case-control study involving 222 Iraqi patients (153 men and 69 women) between the ages of 22 and 80 who had AMI and a control group of 225 sex- and age-matched people without CAD. Additionally, vitamin D insufficiency was more common in male AMI patients than in female AMI patients [48]. Low blood vitamin D is an independent risk factor for AMI, according to a comparable case-control research from Bangladesh. Serum vitamin D levels below 20 ng/mL were associated with a greater risk than those over 20 ng/mL.

Three groups were created from the registered research participants. Patients with ST-segment elevation myocardial infarction (STEMI) were in Group A, non-ST-segment elevation myocardial infarction (NSTEMI) was in Group B, and age- and sex-matched people without AMI were in Group C. The STEMI, NSTEMI, and control groups had mean blood vitamin D levels of 20.17 ng/mL, 20.8 ng/mL, and 24.77 ng/mL, respectively. The STEMI and NSTEMI groups had significantly lower levels of vitamin D than the control group ($p < 0.001$ and $p = 0.004$) [49].

According to a Saudi Arabian case-control study, individuals with vitamin D deficiency (serum 25(OH)D <20 ng/mL) were 6.5 times more likely to develop coronary heart disease than those with adequate vitamin D levels (serum 25(OH)D ≥20 ng/mL; 95% CI: 2.7-15, $p < 0.001$). One of the drawbacks is that circulating vitamin D levels were not measured before the patients were diagnosed [50]. A descriptive cross-sectional study from India revealed that blood calcium levels are lower in CAD patients than in controls ($p < 0.0001$) and that vitamin D insufficiency is quite common in the Indian subcontinent.

Of the patients, 44.6% had inadequate vitamin D levels and 51.2% were vitamin D deficient. Three types of serum vitamin D levels were identified: inadequate (<30 nmol/L), insufficient (30-75 nmol/L), and sufficient (>75 nmol/L). The study's descriptive methodology and lack of randomization are among its limitations. Causality was not established since the impact of unidentified confounders on the observed relationship was not taken into consideration. Because the study's data collection was limited to a single site and its sample size was lower, its external validity was questioned [51].

One hundred participants from the Department of Cardiology were assessed in a cross-sectional research carried out over a six-month period in Bangladesh by Chowdhury et al.

Serum 25(OH)D values of less than 20 ng/mL, which indicate moderate to severe deficiency, were identified in 80% of cases, including patients with both STEMI and NSTEMI. The difference between the two groups was found to be statistically significant ($p = 0.04$) despite the limited sample size and shorter follow-up period [52]. Similarly, a high prevalence of vitamin D deficiency was found in a 2015 study by Roy et al. that focused on the association of vitamin D deficiency as a risk factor for AMI in Indians. Severe vitamin D deficiency (<10 ng/mL) was noted in 79.2% of cases and in 46.7% of controls ($p < 0.001$).

Notwithstanding the results, the study's shortcomings included inadequate information on the participants' diets and skin hyperpigmentation, as well as a low representation of women. It's interesting to note that the controls also showed higher-than-expected vitamin D insufficiency [53].

Accordingly, Karur et al. demonstrated that unusually low levels of 25(OH)D were present in 83.5% of patients with significant evidence of

AMI, with 67.5% having levels below 20 ng/mL. However, because the study was not carried out all year round, Karur et al.'s failure to account for seasonal fluctuations constituted a drawback [54].

Vitamin D levels and thrombus burden were shown to be negatively correlated with a p-value of 0.018 in a retrospective analysis of STEMI patients by Uguz et al. [55]. In line with the information above, a meta-analysis of eight observational studies including 9,913 participants—3,411 of whom were MI patients—found that the MI group's vitamin D levels were considerably lower than those of the control group, with a statistical significance of p = 0.007. Sufficient amounts of vitamin D are cardioprotective in Asian and American populations, according to a research [56].

The few studies that were available found more convincing evidence of the impact of vitamin D in the development of MI in the female population in particular. Majeed et al. conducted a cross-sectional investigation in 2022 with 100 postmenopausal women between the ages of 45 and 70. There were 25 controls, 25 with MI, 25 with osteoporosis alone, and 25 with both osteoporosis and MI [57]. In contrast to the control group, which had a mean of 9.20 ± 1.30 ng/mL, women with heart disease alone had mean vitamin D levels of 5.30 ± 0.70 ng/mL, while those with combined heart disease and osteoporosis had mean levels of 4.10 ± 0.30 ng/mL.

According to Ma et al., normotensive postmenopausal women with serum 25(OH)D in the fourth quartile had significantly reduced carotid intima-media thickness (CIMT) and fewer carotid plaques than those in the lower quartiles (p = 0.0039). This three-year research, which included 671 women, used radiographic assessment of the carotid arteries to detect focal thickness greater than 1.5 mm projecting into the lumen or thickening greater than 50% of surrounding tissue. Therefore, it was determined that there is an inverse association between carotid atherosclerosis and 25(OH)D. The study's limitations include its dependence on a single morning fasting value of 25(OH)D without taking albumin and vitamin D binding protein levels into account, as well as its lack of information on confounding variables such physical activity, sun exposure time, and socioeconomic position [59].

