e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(8); 1329-1335

Original Research Article

Efficacy and Safety of Coenzyme Q10 (100 mg/day) as an Adjunctive Therapy in Migraine Prophylaxis: A Randomized Controlled Open-Label Study

P. Vijayabhanu[1], Prabaharan Umachandran[2], Mahalakshmi J.[3]

[1]Assistant Professor, Department of Pharmacology, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu, India

[2]Assistant Professor, Department of Neurology, Government Villupuram Medical College and Hospital, Villupuram, Tamil Nadu, India

[3]Assistant Professor, Department of Pharmacology, Government Medical College, Kallakurichi, Tamil Nadu, India

Received: 01-05-2025 / Revised: 15-06-2025 / Accepted: 21-07-2025

Corresponding author: Dr. P Vijayabhanu

Conflict of interest: Nil

Abstract

Background: Migraine is a highly prevalent and disabling neurological disorder, often underdiagnosed and inadequately treated, prompting the need for effective and well-tolerated prophylactic interventions.

Objective: To evaluate the efficacy of Coenzyme Q10 (100 mg/day) as an adjunctive therapy to standard prophylactic medication in patients with migraine.

Methods: This was a prospective, open-label, randomized controlled study conducted on 68 migraine patients over a three-month treatment period. Participants were assigned to receive either standard prophylactic therapy alone or in combination with Coenzyme Q10 at a dose of 100 mg/day.

Results: At baseline, both the Coenzyme Q10 and control groups (n = 32 each) were comparable in age, gender distribution, migraine duration, frequency of attacks, pain severity, and associated symptoms, with no statistically significant differences. The 50% responder rate for migraine frequency in the Coenzyme Q10 group increased from 6.25% in the first month to 50% by the third month, significantly higher than the control group (21.87%, $p = 0.001$). Mean monthly attack frequency and pain scores also declined more in the Coenzyme Q10 group ($p < 0.001$), with a 40.62% responder rate in pain reduction by the third month (vs. 15.62% in control; $p = 0.025$). Monthly migraine days decreased significantly (from 9.87 to 5.21; $p < 0.001$), and key symptoms like photophobia and phonophobia were markedly reduced in the Coenzyme Q10 group by the third month ($p < 0.001$). During follow-up, symptoms partially recurred in the Coenzyme Q10 group, with no significant differences between groups after discontinuation.

Conclusion: Coenzyme Q10 at a dose of 100 mg/day demonstrated significant efficacy and good tolerability as an adjunctive therapy in the prophylaxis of migraine.

Keywords: Migraine prophylaxis, Coenzyme Q10, Headache, Antioxidant therapy, mitochondrial dysfunction, randomized controlled trial.

Introduction

Headache is a globally prevalent neurological symptom, affecting nearly half of adults and almost all individuals at least once in their lifetime, regardless of age, race, or gender.[1] While often linked to emotional stress, headaches can also indicate underlying conditions such as migraine, hypertension, anxiety, or depression. The World Health Organization ranks headache among the top ten causes of global disability, especially among women.[2] Migraine, a significant subtype of headache, impairs daily functioning in 75% of sufferers, with 50% requiring assistance during attacks.[2] The global prevalence is estimated at 11.6%, with higher rates in women.[3] Characterized by episodic, often unilateral headaches, migraine may include visual or sensory disturbances known as aura.[4] Interestingly, headache is not always the primary symptom, with many patients reporting associated symptoms such as dizziness and pressure.[5] Despite its burden, only 48% of migraine sufferers are diagnosed, and just 29% report satisfaction with treatment, likely due to poor awareness and inadequate management.[6] Migraine is episodic and linked to

triggers like stress, sleep deprivation, dietary factors, alcohol, sensory stimuli, and hormonal fluctuations. Its familial nature is supported by genetic studies. The central nervous system and cranial vasculature are believed to play key roles in its pathophysiology.[7] Migraine involves disruptions in subcortical aminergic pathways, brainstem, thalamic, and hypothalamic structures.[4] Two main theories—vascular and neurogenic—explain its origin. The vascular theory implicates initial vasoconstriction and cerebral ischemia, while the neurogenic theory attributes migraine to cortical spreading depression followed by neurogenic inflammation and release of mediators such as serotonin and substance P.[8]

