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ORIGINAL RESEARCH

# Antioxidant Effects of CoQ10 in Transfusion-Dependent β-Thalassemia Major Patients: Implications for Ferroptosis-Related Pathways

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**Background:** The role of oxidative stress and ferroptosis in the pathogenesis of thalassemia major have been established and have been shown to cause tissue damage and disease progression. The lipophilic antioxidant coenzyme Q10 (CoQ10) can protect against tissue damage by restoring antioxidant enzyme function and decreasing oxidative damage. This study evaluated the effect of CoQ10 supplementation on biomarkers of ferroptosis in patients with thalassemia major.

**Methods:** In this single-arm pre–post study, patients with confirmed thalassemia major (48) received oral CoQ10 (100 mg/day) for 8 weeks. Peripheral blood samples were collected before and after the study period for assays to measure antioxidant enzyme activity: superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). Statistical analyses were conducted using paired *T*-test and Wilcoxon.

**Results:** CoQ10 supplementation significantly increased SOD and GPx activity compared to baseline, ($p < 0.05$). No significant change was seen in CAT activity. No statistically significant differences were observed in hematological parameters and ferritin level after using CoQ10.

**Conclusion:** CoQ10 supplementation appears to exert protective effects against ferroptosis in patients with thalassemia major, primarily by enhancing antioxidant defenses. Our study findings support the hypothesis that CoQ10 may represent a potential adjunctive therapy in mitigating oxidative stress and ferroptotic damage.

**Keywords:** coenzyme Q10, thalassemia major, ferroptosis, oxidative stress, antioxidant enzymes

## Introduction

Thalassemia is a group of blood disorders that run in families. Kids inherit mutations in their globin genes, messing up how much hemoglobin they make.[1,2] Although some individuals just carry the trait without showing symptoms, others get hit hard and require blood transfusions throughout their whole lives.[1] The treatment has both benefits and drawbacks: the patients require regular blood transfusions and iron-binding drugs to survive, but the same treatments load their bodies with toxic levels of iron, damaging their organs.[3] Currently, the only possible real treatment is a bone marrow transplant. However, there might be some gene therapy stuff in the pipeline that might change things in the future.[4] Despite advancements in survival for thalassemia patients, heart disease due to iron overload remains the main cause of death in this population.[2]

Thalassemia patients face significant challenges related to iron overload. The excessive iron in thalassemia patients originates from three main routes: 1: their digestive system's over absorption of dietary iron, 2: iron administered during regular blood transfusions, and 3: ongoing chronic hemolysis. The accumulation of excess iron leads to oxidative stress,

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resulting in the generation of harmful reactive oxygen species (ROS) that can overwhelm the body's antioxidant defenses.[5–7] The results are quite severe: the excess iron leads to the creation of harmful oxygen molecules (ROS), overpowering the body's remaining protective antioxidants and resulting in widespread cell damage, while also damaging the heart, causing liver dysfunction, and potentially impairing the function of cellular mitochondria, which are crucial for energy production.[6,8]

Recent research has identified a distinct form of regulated cell death, known as ferroptosis, which is iron-dependent and characterized by lipid peroxidation. Unlike apoptosis or necrosis, ferroptosis involves accumulation of iron and reactive oxygen species leading to oxidative damage, and has been recognized as a major contributor to tissue injury observed in β-thalassemia patients.[8,9] This is an interesting observation: thalassemia patients having moderate forms of the condition who do not receive regular blood transfusions, weirdly show higher levels of damaging molecules (ROS) and damaged fats in comparison with those who do get transfusions. This may indicate that when tissues are without oxygen, the mitochondria within those cells are triggered to produce even more of these harmful compounds.[6] Fortunately, several iron-chelating medications are available, particularly deferiprone, which reduces intracellular iron levels. These drugs help to lower the production of harmful ROS and can improve cardiac function in β-thalassemia patients.[5]

The human body naturally defends against "rusting" damage using enzymes like SOD, GPx, and CAT, which function to neutralize harmful reactive species: SOD converts harmful substances into hydrogen peroxide, which GPx and CAT then transform into harmless water. However, in thalassemia patients, this system is overworked due to ongoing oxidative stress, leading to exhaustion of these protective enzymes, much like a car breaking down from constant use without maintenance. Recent research shows that these patients have significantly lower levels of SOD and GPx, and the lower these levels, the more severe their health problems tend to be.[10–12] This enzyme breakdown is a significant red flag, demonstrating required treatments that either boost these natural protectors or find another way to control the oxidative damage that's wreaking havoc in the patients.

