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Literature Review

Unveiling the Link Between Low Testosterone and Worsening Heart Failure Symptoms: A Literature Review

Ryan Ardiansyah[*1], Akhmad Sandy Sauqy[2]
[1]General Practitioner, Namira Islamic Hospital, East Lombok, 83611, Indonesia
[2]General Practitioner, West Nusa Tenggara Province General Hospital, Mataram, 83111, Indonesia
[*]Corresponding Author: shorinjiryan9@gmail.com

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ABSTRACT

Background: Heart failure can result from a variety of factors, including hypertension, coronary artery disease, diabetes, obesity, smoking, and genetic predispositions. Research indicates that testosterone levels are markedly lower in patients with heart failure compared to healthy individuals, suggesting that low testosterone may play a role in the onset or progression of heart failure. **Objective:** This review endeavors to examine the impact of low testosterone levels on the clinical manifestations of heart failure patients. **Methods:** A literature review was conducted across three databases, Google Scholar, PubMed, and Science Direct, employing the keywords "testosterone" and "heart failure." The search was further refined through specific inclusion and exclusion criteria, culminating in data from eight international journals. **Results:** Testosterone deficiency was associated with deteriorated outcomes in men diagnosed with HFpEF, with lower testosterone levels corresponding to more severe symptoms and diminished quality of life. It influences myocardial contractility, energy metabolism, apoptosis, and cardiac remodeling processes. **Conclusion:** Men suffering from heart failure with concomitant low testosterone levels are generally observed to have increased mortality rates and less favorable prognoses.

Keywords: heart failure, low testosterone level, worsening symptoms

1. Introduction

Heart failure is a complex clinical syndrome characterized by the inability of the heart to pump enough blood to meet the body's needs. It can be caused by a variety of factors, including high blood pressure, coronary artery disease, diabetes, obesity, smoking, and genetics [1]. The condition is associated with symptoms such as shortness of breath, ankle swelling, and fatigue, and is diagnosed based on demonstration of underlying cardiac dysfunction. The pathophysiology of heart failure is multifaceted and can be caused by structural or functional cardiac abnormalities [2].

According to recent studies, the prevalence of heart failure is increasing, especially in developed countries with aging populations. Heart failure has emerged as one of the leading causes of hospitalization among the elderly population. The prevalence of heart failure varies according to the studied population but is estimated to impact approximately 26 million individuals globally [3]. The prevalence of heart failure has increased over the past 20 years, and this is partly due to the aging of the population and the increasing prevalence of risk factors such as obesity and diabetes. Mortality rates associated with heart failure remain high, despite improvements in treatment strategies and overall outcomes of heart failure patients. Mortality rates are higher in patients with reduced ejection fraction compared to patients with preserved ejection fraction [4].

Testosterone is the principal male hormone responsible for regulating sex differentiation, the development of male secondary sexual characteristics, spermatogenesis, and fertility. It plays a crucial role in various bodily

functions, including the development of male reproductive tissues and the maintenance of muscle mass and bone density. Testosterone levels can be indicative of a range of conditions, and it is essential to understand the normal ranges and implications of low levels. The normal range for testosterone levels in adult men is typically between 280 and 1,100 nanograms per deciliter (ng/dL). However, the American Urological Association (AUA) guideline suggests using a cutoff for low testosterone of 300 ng/dL when evaluating adult men [5].

Studies have shown that heart failure is more prevalent in elderly populations and is often associated with uncontrolled hypertension. Testosterone levels have been observed to be markedly diminished in patients with heart failure when compared to healthy individual controls [6]. This suggests that low testosterone levels may play a role in the development or progression of heart failure.

Furthermore, sarcopenia, or the loss of muscle mass, is a common feature in heart failure patients and affects 20-47% of them. Sarcopenia is an independent predictor of impaired functional capacity in heart failure patients, even after adjusting for clinically relevant variables. Lower testosterone levels have been associated with increased sarcopenia in heart failure patients, indicating that testosterone therapy might help improve muscle mass and physical performance [7].

The link between testosterone and heart failure is intricate and needs more research. Nonetheless, current evidence indicates that testosterone therapy could offer benefits to heart failure patients, especially in enhancing muscle mass and physical capacity performance. Additional research is needed better to understand the role of testosterone in heart failure and to determine the optimal therapeutic strategy for its management. Based on these data, we decided to further investigate how low testosterone levels affect the symptoms of heart failure patients.