Migraine is diagnosed clinically, though its variable presentation leads to frequent underdiagnosis. The International Headache Society (IHS) provides criteria for accurate classification. The clinical course includes prodrome (neurological symptoms), aura (sensory or visual disturbances), headache (4–72 hours of throbbing pain), and postdrome (fatigue, light sensitivity). Not all patients experience all phases.[9] Treatment includes acute and preventive strategies. Acute therapy—analgesics, triptans, ergot derivatives, and antiemetics—aims to abort attacks. Preventive therapy, indicated for those with ≥4 attacks per month, includes daily use of antihypertensives, antidepressants, or anticonvulsants.[10] However, medication overuse can lead to Medication Overuse Headache (MOH), complicating treatment and leading to non-compliance. An ideal prophylactic should offer sustained efficacy with minimal side effects.[11]

This has led to increased interest in nutraceuticals such as magnesium, riboflavin, alpha-lipoic acid, and herbal agents like feverfew and butterbur. Coenzyme Q10 (CoQ10) has gained prominence for its potential role in cardiovascular, metabolic, neurodegenerative diseases, and migraine.[12] CoQ10 is a lipid-soluble, vitamin-like compound found in all cells, particularly in mitochondria, where it supports ATP synthesis.[13] Its antioxidant and anti-inflammatory properties, along with its role in mitochondrial energy production, make it a promising agent in migraine prophylaxis.[14] CoQ10 may reduce pain mediators like CGRP and TNF-α, improving clinical symptoms without significantly altering IL-6 and IL-10 levels.(15) While most studies have used CoQ10 at doses exceeding 150 mg/day, limited data exists on its efficacy at lower doses. Against this background, the objective of the present study was to evaluate the efficacy of Coenzyme Q10 (100 mg/day) as an adjunctive therapy to standard prophylactic medication in patients with migraine over a one-year period by analyzing changes in the frequency of migraine attacks and headache intensity, as measured using the Numerical Rating Scale (NRS).

Materials and Methods

Study design and sample size: This prospective, randomized, open-labelled, two-arm, single-centre interventional study was conducted at the Department of Neurology, Government Kilpauk Medical College, Chennai, over a period of twelve months, from May 2018 to April 2019. Approval for the study was obtained from the Institutional Ethics Committee prior to initiation, and all study procedures were conducted in accordance with the Indian Council of Medical Research (ICMR) guidelines for biomedical research involving human participants. The sample size was calculated using OpenEpi software, with the power of the study set at 80% and a two-sided confidence level of 95% ($\alpha = 0.05$). The sample size was based on an assumed responder rate of 48% in the Coenzyme Q10 group and 14% in the control group, resulting in a calculated total of 62 participants (31 in each group). To accommodate potential dropouts, the sample size was increased to 68, with 34 participants in each group.

Study population: Patients were screened at the Neurology Outpatient Department. Those aged above 18 years, of either sex, with a known diagnosis of migraine or who were receiving standard prophylactic treatment consisting of T. Amitriptyline and T. Propranolol were considered for inclusion. Eligibility required a migraine attack frequency of more than three episodes per month and a pain intensity greater than 3 on the NRS. The diagnosis was confirmed based on clinical history, physical examination, and NRS scoring, and basic laboratory investigations were conducted prior to recruitment. Patients were excluded if they had a history of non-migraine headaches, complicated migraine types (e.g., ophthalmic, hemiplegic, or basilar migraine), psychiatric disorders such as schizophrenia, mania, or bipolar disorder, severe comorbid conditions including heart, thyroid, renal, or liver disease, uncontrolled diabetes or hypertension, or asthma. Patients with a history of substance abuse, those who had received Coenzyme Q10 for any reason within six months prior to the study, and pregnant or lactating women were also excluded.