CoQ10, or Coenzyme Q10, is a fat-soluble antioxidant produced by the human body on its own. It helps the mitochondria, similar to the powerhouses of human cells, to produce energy, and at the same time, it fights off the mentioned damaging oxygen molecules. However, CoQ10 does not just directly get rid of those harmful molecules; it also helps to recharge other antioxidants, like vitamin E, so they can keep working even after they have been used.[13] Scholars tested CoQ10 in labs and actual patients with various chronic diseases. The results demonstrated significant potential. It was like reducing oxidative stress in folks with heart problems and brain disorders.[14] What CoQ10 might do for thalassemia patients has been investigated. Some research hints that giving these patients CoQ10 supplements improves antioxidant defenses and lowers markers showing oxidative damage. However, nobody's looked at whether CoQ10 affects that iron-driven cell death pathway - ferroptosis.[15]

The current study aims to fill the above-described knowledge gap. We conducted a real-world clinical study in adult patients with β-thalassemia to investigate the effects of oral CoQ10 supplementation over 8 weeks. We primarily measured changes in endogenous antioxidant enzyme activities (SOD, GPx, CAT) and explored the potential implications of these effects on ferroptosis-related pathways. If CoQ10 demonstrates effectiveness in enhancing the antioxidant defense system, it could support the rationale for a new adjunct treatment aimed at reducing oxidative damage in thalassemia. These preliminary findings may also contribute to understanding iron overload-related cell damage and inform future research into more targeted therapies.

# Materials and Methods

## Study Design

This study was a before-and-after clinical trial approved by the Ethical Committee of Shahrekord University of Medical Sciences (IR.SKUMS.REC.1401.116). The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment. This study was registered in the Iranian Registry of Clinical Trials (IRCT20221211056777N1). Given the limited number of transfusion-dependent β-thalassemia major patients in our region, a census sampling approach was used. All eligible patients who were referred to Hajar Hospital during the study period were approached for inclusion. A total of 60 patients met the eligibility criteria and

were initially enrolled. During the follow-up, 10 participants discontinued or were lost to follow-up, resulting in a final cohort of 48 patients included in the analysis. This approach ensured maximal representativeness of the regional patient population.

## Patient Recruitment and Study Procedure

Patients diagnosed with β-thalassemia major (18–50 years) who had been referred to Hajar Hospital were selected.

Inclusion criteria: Individuals diagnosed with β-thalassemia major, confirmed through β-globin gene sequencing, requirement of blood transfusion at least once per month (approximately 15 mL of packed red blood cells per kilogram body weight at each transfusion), and being under chelation therapy (with deferoxamine, and Deferasirox).

Exclusion criteria were Patients with infectious diseases, pregnancy and lactation, history of smoking or alcohol consumption.

After the definitive diagnosis, and before the blood transfusion, the patients were examined for the parameters of interest and then received Q10 tablets (100 mg/day) for 8 weeks. At the end of the intervention period, the same parameters were re-assessed for comparison.

## Blood Fractions Preparation

Fasting venous blood samples were collected in gel and clot activator tubes, centrifuged at 3000 × g for 8 minutes, and the obtained serum sample was carefully separated and aliquoted, and then were stored at −80°C for the further biochemical analyses.

Hemoglobin concentration was measured using an automated hematology analyzer (Sysmex Kx21N, Kobe, Japan). Serum ferritin was determined via an enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's protocol (Pishgaman sanjesh, Tehran, Iran).