Women with vitamin D insufficiency had a greater frequency of metabolic syndrome than women with adequate vitamin D levels, according to a comprehensive assessment of many observational studies. Obesity, high blood pressure, increased triglycerides, and HDL deficiency—all of which are strongly linked to the development of MI—were included in this connection [60]. The frequency of metabolic syndrome, hypertriglyceridemia, and HDL deficit were all greater in postmenopausal women with vitamin D deficiency, which increased the risk of cardiovascular death [61].

Vitamin D intake also seems to have positive effects. A group of patients who had taken vitamin D supplements and maintained blood levels over 20 ng/mL showed a substantially decreased risk of all-cause death, according to data from a retrospective, observational case-control research by Acharya et al. Additionally, individuals with serum levels greater than 30 ng/mL showed significant outcomes, indicating a decreased risk of MI in comparison to untreated patients with levels less than 20 ng/mL. The inability to assess treatment compliance and duration, as well as the failure to take racial disparities into account, were among the limitations [29].

Cruijsen et al. performed a prospective cohort investigation in which post-MI patients between the ages of 60 and 80 were monitored for 12 years. Their blood vitamin D levels were categorized into tertiles, with cut-offs at 18.3 and 26.2 ng/mL. The findings showed that the risk of recurrent CVDs and death rates dropped when vitamin D levels increased across tertiles. Interestingly, in comparison to the lower tertile, the hazard ratios for the mid and upper tertiles were 0.76 and 0.67, respectively. Those who consumed more calcium further demonstrated this unfavorable association [62].

Lower levels of vitamin D are independently linked to poor reperfusion, according to a sequential cohort of 450 patients treated with percutaneous coronary intervention (PCI) after being hospitalized for STEMI and divided based on tertile values of 25(OH)D [63]. In fact, Naesgaard et al. found that patients with lower vitamin D levels had a higher hazard ratio for follow-up all-cause mortality, indicating that vitamin D is a measure of morbidity, including the risk of sudden cardiac death in post-MI individuals. The scientists did point out a drawback, though: there is a significant chance of confounding and power estimates cannot be directly applied to observational research. As a result, judgments are just speculative and not conclusive [64].

To determine the relationship between 25(OH)D levels and the kind of MI, Safaie et al. carried out a prospective case-control research. The study included 88 patients in total, 40 of whom

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were in the STEMI group and 48 of whom were in the NSTEMI group. 59.1% of individuals had hypovitaminosis, with STEMI patients having a much higher prevalence rate (77.5% vs. 43.7%, p = 0.001). The STEMI group's blood plasma levels of 25(OH)D were significantly lower than those of the NSTEMI group (13.5 ± 7.7 and 24.3 ± 14.9, respectively). A significant risk factor for the development of STEMI type was found to be vitamin D insufficiency (odds ratio = 8.1, 95% CI: 2.3-28.2, p = 0.001) [65].

Tokarz et al. recorded the 25(OH)D levels of 59 patients with an uncomplicated MI over the course of the year to examine if there were any differences between the January–March and September–December time frames. No patient had vitamin D levels within the specified reference range, and 53 patients (89.9%) of the 59 patients who were recruited in the research had levels below 20 ng/mL. In contrast to the United States, where levels below 10 ng/mL are deemed abnormal, this investigation was carried out in Poland, where changed results indicated insufficient vitamin D levels.

Seasonal variations were seen, with the maximum vitamin D levels occurring between October and December and the lowest levels occurring between January and March, which is the first quarter of the year. This pattern was also observed in the northern hemispheres, where sunshine is less strong, notwithstanding the differences between normal and pathological vitamin D levels. Additionally, the study found that vitamin D levels were substantially higher in normoglycemic individuals (9.2 (2.3-16.8) ng/mL) than in patients with diabetes mellitus (8.5 (2.5-13.3) ng/mL) or impaired glucose tolerance (2.3 (2.3-3.9) ng/mL) (p = 0.01) [66].

Patients between the ages of 45 and 75 who had had at least one colorectal adenoma removed within 120 days prior to enrollment and had no residual disease were recruited for a randomized, multicenter, double-blind, placebo-controlled trial of calcium and vitamin D supplementation for the prevention of colorectal adenomas in the United States. Each of the four groups of patients was required to take two tablets, which may contain either 1000 IU of vitamin D3, 1200 mg of calcium, both agents, or a placebo. Along with side effects like MI, whether or not revascularization was used, the incidence of adenomas was examined.

II. CONCLUSIONS

A recently discovered risk factor for CVDs, especially MI, is vitamin D insufficiency. A number of observational studies have shown a relationship between the frequency of vitamin D insufficiency and the incidence of MI in patients; however, it is unclear if this is a marker or a risk factor for the condition. Supplementing with vitamin D to reduce the risk of MI has been the subject of contentious and clinically questionable randomized controlled studies.

To find relevant information on the role of vitamin D in primary and secondary prevention of MI, lengthier longitudinal studies with high sample numbers across diverse ethnic groups are essential. It is necessary to investigate the relationship between circulating levels of 25(OH)D and other risk variables, such as blood glucose and cholesterol levels. Establishing precise criteria for CVD is crucial for optimal diagnostic and therapeutic implications in a clinical context. The current guidelines for vitamin D deficiency and insufficiency cut-offs are connected to bone metabolism.

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