2. Methods

The research methodology employed is a Literature Study with a Literature Review type. The strategy for selecting references or literature involved conducting searches across Google Scholar, PubMed, and Science Direct databases utilizing the keywords "testosterone" and "heart failure." This literature search was conducted in accordance with predefined inclusion and exclusion criteria. The inclusion criteria encompassed: 1) journal publications from the year 2010 to the present, 2) research articles related to the impact of low testosterone levels on the symptoms of heart failure patients, 3) articles that are research journals rather than literature reviews, 4) both national and international journals, and 5) full-text journal articles. Exclusion criteria included: 1) journals published prior to 2010, 2) journals providing only abstracts, and 3) articles that do not address the influence of low testosterone levels on the symptoms of heart failure patients.

Searches conducted on various academic databases yielded 169,000 journals via Google Scholar, 400 journals through PubMed, and 17,923 journals via Science Direct. In total, this resulted in 187,323 journals. Subsequent screening was performed according to specific inclusion and exclusion criteria, narrowing the selection to 8 journals. Ultimately, ten international journals were analyzed and utilized as data sources in this study.

3. Results

Analysis of 8 journals on how low testosterone levels affect the symptoms of heart failure patients showed that there were four studies [8,11] that displayed Hazard Ratio (HR) values, all of which showed that testosterone levels were associated with worsening symptoms in heart failure patients (Table 1).

Table 1. Results of each journal on how low testosterone levels affect the symptoms of heart failure patients

No.	Author	Prognosticators	HR	Patients without testosterone deficiency	Patients with testosterone deficiency	Before Testosterone Supplementation	After Testosterone Supplementation
1.	Hai-Yun Wu et al[10]	TT, increased per 1 nmol	0.97				
2.	Anna Florvaag et al[12]	IVSd (mm)		12	11		
		Ejection fraction (%)		39	38		
		PAP (mmHg + CVP)		27	25		
3.	Martin Stout et al[13]	NYHA				2.5	1.8
4.	Ahmad Mirdamadi et al[14]	Quality of life (Short Form Health Survey questionnaire)				128.96	130.95
		Ejection fraction				34.52	37.12
5.	Marcelo Rodrigues dos Santos et al[11]	Readmission within 90 days	2.77				
		Mortality	4.65				
6.	Akiomi Yoshihisa et al[15]	In the Kaplan–Meier analysis, all-cause mortality progressively increased from the first to the fourth quartile (log-rank, p = 0.010). Patients were divided into quartiles based on their TT levels: first (632 ng/dl ≤ TT, n = 154), second (463 ≤ TT ≤ 631, n = 155), third (462 ≤ TT ≤ 301, n = 156), and fourth quartiles (TT ≤ 300, n = 153). The TT of ≤ 300 ng/dl is generally considered a low TT level.					
7.	Marina Navarro-Pen˜alver et al[16]	NYHA class				2.18	2.8
		LVEF, %				29.88	29.90
8.	Ahmed Hamam et al[17]	NYHA III (n)		16	20		
		Testosterone levels can be used as independent predictors of NYHA class III.					P value = 0.008

Hai-Yun Wu et al.[10] found that each one nmol increase in testosterone reduces the risk of worsening heart failure symptoms by 0.97 times. Additionally, Marcelo Rodrigues dos Santos et al.[11] demonstrated that testosterone levels are linked to death and readmission within 90 days. Patients with heart failure who experience a decline in testosterone levels face a 2.77 times higher risk of readmission within 90 days and a 4.65 times higher risk of mortality.

Anna Florvaag et al.[12] compared patients with testosterone deficiency with those who did not; they found that IVSd, ejection fraction, and PAP of the group that did not have testosterone deficiency had better values than the group with testosterone deficiency. Ahmed Hamam et al.[13-17] also compared the number of NYHA III cases between patients who had testosterone deficiency and those who did not, and the results were found to be more in the testosterone-deficient group. In addition, this study also found that testosterone levels can be used as independent predictors of NYHA class III (p-value = 0.008).

Three studies examined the effect of testosterone levels on symptoms of heart failure patients while providing interventions in the form of testosterone supplementation. Martin Stout et al.[13] showed that the NYHA grade decreased after testosterone supplementation. In contrast, Ahmad Mirdamadi et al.[14] showed that the quality of life and ejection fraction of patients who received testosterone supplementation improved. Marina Navarro-Pen˜alver et al.[16] showed that after testosterone supplementation, there was a decrease in NYHA degree and an increase in LVEF. One study[15] found that all-cause mortality progressively increased from the first to the fourth quartile (log-rank, p = 0.010).

