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Mini Review

Fueling the Failing Heart: A Mini Review of Coenzyme Q10 Utilisation in Heart Failure Treatment

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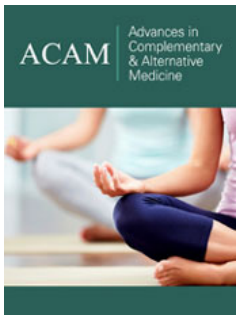

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Abstract

Heart failure is a complex clinical syndrome increasingly recognized as a state of myocardial energy starvation driven by mitochondrial dysfunction and reduced ATP production. While standard pharmacotherapy targets neurohormonal pathways, it often leaves the metabolic deficit of the failing heart unaddressed. This mini review examines the pathophysiological rationale of CoQ10 supplementation - a critical component of the mitochondrial electron transport chain and a potent antioxidant- and evaluates its clinical utility in HF management. Myocardial biopsies indicate that CoQ10 deficiency correlates with increased heart failure severity. Clinical evidence, anchored by the landmark Q- Symbio trial, demonstrates that long-term adjunctive CoQ10 therapy significantly reduces MACE and cardiovascular mortality compared to placebo. Furthermore, CoQ10 supplementation is associated with improved NYHA functional class, and potential recovery in left ventricular ejection fraction. Emerging research also suggests that CoQ10 levels influence the functionality of the anti-aging and cardioprotective protein Sirtuin1. This review notes that while CoQ10 demonstrates an excellent safety profile with no significant adverse events, data regarding its efficacy in HFpEF remain scarce and inconclusive. The supplementation of CoQ10 in addition to traditional pharmacotherapy is a scientifically sound, valuable adjunctive therapy, though further large-scale, rigorous trials are needed to standardize dosage and confirm benefits across all HF phenotypes.

Keywords: Coenzyme Q10; Heart failure; MACE; HFpEF; Ubiquinone; Ubiquinol

Abbreviations: ATP: Adenosine Triphosphate; CoQ10: Coenzyme Q10; CoQH2: Ubiquinol; HF: Heart failure; HFpEF: Heart Failure with Preserved Ejection Fraction; LDL: Low-density lipoprotein; MACE: Major Adverse Cardiovascular Events; NAD: Nicotinamide Adenine Dinucleotide; NYHA: New York Heart Association; Sirt1: Sirtuin 1; YLD: Years Lived with Disability

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Introduction

The heart has one of the highest metabolic rates in the human body, continuously producing and consuming ATP. Energy production and availability are crucial for the heart muscle to maintain its ability to contract in order to sustain systemic and pulmonary blood pressures. Most of the energy for cardiac muscle contraction arises from oxidative metabolism in mitochondria [1]. Due to this fact, Coenzyme Q10 plays a key role in energy production and supply for the heart muscle, as CoQ10 is a member of the mitochondrial respiratory chain [2]. CoQ10 is a fat-soluble molecule found in the inner membrane of the mitochondria. Its main function is the movement of electrons via the electron transport chain, the process from which ATP arises. It is a redox molecule found in the human body in two bioactive states. Those states are ubiquinone, as the oxidised state, and ubiquinole (CoQH2) as the reduced state.

It is the only fat-soluble antioxidant that can be synthesized de novo in the human body. CoQH2 is an essential fat-soluble antioxidant, which prevents peroxidation of LDL in circulation, providing also anti-inflammatory activity. The reduced state is highly unstable and

rapidly oxidises back to the oxidised state [3]. In the pathological condition of heart failure, the metabolic ability of cardiac muscle to effectively produce energy is heavily impacted [1]. Heart failure is characterised by mitochondrial dysfunction leading to a dramatic reduction in ATP production. Lower CoQ10 levels are associated with increasing severity of heart failure symptoms. In the myocardium of patients with NYHA class III or IV has been found significantly less CoQ10 present when compared with the myocardia of patients with NYHA class I or II [4]. According to the aforementioned, it is easily deduced that CoQ10 deficiency plays a serious role in the pathophysiology of heart failure.

Heart failure is a complex clinical syndrome which results in an inadequate cardiac output and reduced ejection capacity, due to serious structural and functional abnormalities [5]. As a clinical entity, heart failure has a high prevalence (8.52 per 1,000 inhabitants). It is one of the most common causes of disability and admission to hospital in older individuals. It is associated with high morbidity and mortality globally. Heart failure has the largest burden for patients over 60 years of age for both prevalence and YLDs, and this has increased by 3.9% and 4.5% respectively, in very elderly people during the last 28 years [6]. Based on the energy deprivation of the myocardium in heart failure, combined with the role of CoQ10 in ensuring energy supply and its low concentrations found in heart failure, it is reasonable to raise the question of the possible involvement of CoQ10 in the treatment of heart failure.

Discussion

Heart failure remains one of the leading causes of morbidity and mortality worldwide [7]. It is a complex clinical syndrome with structural and functional cardiac abnormalities that affect patients' quality of life. The standard pharmacological treatment for HF primarily targets the neurohormonal axis and it is currently the cornerstone of therapy [8]. The nowadays better-known pathophysiological mechanism of mitochondrial dysfunction and its role in the development and progression of HF, can also be included among the targets of treatment. CoQ10, as a co-factor of energy and a powerful antioxidant, can be utilized and contribute to the improvement of mitochondrial function and, consequently, cardiac function.

