

Review Article

The Use of Vitamin D in Heart Failure Management: A Literature Review

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Abstract: Heart failure is a clinical syndrome characterized by structural or functional cardiac dysfunction that leads to hemodynamic disturbances and increased morbidity and mortality. Vitamin D, a prohormone with a vital role in metabolism, is known to exert cardioprotective effects through anti-inflammatory and anti-fibrotic mechanisms, modulation of calcium homeostasis, and inhibition of the renin–angiotensin–aldosterone system. Vitamin D deficiency has been shown to be highly prevalent among patients with heart failure and is correlated with poorer clinical outcomes. Numerous preclinical studies support the protective role of vitamin D against ventricular remodeling and myocardial hypertrophy; however, clinical trials in humans have yielded inconsistent and often contradictory findings regarding its effects on systolic function, surrogate parameters, and primary outcomes such as mortality and hospitalization. Consequently, vitamin D supplementation cannot yet be recommended as a standard therapy for heart failure, except in patients with confirmed deficiency. Further research with robust methodologies and stratified populations remains essential to elucidate its potential clinical benefits.

Keywords: Cardioprotective; Heart Failure; Myocardial Hypertrophy; Renin; Vitamin D.

1. Introduction

Heart failure (HF) is a complex clinical syndrome characterized by signs and symptoms resulting from structural and/or functional abnormalities of the heart, accompanied by elevated levels of natriuretic peptides and/or objective evidence of pulmonary or systemic congestion (Ponikowski et al., 2021). It represents a pathological condition in which the heart fails to maintain adequate circulation to meet the metabolic demands of peripheral tissues, leading to diminished perfusion and oxygen delivery (McDonagh et al., 2023). The pumping capacity of the heart is primarily determined by myocardial contractility, which itself depends on sarcomere function and is influenced by preload (volume load), afterload (pressure load), and heart rate (Borlaug & Kass, 2020).

The burden of heart failure continues to rise globally, including in Indonesia. According to the Indonesian Health Survey (SKI, 2023), the national prevalence of heart disease reached 0.85%, with the highest prevalence observed in the Special Region of Yogyakarta (1.67%) and the Mountain Papua Province (1.65%). Local data from the Lampung Provincial Health Office reported that hospitalizations due to heart failure increased from 637 cases in 2015 to 1,106 cases in 2016, marking a 42.6% rise, while outpatient cases grew from 794 to 1,462 during the same period an increase of 45.6%. These data indicate a growing public health concern that demands more effective management and preventive strategies.

Epidemiologically, the annual incidence of heart failure in men increases sharply with age, from 3 per 1,000 in individuals aged 50–59 years to 27 per 1,000 in those aged 80–89 years, while women exhibit relatively lower incidence rates (Roger, 2021). Major risk factors include age, hypertension, diabetes mellitus, obesity, smoking, and sedentary lifestyle (Yancy

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et al., 2022). Despite significant advances in the understanding and management of HF, its morbidity and mortality remain high, particularly in low- and middle-income countries where awareness and preventive measures are limited.

Current management of heart failure includes both pharmacological and non-pharmacological interventions. The standard pharmacological therapies consist of Angiotensin-Converting Enzyme Inhibitors (ACE inhibitors), Beta-Blockers, Mineralocorticoid Receptor Antagonists (MRAs), Angiotensin Receptor Blockers (ARBs), Angiotensin Receptor–Neprilysin Inhibitors (ARNIs), diuretics, and Isosorbide Dinitrate (ISDN) (McDonagh et al., 2023). Non-pharmacological approaches focus on lifestyle modifications, such as fluid and weight monitoring, dietary regulation, and cardiac rehabilitation exercises (Ponikowski et al., 2021). However, these approaches often fail to fully address the complex pathophysiology of heart failure, prompting the exploration of adjunctive therapies such as micronutrient supplementation. One promising adjunctive therapy under investigation is vitamin D supplementation.