## Analysis of Antioxidant Biomarkers

### Catalase (CAT) Activity Assay

Catalase (CAT), an essential antioxidant enzyme that converts hydrogen peroxide ($H_2O_2$) into water and oxygen to prevent oxidative cellular damage, was measured using a commercial Catalase Activity Kit (Kiazist, Hamadan, Iran). Serum catalase activity was determined based on the colorimetric detection of formaldehyde produced by the reaction of methanol and $H_2O_2$ in the presence of CAT. The color change of the Purpald reagent was monitored according to the manufacturer's instructions. Activity of CAT was calculated using a formaldehyde standard curve and expressed as mU/mL using the following formula:

$$\text{CAT activity}\left(\frac{\text{mU}}{\text{mL}}\right) = \frac{\text{sample valume}}{20} \times 12 \times \text{sample dilution}$$

### Superoxide Dismutase (SOD) Activity Assay

Superoxide dismutase (SOD) is a key antioxidant enzyme that catalyzes the dismutation of superoxide radicals ($O_2\bullet^-$) into hydrogen peroxide and molecular oxygen, thereby protecting cells from oxidative stress–induced injury. Serum SOD activity was assessed using a commercial SOD Activity Kit (Kiazist, Hamadan, Iran) according to the manufacturer's instructions.

In this colorimetric assay, SOD inhibits the reduction of a tetrazolium salt by superoxide radicals, and the degree of inhibition is proportional to the enzyme activity in the sample. Activity was calculated using a standard curve and expressed as U/mL. The inhibition rate was determined using the following formula:

$$\text{inhibition rate} = (\text{B2 absorbance} - \text{B1 absorbance}) - (\text{sample absorbance} - \text{control absorbance})/(\text{B2 absorbance} - \text{B1 absorbance}) \times 100$$

### Glutathione Peroxidase (GPx) Activity Assay

Glutathione peroxidase (GPx) is an essential antioxidant enzyme that reduces hydrogen peroxide and lipid peroxides using reduced glutathione (GSH), thereby preventing oxidative damage to cellular components. Serum GPx activity was measured using a commercial GPx Activity Kit (Kiazist, Hamadan, Iran), following the manufacturer's protocol.

In this coupled assay, GPx catalyzes the reduction of hydrogen peroxide in the presence of GSH, generating oxidized glutathione (GSSG). Glutathione reductase then converts GSSG back to GSH using NADPH as an electron donor. The resulting decrease in NADPH absorbance is monitored colorimetrically and is proportional to GPx activity. Activity values were calculated according to the kit's instructions and expressed as U/mL.

All assays were performed according to the manufacturer's standardized instructions, and all samples were incubated under identical recommended conditions.

## Statistical Analysis

Descriptive statistics were reported as frequency (%), mean± SD or median (IQR) for the qualitative, quantitative variables with normal distribution and quantitative variables without normal distribution respectively. The paired *T*-test was used to compare before, after values of Catalase, Superoxide dismutase and Glutathione peroxidase. Moreover, the Wilcoxon signed-rank test was used to compare before, after values of Hemoglobin, Ferritin, WBC and Platelet in SPSS software version 23.0. $P<0.05$ was considered as statistically significant.

# Results

## Baseline Characteristics

Table 1 shows the basic information about the people who took part in the study. The study included 48 individuals with β-thalassemia major, 15 of whom were female (31.3%) and 33 of whom were male (68.8%). The average age was 29.66 ± 7.86 years. The table also includes baseline laboratory measures such as hemoglobin (Hb), ferritin, white blood cell count (WBC), and platelet count. Prior to the intervention, there were no significant differences in these measures.

## Antioxidant Enzyme Activity

The following table (Table 2) shows the impact of taking 100 mg of CoQ10 every day for 8 weeks on the activity of antioxidant enzymes.

The average activity of SOD rose from 1.79 ± 0.94 U/mL at the start to 2.50 ± 0.93 U/mL after the intervention ($P < 0.001$). The GPx activity increased from 3.45 ± 1.03 U/mL to 3.91 ± 1.00 U/mL ($P = 0.001$). Catalase (CAT) activity increased slightly from 3.43 ± 0.81 mU/mL to 3.53 ± 0.80 mU/mL, but was not statistically significant ($P = 0.48$). These findings show that CoQ10 supplementation significantly increased antioxidant enzyme activity, especially SOD and GPx.