Study procedure: Patients meeting the eligibility criteria were provided with a detailed explanation of the study. Written informed consent was obtained from willing participants, and a thorough clinical history and general and systemic examination were conducted. Randomization was performed using a computer-generated randomization table. Participants were then allocated to one of two groups: the control group, which received only standard prophylactic therapy

(T. Amitriptyline 25 mg once daily and T. Propranolol in a dose range of 40 to 80 mg/day), and the intervention group, which received Coenzyme Q10 100 mg orally once daily in addition to standard treatment. Coenzyme Q10 was supplied in capsule form, packaged in containers of 30, and administered over a three-month period. Patients were asked to return the containers, regardless of whether the medication was consumed, during their next scheduled visit to ensure compliance.

The participants were issued a migraine calendar to document daily symptoms, including the date of onset and nature of migraine episodes. The study comprised three sequential phases: a pre-intervention period (first month), an intervention period (second to fourth months), and a follow-up period (fifth and sixth months). During the pre-intervention period, patients were instructed to record their symptoms daily in the calendar. At the end of the first month, these data were collected and used to establish baseline values, including migraine frequency, number of headache days, associated symptoms, and pain intensity assessed using the NRS.

In the intervention period, patients attended monthly visits where they returned their symptom calendars, and fresh ones were issued. Pain intensity was again rated using the NRS, and data were collected for ongoing analysis. The Coenzyme Q10 group received the study drug in addition to standard prophylactic medications for three months. Following the intervention, all participants entered a two-month follow-up period, during which they were monitored for symptom recurrence and treatment sustainability. The study drug was discontinued after the third month, and only standard treatment was continued. All patients underwent detailed clinical assessment at each visit. Findings were recorded in a structured Case Report Form (CRF), and vital signs, systemic findings, and adverse events, if any, were documented. The structured visit schedule included a baseline visit (Day 0), followed by monthly assessments at 30, 60, 90, and 120 days. At each visit, patients in both groups were evaluated using their calendars and NRS scores. Coenzyme Q10 was discontinued after 90 days, and patients were continued on standard drugs through the follow-up period. The outcomes of the study were assessed by comparing data from baseline to the third month of treatment (Day 120) between the two groups. The primary outcome was the 50% responder rate, determined by the proportion of patients who achieved a ≥50% reduction in the frequency of migraine attacks, along with changes in the mean monthly attack frequency. Secondary outcomes included the number of patients with a ≥50% reduction in headache intensity, measured using the 0–10 NRS, as well as changes in average pain scores, associated symptoms, and the number of migraine days per month.

Statistical analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 27. Descriptive statistics such as mean and standard deviation were used for continuous variables, while frequencies and percentages were reported for categorical data. Intergroup comparisons were performed using the independent sample t-test for continuous variables and the Chi-square test for categorical variables. Repeated Measures ANOVA was applied to assess within-group changes over time. A p-value of less than 0.05 was considered statistically significant.

Results

At baseline, both the Coenzyme Q10 and control groups consisted of 32 patients each, all diagnosed with migraine without aura. The mean age was comparable between the groups, with 39.75 ± 8.73 years in the Coenzyme Q10 group and 39.53 ± 8.81 years in the control group ($p = 0.460$). The sex distribution was predominantly female in both groups, with 28 females and 4 males in the Coenzyme Q10 group, and 30 females and 2 males in the control group. The average duration of migraine attacks was similar between groups (4.25 hours vs. 4.34 hours; $p = 0.410$). The mean number of migraine days per month and attack frequency were also comparable (11.25 ± 2.31 vs. 11.37 ± 2.35 days; $p = 0.410$ and 5.62 ± 1.16 vs. 5.68 ± 1.17 attacks; $p = 0.420$, respectively). NRS pain scores were nearly identical (6 ± 1.19 vs. 6.06 ± 1.21; $p = 0.420$). The proportion of patients experiencing associated symptoms such as nausea (86% vs. 78.12%; $p = 0.520$), vomiting (18.75% vs. 15.62%; $p = 0.740$), photophobia (71.87% vs. 75%; $p = 0.770$), and phonophobia (58% vs. 62.5%; $p = 0.790$) showed no statistically significant differences between the groups, indicating comparable baseline clinical profiles.