Vitamin D has been increasingly recognized not only for its role in calcium and phosphate homeostasis but also for its regulatory effects on cardiovascular physiology. Mechanistically, vitamin D modulates cardiomyocyte gene expression and signaling pathways, exerts anti-inflammatory, anti-apoptotic, and anti-fibrotic actions, and prevents maladaptive cardiac remodeling and aberrant calcium handling (Li et al., 2018). Furthermore, preclinical studies suggest that vitamin D deficiency contributes to the activation of the renin–angiotensin–aldosterone system (RAAS), a key driver of heart failure progression (Andress, 2019). Despite these mechanistic insights, clinical evidence regarding vitamin D supplementation in heart failure remains inconclusive. Several randomized controlled trials have demonstrated potential improvements in left ventricular function and exercise capacity following vitamin D therapy (Witham et al., 2014; Witte et al., 2016), while others found no significant benefits on primary outcomes such as mortality, hospitalization rates, or functional status (Dalbeni et al., 2020).

This inconsistency underscores the need for further research utilizing robust methodologies and stratified populations to determine which subgroups of patients may derive the greatest benefit. The problem addressed in this study lies in the inconsistent clinical evidence concerning vitamin D supplementation as an adjunctive therapy for heart failure, particularly regarding its role in improving cardiac function and clinical outcomes. The proposed approach of this research is to synthesize current findings, identify methodological limitations in existing trials, and explore potential mechanisms that may explain the variability in outcomes. The main contributions of this study are as follows: (1) To provide an updated review of the epidemiological and pathophysiological aspects of heart failure in the Indonesian and global context; (2) To critically assess the current evidence on the role of vitamin D in cardiac function and remodeling; (3) To highlight gaps in existing clinical research and propose directions for future studies; and (4) To formulate clinical considerations for the potential inclusion of vitamin D supplementation in heart failure management.

2. Preliminaries or Related Work or Literature Review

2.1. Pathophysiology and Epidemiology of Heart Failure

Heart failure (HF) is a progressive clinical syndrome characterized by the inability of the heart to maintain adequate blood circulation to meet metabolic demands. The underlying pathophysiology involves complex interactions between structural cardiac abnormalities, neurohormonal activation, and systemic inflammation (Ponikowski et al., 2021). Chronic activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system (SNS) contributes to myocardial hypertrophy, fibrosis, and adverse ventricular remodeling, which in turn exacerbate cardiac dysfunction (McDonagh et al., 2023). Globally, the prevalence of HF continues to rise, with an estimated 64 million people affected worldwide (Groenewegen et al., 2020).

In Indonesia, recent data from the Indonesian Health Survey (SKI, 2023) reported a heart disease prevalence of 0.85%, with regional variations suggesting disparities in healthcare access and preventive measures. The prevalence increases significantly with age, affecting more than 10% of individuals over 70 years old (Roger, 2021). The most common risk factors include hypertension, diabetes mellitus, obesity, and coronary artery disease (Yancy et al., 2022). Despite substantial improvements in pharmacological management, HF remains a

major cause of hospitalization and mortality worldwide, emphasizing the need for adjunctive and preventive therapeutic strategies.

2.2. Vitamin D: Biological Mechanisms and Cardiovascular Function

Vitamin D, traditionally known for its role in calcium and phosphate metabolism, has emerged as a key regulator in cardiovascular health. The active form, 1,25-dihydroxyvitamin D₃ (calcitriol), binds to vitamin D receptors (VDRs) expressed in cardiomyocytes, vascular smooth muscle cells, and endothelial cells, influencing gene transcription involved in myocardial contractility, inflammation, and fibrosis (Li et al., 2018). Through these pathways, vitamin D exerts several cardioprotective mechanisms, including suppression of pro-inflammatory cytokines (IL-6, TNF- α), inhibition of myocardial hypertrophy, modulation of calcium handling, and downregulation of the RAAS pathway (Andress, 2019). Experimental studies have demonstrated that vitamin D deficiency leads to upregulation of renin expression, increased cardiac fibrosis, and impaired diastolic function (Bae et al., 2011). Conversely, supplementation has shown to attenuate ventricular remodeling and improve myocardial relaxation in animal models (Chen et al., 2019). These biological findings provide a strong rationale for exploring the therapeutic potential of vitamin D in patients with heart failure.