**Table 1** Baseline Characteristics of the Study Participants (N = 48)

| Characteristic | Value |
|---|---|
| Total patients | 48 |
| Gender, n (%) | Female: 15 (31.3%) Male: 33 (68.8%) |
| Age, years (Mean ± SD) | 29.66 ± 7.86 |
| Hemoglobin, g/dL (Median, IQR) | 9.60 (9.10–10.20) |
| Ferritin, ng/mL (Median, IQR) | 1723 (743–3920) |
| White Blood Cell count, $\times 10^3/\mu L$ (Median, IQR) | 11400 (8800–24400) |
| Platelet count, $\times 10^3/\mu L$ (Median, IQR) | 499000 (305000–648000) |
| Splenectomy status, n (%) | 19 (40%) |
| Chelation therapy, n (%) | Deferasirox only: 29 (60%), Deferasirox + Deferoxamine: 14 (30%), Deferoxamine only: 5 (10%) |
| T2* MRI performed, n (%) | 38 (80%) |
| Cardiac iron overload on T2* MRI, n (%) | 4 (10% of those scanned) |

**Table 2** Antioxidant Enzyme Activity Before and After CoQ10 Supplementation

| Biomarker | Before Intervention (Mean ± SD) | After Intervention (Mean ± SD) | P-value |
|---|---|---|---|
| Catalase (CAT) | 3.43 ± 0.81 | 3.53 ± 0.80 | 0.48 |
| Superoxide dismutase (SOD) | 1.79 ± 0.94 | 2.50 ± 0.93 | <0.001 |
| Glutathione peroxidase (GPx) | 3.45 ± 1.03 | 3.91 ± 1.00 | 0.001 |

**Table 3** Hematological Parameters and Ferritin Before and After CoQ10 Supplementation

| Parameter | Before Intervention (Median, IQR) | After Intervention (Median, IQR) | P-value |
|---|---|---|---|
| Hemoglobin (g/dL) | 9.60 (9.10–10.20) | 10.00 (9.60–10.20) | 0.407 |
| Ferritin (ng/mL) | 1723 (743–3920) | 1684 (697–3330) | 0.772 |
| WBC ($\times 10^3/\mu L$) | 11400 (8800–24400) | 10700 (8000–23900) | 0.214 |
| Platelet ($\times 10^3/\mu L$) | 499000 (305000–648000) | 516000 (291000–646000) | 0.089 |

## Hematological Parameters and Ferritin

Table 3 and Figure 1 shows how hematological parameters and ferritin changed before and after 8 weeks of using CoQ10. There were no statistically significant differences were observed, indicating that the hematological status remained steady during the intervention period.

In summary, 8-week CoQ10 supplementation markedly enhanced antioxidant enzyme activity, especially SOD and GPx, whereas hematological parameters and ferritin levels remained unchanged. These results indicate that CoQ10 may improve antioxidant defenses in patients with β-thalassemia major without negatively impacting hematological status.

# Discussion

Superoxide dismutase (SOD) and glutathione peroxidase (GPx), two important antioxidant enzymes, showed a significant increase in activity after 8 weeks of supplementing with 100 mg/day CoQ10 in β-thalassemia major patients in this current before-and-after clinical trial. Catalase (CAT) activity, on the other hand, showed a slight, non-significant increase. This lack of significant change in CAT may be due to the relatively short intervention period (8 weeks) and its lower sensitivity to moderate oxidative stress compared to SOD and GPx. These findings suggest that CoQ10 can effectively strengthen endogenous antioxidant defenses, potentially mitigating oxidative stress induced by chronic iron overload.