The 50% responder rate for frequency of migraine attacks progressively increased in the Coenzyme Q10 group (6.25% at 1st month, 21.88% at 2nd month, and 50% at 3rd month), with a statistically significant difference observed at the 3rd month compared to the control group (21.87%, $p = 0.001$). Similarly, the mean frequency of migraine attacks per month decreased more significantly in the Coenzyme Q10 group, with values dropping from 5.125 ± 1.51 in the 1st month to 2.721 ± 1.17 by the 3rd month ($p < 0.001$), confirmed by repeated measures ANOVA ($p < 0.001$). The 50% responder rate in pain score also showed a significant difference at the 3rd month (40.62% in the Coenzyme Q10 group vs. 15.62% in the control group; $p = 0.025$). Pain scores declined significantly over time in the Coenzyme Q10 group

(from 5.34 ± 1.57 to 3.28 ± 1.11; $p < 0.001$), and a similar trend was observed for monthly migraine days (from 9.87 ± 3.32 to 5.21 ± 2.15; $p < 0.001$). In contrast, the control group showed less pronounced improvement.

During the first month, the prevalence of symptoms such as nausea, vomiting, photophobia, and phonophobia was comparable between the two groups, with no statistically significant differences ($p > 0.05$). By the second month, photophobia was significantly less frequent in the Coenzyme Q10 group (40.62%) compared to the control group (68.75%), with a p-value of 0.002. Although other symptoms in the second month did not reach statistical significance, nausea and phonophobia showed trends toward reduction in the Coenzyme Q10 group. In the third month, the differences became more pronounced: the Coenzyme Q10 group had significantly fewer patients with nausea (34.37% vs. 62.50%, $p = 0.002$), photophobia (25.00% vs. 65.60%, $p < 0.001$), and phonophobia (21.88% vs. 59.37%, $p < 0.001$). Although the reduction in vomiting was not statistically significant, the overall data suggest that Coenzyme Q10 supplementation was associated with a significant reduction in key migraine-associated symptoms, particularly photophobia and phonophobia, by the third month of treatment.

At the end of the treatment period (3rd month), the frequency of migraine attacks was significantly lower in the Coenzyme Q10 group (2.71 ± 1.17) compared to the control group (3.56 ± 1.01), with a p-value of 0.001, indicating a statistically significant reduction. However, by the 2nd month of the follow-up period—after discontinuation of Coenzyme Q10—the frequency of attacks increased slightly in the Coenzyme Q10 group (3.37 ± 1.12), approaching the level seen in the control group (3.31 ± 0.85), and this difference was not statistically significant ($p = 0.140$). Similarly, when comparing the groups during the follow-up period, no significant difference was observed ($p = 0.412$). In terms of pain scores, the Coenzyme Q10 group demonstrated a lower mean score at the end of treatment (3.28 ± 1.11) than the control group (4.18 ± 1.42), though this difference did not reach statistical significance ($p = 0.060$). During the follow-up, both groups showed a slight increase in pain scores, and intergroup differences remained statistically non-significant ($p = 0.057$ and 0.452).

Table 1: Baseline Characteristics of the Study Groups

Parameter	Coenzyme Q10 Group	Control Group	p-value
Subjects (migraine without aura)	32	32	
Subjects (migraine with aura)	0	0	
Age (years)	39.75 ± 8.73	39.53 ± 8.81	0.460
Sex - Female	28	30	
Sex - Male	4	2	
Duration of disease/attack (in hours)	4.25	4.34	0.410
No. of migraine days per month	11.25 ± 2.31	11.37 ± 2.35	0.410
Frequency of attack	5.62 ± 1.16	5.68 ± 1.17	0.420
NRS pain score	6 ± 1.19	6.06 ± 1.21	0.420
No. of patients experienced nausea	27 (86%)	25 (78.12%)	0.520
No. of patients experienced vomiting	6 (18.75%)	5 (15.62%)	0.740
No. of patients experienced photophobia	23 (71.87%)	24 (75%)	0.770
No. of patients experienced phonophobia	19 (58%)	20 (62.5%)	0.790

Table 2: Comparison of Clinical Outcomes between Coenzyme Q10 and Control Groups Over a 3-Month Treatment Period