2.3. Clinical Evidence on Vitamin D and Heart Failure Outcomes

Several observational studies have established a significant association between low serum 25-hydroxyvitamin D levels and adverse cardiovascular outcomes. Zittermann et al. (2012) reported that vitamin D deficiency (<20 ng/mL) was prevalent in up to 70% of HF patients and was independently associated with higher mortality and hospital readmission rates. Similarly, Drechsler et al. (2010) found that lower vitamin D levels predicted worse functional capacity and increased all-cause mortality in chronic HF populations. Interventional studies, however, have produced mixed results. The VINDICATE trial (Witte et al., 2016) demonstrated that high-dose vitamin D supplementation (4,000 IU/day) over 12 months significantly improved left ventricular ejection fraction (LVEF) and reduced left ventricular end-systolic volume in patients with systolic HF. In contrast, the EVITA trial (Schroten et al., 2017) found no significant effect on mortality or cardiovascular hospitalizations despite similar dosing regimens. Meta-analyses such as that by Dalbeni et al. (2020) further highlight these inconsistencies, showing minor improvements in surrogate endpoints (e.g., NT-proBNP reduction, LVEF improvement) but not in major clinical outcomes such as survival.

2.4. Identified Gaps and Theoretical Implications

Despite biological plausibility and encouraging preclinical data, current clinical evidence remains inconclusive regarding the efficacy of vitamin D supplementation in improving HF outcomes. Several methodological limitations have been identified in previous studies, including heterogeneous dosing strategies, short follow-up durations, variations in baseline vitamin D levels, and the inclusion of non-stratified patient populations (Pilz et al., 2019). Moreover, confounding factors such as seasonal variation, comorbidities, and concurrent medication use often obscure the direct impact of vitamin D on cardiac function. From a theoretical standpoint, it is hypothesized that vitamin D exerts its beneficial effects primarily in populations with confirmed deficiency and in early stages of heart failure where ventricular remodeling is still reversible (Bikle, 2021). This suggests that future studies should adopt a precision-medicine approach stratifying patients based on vitamin D status, genetic polymorphisms affecting VDR activity, and stage of HF progression.

2.5. Research Position and Contribution of the Present Study

The present literature review distinguishes itself from prior works by providing an integrative synthesis of both molecular and clinical evidence concerning vitamin D in heart failure management. While earlier studies (Witte et al., 2016; Schroten et al., 2017; Dalbeni et al., 2020) primarily focused on clinical endpoints, this study bridges the mechanistic underpinnings with observed clinical phenomena, identifying potential explanatory models for the observed inconsistencies. The review further proposes a conceptual framework emphasizing the timing, dosage, and biological context of vitamin D intervention as critical determinants of therapeutic efficacy. By addressing methodological heterogeneity and proposing a stratified clinical approach, this paper contributes to the ongoing discourse on

the adjunctive role of vitamin D in heart failure therapy. The findings are expected to inform future research design, support guideline development, and encourage clinicians to consider individualized supplementation strategies based on biochemical and physiological evidence.

3. Materials and Method

This study is a literature review conducted by collecting data from various sources relevant to the research topic. The analytical approach in this review follows the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The process began with a comprehensive search and selection of literature from several indexed scientific databases, including PubMed and Google Scholar.

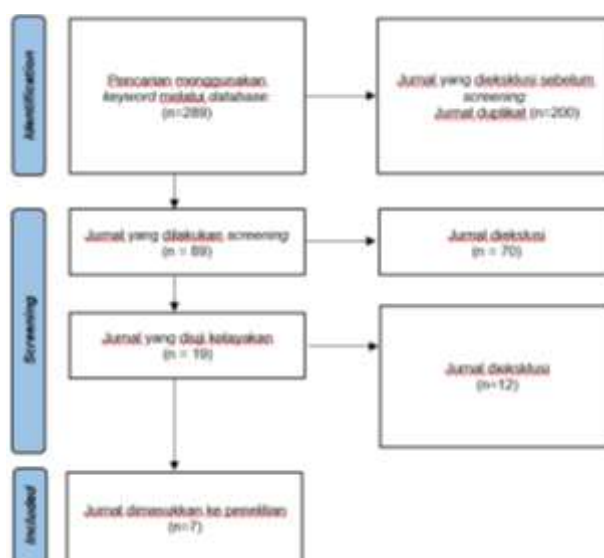


Figure 1. PRISMA flow diagram.