In β-thalassemia, oxidative stress is a known cause of cellular damage, mostly brought on by excessive iron accumulation and repeated blood transfusions, which produce reactive oxygen species (ROS).[1,2] Increased ROS can damage mitochondria, proteins, and lipids, exacerbating tissue damage and organ failure. CoQ10 supplementation may reduce oxidative damage by increasing SOD and GPx activity, which in turn may increase hydrogen peroxide reduction and superoxide radical scavenging. A longer supplementation period or higher dosages may be required to see a discernible impact on hydrogen peroxide detoxification, as evidenced by the minimal improvement in CAT activity.[3]

Crucially, following the intervention, hematological indicators such ferritin, hemoglobin, white blood cell count (WBC), and platelet count remained steady. CoQ10 supplementation is safe for β-thalassemia patients, as it does not interfere with normal transfusions or chelation therapy. Maintaining consistent ferritin levels is especially critical in this population since high iron levels are a major source of ROS production and oxidative damage.[2,4] Likewise, unchanged platelet and WBC counts imply that CoQ10 has no negative effects on bone marrow function.

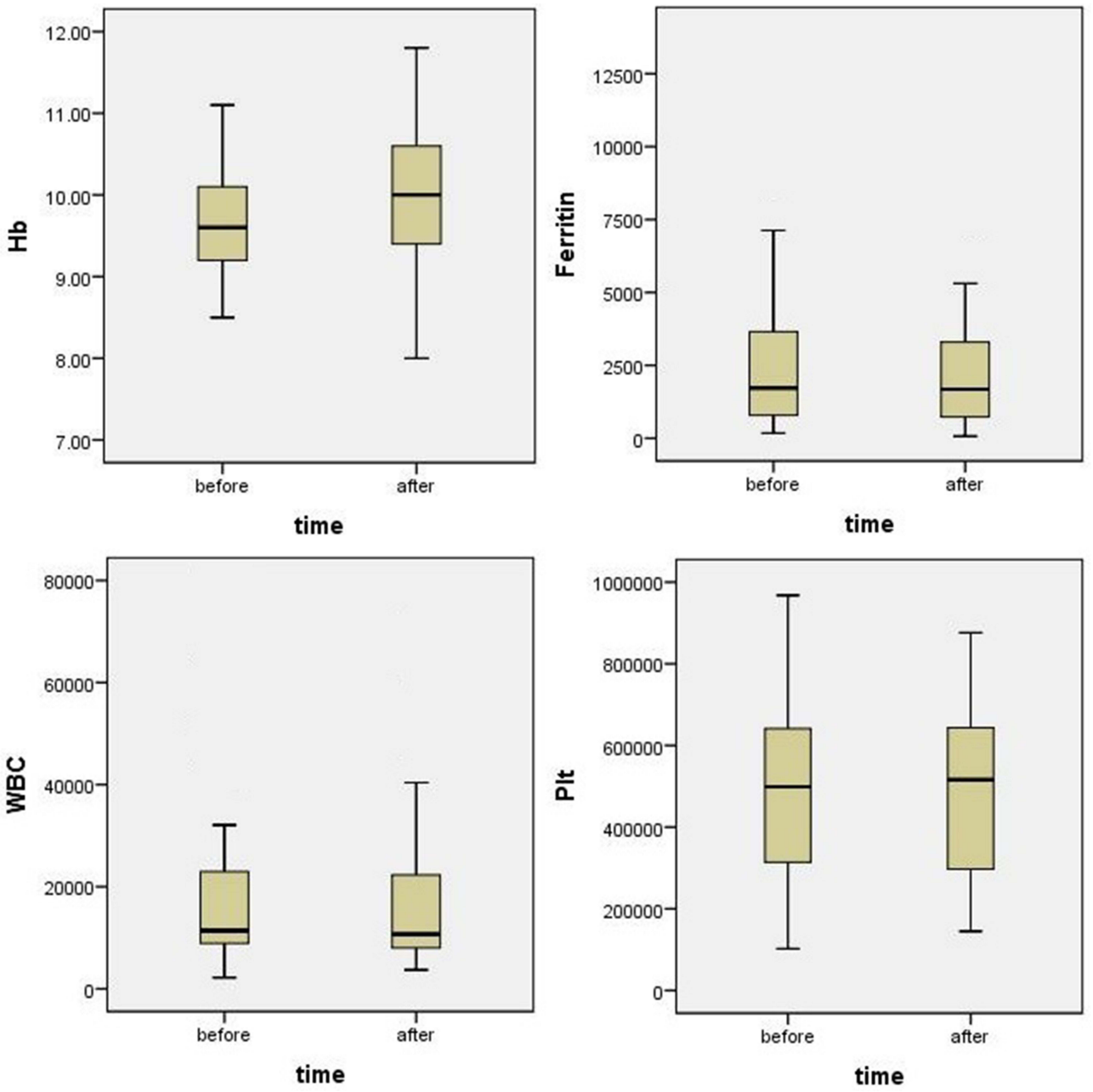


**Figure 1** The box plot diagram is related to the 4 variables (Hb, Ferritin, WBC, Plt) before and after the intervention.