4. Discussion

Testosterone levels tend to decline after the age of 40, and this reduction has been linked to an increased risk of all-cause mortality and cardiovascular disease (CVD) [18]. Heart failure, a chronic condition marked by the heart's inability to pump sufficient blood to meet the body's requirements, is frequently associated with other

health complications, including hormonal imbalances. One such hormone is testosterone, which plays a crucial role in maintaining men's health and well-being. Endogenous and exogenous testosterone can have both positive and negative effects on the cardiovascular system. On one hand, testosterone has been shown to have cardioprotective effects, such as reducing the risk of heart attack and stroke. Conversely, elevated testosterone levels may promote atherosclerosis, a condition where arteries get clogged with plaque, reducing blood flow and raising the risk of heart attack and stroke [19].

Depression and anxiety are prevalent among patients suffering from heart failure, and diminished testosterone levels have been associated with heightened severity of depression. In a study involving 78 Caucasian male patients diagnosed with dilated cardiomyopathy and chronic heart failure, researchers identified a correlation between testosterone levels and depression severity, as measured by the PHQ-9 Depression Test Questionnaire [20]. This indicates that management of testosterone deficiency could potentially contribute to the enhancement of mood and overall quality of life in individuals with heart failure.

A recent study published in the Journal of Cardiovascular Development and Disease investigated the relationship between testosterone deficiency and HFpEF in men. The study found that testosterone deficiency was associated with a worsening of HFpEF in men. The researchers measured testosterone levels in 100 men with HFpEF and found that those with lower testosterone levels had worse symptoms and a lower quality of life [17]. Testosterone deficiency (TD) is a well-established, significant medical condition that adversely affects male sexuality, overall health, and quality of life. The symptoms encompass decreased libido, erectile dysfunction, reduced energy levels, depressed mood, irritability, and a diminished sense of well-being. In an appropriate clinical context, the diagnosis of TD is typically confirmed by measuring low serum concentrations of total testosterone (e.g., < 200 ng/mL) obtained in the early morning. However, there is no definitive value that reliably discriminates men who exhibit signs and symptoms of TD from those who do not, nor those who are likely to respond to treatment. The interpretation of total testosterone levels is complicated by individual variability, fluctuations in serum sex hormone-binding globulin (SHBG), and differences in androgen sensitivity [18]. Moreover, substantial debate persists regarding the accuracy of current commercial testosterone assays, particularly those indicating testosterone levels within the lower end of the “normal” range [19].

Testosterone has a variety of effects on cardiovascular physiology, which may impact the hormone’s effect on CVD. Clinical data strongly suggest that low testosterone levels are associated with longer heart-rate–corrected QT intervals and that TRT results in interval shortening [20]. Prolonged heart-rate–corrected QT intervals can result in an increased ventricular arrhythmia incidence and subsequent sudden cardiac death [21].

The majority of preclinical studies have found testosterone to have vasodilatory effects. It is believed that this process involves the downregulation of L-type voltage-gated calcium channels [22] and upregulation of calcium-activated potassium channels [23]. The immediacy of the vasodilation has raised questions as to whether the underlying mechanism involves nongenomic actions of testosterone. Further, testosterone has been shown to increase cardiac contractility [22] and cardiomyocyte relaxation speed [24]. It is not clear whether these vascular effects are dependent on the endothelium and/or AR. It is important to note that these findings oppose those of other studies reporting that testosterone intensifies vasocontraction [25].

Testosterone deficiency is common in men with heart failure, and studies have shown an association between low testosterone levels and poor cardiovascular outcomes. Testosterone exerts an important regulation of cardiovascular function through genomic and nongenomic pathways. It influences contractility, energy metabolism of myocardial cells, apoptosis, and the remodeling process. Reduced testosterone levels in men with heart failure are associated with increased mortality and poor prognosis. Testosterone replacement therapy has demonstrated benefits in enhancing myocardial ischemia, exercise capacity, and serum glucose levels in men suffering from heart failure. Nevertheless, the effects of testosterone on the cardiovascular system remain incompletely understood and may vary between normal physiological conditions and pathological states [26,29].

A study published in 2012 established that reduced testosterone levels in males are correlated with a heightened risk of cardiovascular disease, including heart failure. The research proposed that testosterone could exert a protective influence on the heart and that diminished testosterone levels may play a role in the pathogenesis of heart failure [30]. Another study published in 2021 explored the relationship between testosterone and cardiovascular risk factors, including subclinical atherosclerosis, lipoprotein function, and heart failure. The study discovered that low testosterone levels are correlated with a heightened risk of heart failure, and that testosterone replacement therapy might serve as a viable treatment option for men suffering from heart failure [31].