The literature search was conducted using a combination of keywords related to the study focus, namely vitamin D and heart failure. To ensure the relevance and quality of the data, inclusion criteria were established, which included accessibility of the articles, a publication year range between 2021 and 2025, and availability of the full-text version. Following the selection process, a total of seven (7) articles met the predefined criteria. Accordingly, this review aims to provide an overview of the potential role of vitamin D in heart failure management, thereby contributing to future clinical decision-making processes.

4. Results and Discussion

Vitamin D is a prohormone that plays a crucial role in daily metabolic processes. Over the past decade, an increasing number of researchers have explored its role in various non-skeletal conditions. Vitamin D deficiency has been associated with an elevated risk of malignancies, hypertension, diabetes mellitus, cardiovascular diseases, and even infectious diseases (Holick, 2011). The definition of vitamin D deficiency status, however, remains a matter of debate among clinicians and researchers. Therefore, a consensus is still needed to establish a clear “cut-off” value for serum 25(OH)D levels in order to optimize therapeutic strategies and avoid potential issues related to vitamin D treatment.

4.1. Mechanisms of Vitamin D in the Cardiovascular System

The influence of vitamin D on the cardiovascular system is primarily linked to the activity of vitamin D receptors (VDR) and the enzyme 1-alpha-hydroxylase, both of which are expressed in vascular smooth muscle cells, endothelial cells, and cardiomyocytes. These receptors enable vitamin D to modulate calcium influx, thereby influencing vascular smooth muscle contractility, while also regulating the secretion of nitric oxide (NO) and acting as a negative regulator of the renin-angiotensin-aldosterone system (RAAS). Through these mechanisms, vitamin D contributes to the maintenance of vascular tone, endothelial integrity, and blood pressure homeostasis. Consequently, vitamin D exerts inhibitory effects on cardiac hypertrophy and may possess anti-heart failure properties, highlighting its potential role in cardiovascular protection.

4.2 Preclinical and Early Clinical Evidence

Evidence from preclinical studies consistently demonstrates the cardioprotective effects of vitamin D, including its anti-inflammatory, anti-apoptotic, and anti-fibrotic properties, as well as its capacity to prevent ventricular remodeling and the progression of hypertrophy toward heart failure. Vitamin D suppresses RAAS activity—an overactive RAAS is a well-known driver of pathological hypertrophy. In VDR knockout animal models, excessive RAAS stimulation led to cardiac hypertrophy, confirming vitamin D's regulatory role in this pathogenic pathway. Moreover, several observational studies and randomized clinical trials (RCTs) have shown that vitamin D supplementation may improve surrogate cardiac parameters such as left ventricular ejection fraction (LVEF), left ventricular dimensions, and NT-proBNP levels, especially among patients with heart failure with reduced ejection fraction (HFrEF) or severe vitamin D deficiency.

4.3 Clinical Effectiveness of Vitamin D in Heart Failure

The clinical benefits of vitamin D supplementation in heart failure patients are supported by a large-scale cohort study using data from Japan's JROAD-DPC national registry, involving 93,692 hospitalized heart failure patients. The findings demonstrated that vitamin D supplementation was associated with reduced in-hospital mortality. A propensity score-matched analysis of 10,974 patients showed lower mortality rates in those who received vitamin D supplementation—both overall and at 7-day and 30-day intervals (OR 0.67, 0.34, and 0.56; $p < 0.001$). These effects remained significant after multivariate adjustment for major comorbidities such as hypertension, chronic kidney disease, atrial fibrillation, and the use of mechanical ventilation or catecholamines.