Our findings are consistent with earlier research revealing CoQ10's antioxidant capability in many chronic diseases. Quantitatively, the increase in SOD activity in our β-thalassemia cohort (from 1.79 ± 0.94 to 2.50 ± 0.93 U/mL) is consistent with previous reports in patients with chronic conditions. For example, in a study by BJ Lee, 2012, CoQ10 supplementation was associated with an SOD increase of ~12–14 U/mg protein, and CAT activity increased by ~44–99 U/mg protein depending on adjustment models.[16] These findings suggest that the antioxidant effect of CoQ10 is conserved across different pathological conditions and supports the biological plausibility of its protective role in β-thalassemia. Supplementation, for example, has been demonstrated to improve SOD and GPx activity in patients with cardiovascular disease and metabolic syndrome.[5,6] Despite the scarcity of direct studies in thalassemia, comparable improvements in oxidative markers have been observed, indicating a mechanism that is conserved across oxidative stress-related disorders.[7,8] The observed increases in antioxidant enzyme activity may have therapeutic implications that

go beyond laboratory findings. CoQ10, by improving the body's enzymatic defenses, may help minimize ROS-induced cellular damage, potentially reducing the progression of iron-induced organ dysfunction, particularly in the heart and liver. While our study did not directly assess clinical outcomes such as heart function or liver enzymes, future research could look into these topics.

The present study has several limitations. First, the 8-week intervention period was relatively short, which restricts the ability to evaluate long-term or clinically meaningful improvements in major organs such as the heart, liver, and endocrine system. While our findings demonstrate the safety of CoQ10 and its beneficial effects on antioxidant enzyme activity, longer treatment duration and extended follow-up would be necessary to assess sustained clinical outcomes. Additionally, the sample size was limited, and the long-term effects and optimal therapeutic dose of CoQ10 in thalassemia patients remain to be established. Furthermore, while we investigated major antioxidant enzymes, we did not examine the influence on downstream oxidative damage markers such as lipid peroxidation products or mitochondrial function. Similarly, GPX4, a key regulator of ferroptosis, was not measured due to resource limitations. Therefore, our conclusions regarding potential ferroptosis-related effects remain preliminary and hypothesis-generating. Longer-term trials with bigger cohorts are planned to examine biochemical and clinical outcomes, such as cardiac and hepatic function. Combining CoQ10 with other antioxidant techniques could also be investigated to enhance protective effects in thalassemia patients. Our study provides preliminary evidence that CoQ10 supplementation may influence ferroptosis-related pathways in transfusion-dependent β-thalassemia major patients. While the current results are limited to biochemical outcomes, they offer a foundation for future investigations into molecular mechanisms and potential clinical implications.

## Conclusion

Supplementing individuals with β-thalassemia major with 100 mg/day CoQ10 for eight weeks improved antioxidant enzyme activity, especially SOD and GPx, without affecting hematological parameters or ferritin levels. These findings imply that CoQ10 is a safe and effective adjunct therapy for improving antioxidant defenses, and it may help to reduce oxidative stress-related tissue damage in this patient population. Further research is needed to assess long-term therapeutic benefits and effective dosing regimens.

## Data Sharing Statement

The authors confirm that the data supporting the findings of this study are available upon reasonable request. Individual de-identified participant data (including demographic information, laboratory values, and outcome measures) will be shared. The study protocol, informed consent form, and statistical analysis files will also be available. Data will be accessible by contacting the corresponding author at Shimarahmati1987@gmail.com. The data will be available immediately upon publication and will remain accessible for 5 years.

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## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

The authors declare that they have no conflicts of interest in this work.

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