Research has shown that there is a link between low testosterone levels and heart failure [32]. Testosterone is a hormone that is important for the development and maintenance of male sexual characteristics, but it also plays a role in the cardiovascular system. Low levels of testosterone have been associated with an increased risk of cardiovascular disease, including heart failure. Conversely, there is also evidence indicating that elevated levels of testosterone may have deleterious effects on the cardiovascular system. A study published in The Lancet documented that genetically predicted elevated testosterone levels were correlated with an increased risk of thromboembolism, heart failure, and myocardial infarction [33].

Several mechanisms have been proposed to explain the relationship between testosterone and cardiovascular health. These include 1) Testosterone's effect on thromboxane A2 receptors, 2) Vascular adhesion molecule one receptors, 3) Erythropoiesis, and 4) Obstructive sleep apnea [34]. Epidemiological research indicates that testosterone levels exceeding 500 ng/dL are correlated with a heightened risk of cardiovascular disease. Nonetheless, the association between testosterone and cardiovascular risk is intricate and may be influenced by numerous factors, including age and the existence of additional risk indicators for cardiovascular pathology. Testosterone has been found to have both positive and negative effects on cardiovascular health. On one hand, testosterone has been shown to have anti-inflammatory and antithrombotic effects, which could potentially reduce the risk of cardiovascular disease. On the other hand, higher testosterone concentrations have been associated with increased levels of C-reactive protein (CRP), a marker of inflammation, which could contribute to the development of cardiovascular disease [35].

Central effects of testosterone involve its influence on cardiomyocytes and electrophysiology. Testosterone modulates cardiac contraction and calcium homeostasis, which are essential for maintaining the proper functioning of the heart. Additionally, testosterone has been shown to affect electrophysiology, which is crucial for the proper conduction of electrical impulses in the heart. Peripheral effects of testosterone include its influence on blood vessels, baroreceptor reactivity, skeletal muscles, and erythropoiesis. These effects may contribute to the beneficial effects of testosterone in the pathophysiology of HF syndrome. However, the central, or cardiac, effects of testosterone are still to be further explored [35].

Oral testosterone tablets such as methyltestosterone should generally not be used to treat testosterone deficiency due to significant hepatic adverse effects. The buccal form should not be chewed or swallowed. This dose form is no longer available in the US. Transdermal testosterone formulations, including testosterone gels, patches, pellets, and intramuscular testosterone injections, are the most popular dosage forms of testosterone supplementation in symptomatic hypogonadal men. Testosterone gels are generally the recommended formulation due to patient preference, cost, convenience, minimal hormonal level variability, and insurance coverage. The chief advantage of gels is maintaining stable serum testosterone concentrations, resulting in stable libido, energy, and mood. There are various formulations of testosterone gels. A study showed that the bioavailability of testosterone gel is 30% lower when applied to the abdomen than to the arms and shoulders. Intramuscular injections of testosterone include testosterone enanthate and testosterone cypionate. These injections are generally recommended at initial doses of 100 mg every week. In 2014, the FDA approved an extra-long-acting intramuscular injection form of testosterone called testosterone undecanoate, which is dosed at 750 mg, followed by a second dose 4 weeks later and subsequent doses every 10 weeks [36-38].

5. Conclusion

Testosterone deficiency may worsen HFpEF in men. The role of testosterone in cardiovascular health is multifaceted and depends on various factors. Testosterone demonstrates a nuanced effect on cardiovascular health, presenting potential benefits and risks for men and women. While more research is needed to fully understand the relationship between testosterone and heart failure, it is clear that testosterone levels play a role in cardiovascular health.

6. Data Availability Statement

The datasets generated and analyzed during the current study are not publicly available due to privacy and ethical considerations, but are available from the corresponding author upon reasonable request.

7. Ethical Statement

Sumatera Medical Journal (SUMEJ) is a peer-reviewed electronic international journal. This statement below clarifies the ethical behavior of all parties involved in the act of publishing an article in Sumatera Medical Journal (SUMEJ), including the authors, the chief editor, the Editorial Board, the peer-reviewer, and the publisher (TALENTA Publisher Universitas Sumatera Utara). This statement is based on COPE's Best Practice Guidelines for Journal Editors.

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All authors contributed to the design and implementation of the research, data analysis, and finalizing the manuscript.

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11. Conflict of Interest
The authors declare that there is no conflict of interest regarding the publication of this paper.

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