Interestingly, subgroup analyses revealed that patients with osteoporosis and those not receiving ACE inhibitors or ARBs appeared to derive greater benefit from vitamin D supplementation. This may be attributed to vitamin D's modulation of RAAS inhibition, calcium homeostasis, and suppression of myocardial inflammation and fibrosis. Nevertheless, the clinical benefits are not universal, and further targeted RCTs focusing on high-risk populations are required to establish definitive therapeutic recommendations.

4.4 Prevalence and Pathophysiological Role of Vitamin D Deficiency

Vitamin D deficiency (VDD) is highly prevalent among heart failure patients, even in tropical countries with abundant sunlight exposure. Studies have shown that hospitalized heart failure patients have significantly lower 25(OH)D levels compared to healthy controls, with a deficiency prevalence of 27.3% versus 9.8%, respectively. VDD is associated with adverse metabolic profiles, including elevated hemoglobin A1c, total cholesterol, LDL, and parathyroid hormone (PTH) levels, along with decreased glomerular filtration rate (GFR). These alterations contribute to the pathophysiology of heart failure through mechanisms involving metabolic dysregulation, endothelial dysfunction, RAAS hyperactivation, and secondary hyperparathyroidism—all of which can exacerbate cardiac remodeling and functional decline.

4.5 The U-Shaped Relationship and Atherosclerotic Risk

Interestingly, the relationship between vitamin D levels and cardiovascular outcomes appears to follow a U-shaped curve. Both deficiency and excessive supplementation may have detrimental effects, particularly concerning atherosclerotic risk. The underlying mechanisms are complex and multifactorial. On one hand, vitamin D exhibits anti-inflammatory effects, improves endothelial function by enhancing nitric oxide synthesis, and reduces oxidative stress. On the other, vitamin D upregulates genes involved in cholesterol efflux, thereby inhibiting foam cell formation—a key step in atherogenesis. When balanced, these effects promote plaque stability; however, excessive vitamin D may lead to vascular calcification, negating its benefits.

4.6 Recent Clinical Findings

Recent human clinical trials investigating the role of vitamin D in heart failure have yielded inconsistent results. While some studies report improvements in LVEF, blood pressure, and quality of life, others fail to demonstrate significant benefits, including on mortality outcomes. These discrepancies likely stem from differences in age, sex, ethnicity, dosage, supplement type, and study design, particularly in the assessment of vitamin D status. A meta-analysis of 10 RCTs involving 1,099 heart failure patients found no significant

improvement in LVEF, left ventricular end-diastolic diameter (LVEDD), brain natriuretic peptide (BNP), or serum 25(OH)D levels compared with controls.

Moreover, the absence of heterogeneity across most parameters strengthens the conclusion that vitamin D supplementation does not markedly improve ventricular remodeling or systolic function. Some studies even report potential adverse effects associated with high-dose, long-term supplementation, such as hypercalcemia and an increased need for mechanical circulatory support. These findings underscore the importance of establishing optimal dosing strategies to maximize potential benefits while minimizing risks.

4.7 Prognostic Relevance of Vitamin D Deficiency

A separate study examining the relationship between vitamin D deficiency and the severity of chronic heart failure (CHF)—using NYHA functional class and NT-proBNP levels—revealed that 90% of patients had either deficient or insufficient vitamin D levels. Although NT-proBNP levels rose with increasing NYHA class, vitamin D levels did not vary significantly across severity categories and showed no correlation with NT-proBNP. This suggests that while VDD is highly prevalent, it may not serve as a reliable prognostic marker for disease severity or clinical outcomes in CHF patients.

Large-scale meta-analyses have also failed to consistently demonstrate benefits of vitamin D supplementation on primary cardiovascular outcomes, such as mortality reduction, heart failure–related hospitalizations, or improved functional capacity. These inconsistencies have fueled debate over whether VDD is merely a marker of poor prognosis or an active causal factor in heart failure progression. Consequently, current evidence does not support routine vitamin D supplementation for all heart failure patients. It remains advisable only for those with confirmed deficiency, while further well-designed, large-scale RCTs with standardized dosing and stratified patient populations are essential to elucidate vitamin D's true clinical value.