In the presence of a known pathophysiological mechanism, real clinical data from double blind randomised trials are needed in order to open the discussion for the use of any substance in the treatment of a clinical entity. The clinical value of supplemental CoQ10 administration was confirmed by the Q-SYMBIO trial. This is the most contemporary, multicenter, randomised, double-blind, placebo- controlled trial that evaluated CoQ10 as an adjunctive treatment in chronic heart failure, specifically addressing the issue of mitochondrial dysfunction and energy starvation. Patients with moderate to severe HF were randomly assigned in a 2-year prospective trial to either CoQ10 100mg three times daily or placebo, in addition to standard therapy. Although short-term functional endpoints were not statistically different in the two groups, the results of the long-term endpoints were more encouraging. Supplementation with CoQ10 for two years significantly reduced the primary long-term composite endpoint of major adverse cardiovascular events (MACE) compared to placebo (15% vs 26%; HR 0.50; p=0.003).

Furthermore, the intervention significantly lowered both cardiovascular mortality (9% vs 16%; p=0.026) and all-cause mortality (10% vs 18%; p0.018) [9,10]. In addition, a significant improvement of NYHA classification was found in the CoQ10 group after 2 years [9]. A post-hoc analysis examining the European sub-group of Q-SYMBIO, confirmed the therapeutic efficacy shown in the original study. It revealed a significant recovery in left ventricular ejection fraction (6%, p=0.021) that was not evident in the total population. In the European subgroup higher serum levels of the CoQ10 were observed, which may be the reason for the findings in ejection fraction increase. These data indicate that CoQ10 is a safe therapeutic agent, as no major adverse events were reported in comparison with the placebo group, that offers benefits to the standard guideline-directed pharmacological therapy [11]. More than 50% of patients have a type of HF known as HFpEF, also known as diastolic HF [8,12]. Mitochondrial dysfunction and the resulting energy deficiency in myocardium, are key pathophysiological mechanisms in this case as well. To this date, studies and data on supplemental CoQ10 administration in HFpEF's patients are scarce.

From a randomised, double-blind, small total number of participants and short duration trial, no remarkable changes in diastolic function of the heart in patients who received CoQ10 are found compared to those who received placebo [13]. The inability of the study to show any positive results is maybe due to the short duration of the trial. Further investigation is clearly needed with longer lasting follow up periods. New trials, designed to evaluate the effectiveness of ubiquinone are in progress and we are anticipating the results [12]. Q-SYMBIO was a study that ultimately lasted less than it was originally planned, and its unexpected results were so impressive that concerns arose as to whether they would be interpreted with the same enthusiasm if emphasis were placed on parameters such as the small total number of patients participating and the lack of sufficient statistical power to assess safety [4]. Meta-analyses that focused on the efficacy of CoQ10 in patients with HF note that there is no consensus among studies regarding dosage and duration of treatment with CoQ10 [5,13].

Furthermore, several trials do not provide clear information on allocation, concealment and randomisation, thus leaving an open window for potential bias [5]. These considerations demonstrate the necessity of conducting high-quality, more rigorous, large- scale, international randomised, double-blind studies with sufficient statistical power and long-term follow-up. This will ensure that the results are as valid as possible and can be safely applied in everyday clinical practice. Additionally, it is worth mentioning that CoQ10 may have other therapeutic effects, too. The anti-aging gene Sirtuin 1 is a key player in regulating cell health and longevity, as it coordinates vital functions such as glucose and lipid metabolism, acts as a deacetylase and repairs DNA [14-17]. The efficacy of Sirt1 seems to be proportional to CoQ10 levels, as their decline -which tends to worsen with age- undermines its function in various ways. NAD+ is an essential substrate for Sirt1 activity.

The availability of NAD+ decreases with CoQ10 deficiency, because as already mentioned under this condition the efficiency of the mitochondrial respiratory chain decreases, leading to energy starvation and a drop in the NAD+/NADH ratio. In addition, oxidative stress affects the stability of the Sirt1 protein and limits the expression of its mRNA [18]. Therefore, CoQ10 deficiency leads to reduced Sirt1 functionality, which is the link between Sirt1 and Heart Failure. In HF, the protective actions of this protein, such as the regulation of mitochondrial biogenesis and the prevention of myocardial cells from apoptosis, are gradually lost [14-16,18]. Enhancement of the anti-aging and cardioprotective mechanisms of Sirt1, especially in patients with HF, can be achieved through the administration of Sirt1 activators (resveratrol and zinc) inaddition to supplemental CoQ10 administration, which provides the necessary energy substrate for its activity [14,18].

Conclusion

Heart failure is fundamentally characterized by mitochondrial dysfunction and myocardial energy starvation. As demonstrated in this review, CoQ10 plays a pivotal role in cellular bioenergetics and antioxidant defense, making it a physiologically sound target for therapeutic intervention. Current clinical evidence, predominantly from the landmark Q-Symbio trial, supports the efficacy of CoQ10 as an adjunctive treatment. Long-term supplementation has been shown to significantly reduce MACE and cardiovascular mortality, while also improving patients' functional status (NYHA class). Furthermore, CoQ10 exhibits a good safety profile with no significant adverse effects reported, distinguishing it as a safe addition to standard heart failure pharmacotherapy. Despite these promising findings, questions remain regarding its efficacy in HFpEF, where data are currently scarce and inconclusive. Future research should focus on large-scale, rigorous, randomized control trials to further elucidate optimal dosage duration and specific patient subgroups that would benefit most. Nevertheless, the existing data suggest that fueling the failing heart with CoQ10 represents a valuable metabolic approach to improving outcomes in heart failure management.

Conflict of Interest

No conflict of interest to be declared.

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