In summary, although biological and preclinical evidence supports the cardioprotective potential of vitamin D through anti-inflammatory, anti-fibrotic, and RAAS-modulating mechanisms, clinical findings remain inconclusive. Vitamin D deficiency is indeed common and associated with worse outcomes in heart failure, yet current supplementation trials have not demonstrated consistent benefits in improving systolic function, ventricular remodeling, or clinical endpoints. Therefore, while maintaining adequate vitamin D levels is important for general health, its use as a therapeutic intervention in heart failure should be individualized and guided by confirmed deficiency until stronger clinical evidence emerges.

5. Comparison

When compared with existing state-of-the-art studies, this literature review provides a more comprehensive and integrative understanding of the role of vitamin D in heart failure management by synthesizing both preclinical and clinical evidence published between 2021 and 2025. Previous systematic reviews and meta-analyses (before 2021) often focused narrowly on the correlation between vitamin D deficiency and heart failure incidence, without differentiating among heart failure phenotypes or considering the interaction of vitamin D with neurohormonal pathways such as the renin–angiotensin–aldosterone system (RAAS). In contrast, the present review not only evaluates the biochemical mechanisms underlying vitamin D's cardioprotective effects but also integrates emerging evidence on dose-response relationships, population-specific outcomes, and potential adverse effects from over-supplementation.

Furthermore, compared to previous analyses that emphasized general associations, this study highlights a shift in research direction toward understanding vitamin D's therapeutic implications rather than its diagnostic relevance. While earlier works concluded that vitamin D deficiency merely serves as a marker of poor prognosis, more recent evidence including large-scale cohort data from the JROAD-DPC registry suggests that supplementation may have protective effects in specific subpopulations, particularly those with severe deficiency or lacking ACEi/ARB therapy. This review critically contrasts these findings and emphasizes the need for stratified intervention trials to identify which patient groups may derive true clinical benefit. In addition, this study contributes to the ongoing scientific discourse by comparing inconsistent outcomes across recent randomized controlled trials (RCTs). Unlike earlier meta-analyses that relied on limited sample sizes or heterogeneous methodologies, the present synthesis considers methodological quality, dosage standardization, and population diversity as critical parameters influencing the interpretation of vitamin D efficacy.

By doing so, it provides a more nuanced and balanced conclusion that bridges the gap between mechanistic plausibility and clinical applicability. Overall, the comparison with state-of-the-art research indicates that while vitamin D continues to be recognized for its biological plausibility in cardiovascular protection, robust evidence for its therapeutic benefit in heart failure remains inconclusive. The present review distinguishes itself by systematically outlining the strengths, limitations, and emerging directions in the field, offering a clearer framework for future precision-based supplementation strategies and targeted clinical investigations.

6. Conclusion

Theoretically, vitamin D possesses a significant cardioprotective potential through multiple mechanisms, including its anti-inflammatory, anti-fibrotic, and calcium homeostasis-regulating effects, as well as its inhibition of the renin-angiotensin-aldosterone system (RAAS). These mechanisms collectively contribute to the prevention of ventricular hypertrophy and cardiac remodeling in heart failure. Vitamin D deficiency (VDD) has been shown to be highly prevalent among patients with heart failure and is consistently associated with poorer clinical outcomes, including increased morbidity and mortality rates. However, evidence from clinical trials investigating vitamin D supplementation in heart failure remains inconclusive and inconsistent, with variable findings regarding its impact on systolic function, surrogate markers, and primary clinical endpoints. Consequently, vitamin D supplementation cannot yet be recommended as a standard therapeutic intervention for heart failure, except in patients with confirmed deficiency. Further well-designed, large-scale, and stratified clinical studies are essential to clarify its true clinical efficacy and to identify patient subgroups that may derive meaningful benefit from such supplementation.

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