		Coenzyme Q10 (n = 32)	Control (n = 32)	p-value
50% responder rate in frequency attacks	1st month	2 (6.25)	1 (3.13)	0.551
	2nd month	7 (21.88)	3 (9.37)	0.172
	3rd month	16 (50)	7 (21.87)	0.001*
Frequency of Migraine Attacks per Month	1st month	5.125 ± 1.51	5.281 ± 1.19	0.362
	2nd month	3.281 ± 1.08	4.21 ± 1.00	<0.001*
	3rd month	2.721 ± 1.17	3.56 ± 1.01	<0.001*
	RM-ANOVA	<0.001*	<0.001*	
50% Responder Rate in Pain Score	1st month	3 (9.37)	1 (3.12)	0.30
	2nd month	7 (21.87)	3 (9.37)	0.16
	3rd month	13 (40.62)	5 (15.62)	0.025
Pain scores	1st month	5.34 ± 1.57	5.59 ± 1.36	0.001*
	2nd month	3.81 ± 1.20	4.75 ± 1.29	<0.001*
	3rd month	3.28 ± 1.11	4.18 ± 1.42	<0.001*

	RM-ANOVA	<0.001*	<0.001*	
Monthly Migraine Days	1st month	9.87 ± 3.32	10.31 ± 2.38	0.272
	2nd month	6.50 ± 2.03	8.18 ± 1.92	<0.001*
	3rd month	5.21 ± 2.15	7.12 ± 2.02	<0.001*

Table 3: Comparison of Associated Migraine Symptoms between Coenzyme Q10 and Control Groups Over Three Months

Month	Symptom	Coenzyme Q10 (n = 32)	Control (n = 32)	p-value
1st	Nausea	22 (68.75)	24 (75.00)	0.572
1st	Vomiting	6 (18.75)	5 (15.62)	0.741
1st	Photophobia	19 (59.37)	23 (71.87)	0.290
1st	Phonophobia	17 (53.12)	20 (62.50)	0.442
2nd	Nausea	16 (50.00)	22 (68.75)	0.120
2nd	Vomiting	3 (9.37)	4 (12.50)	0.681
2nd	Photophobia	13 (40.62)	22 (68.75)	0.002*
2nd	Phonophobia	12 (37.50)	19 (59.37)	0.071
3rd	Nausea	11 (34.37)	20 (62.50)	0.002*
3rd	Vomiting	1 (3.12)	4 (12.50)	0.160
3rd	Photophobia	8 (25.00)	21 (65.60)	<0.001*
3rd	Phonophobia	7 (21.88)	19 (59.37)	<0.001*

Table 4: Comparison of Migraine Attack Frequency and Pain Scores at the End of Treatment and Follow-Up between Coenzyme Q10 and Control Groups

		Coenzyme Q10 (Mean ± SD)	Control (Mean ± SD)	p-value
Frequency of attacks at the end of treatment; and at the end of follow-up	3rd month (treatment period)	2.71 ± 1.17	3.56 ± 1.01	0.001*
	2nd month (follow-up period)	3.37 ± 1.12	3.31 ± 0.85	0.140
	Between groups in 2nd month of follow-up	3.37 ± 1.12	3.31 ± 0.85	0.412
Pain scores at the end of treatment; and at the end of follow-up	3rd month (treatment period)	3.28 ± 1.11	4.18 ± 1.42	0.060
	2nd month (follow-up period)	3.68 ± 0.96	3.65 ± 1.23	0.057
	Between groups in 2nd month of follow-up	3.68 ± 0.96	3.65 ± 1.23	0.452

Discussion

Migraine is a complex neurological disorder characterized by recurrent episodes of headache, typically unilateral and often accompanied by sensory or visual disturbances collectively termed as aura.[12] Several theories have been proposed to explain its pathogenesis, one of which is the mitochondrial hypothesis. This suggests that mitochondrial dysfunction may play a key role in the initiation and perpetuation of migraine. The condition significantly affects the quality of life and socio-economic status of individuals. Given the limited availability of effective prophylactic medications and the need for therapies with fewer adverse effects, both pharmacological and non-pharmacological agents such as magnesium, riboflavin, and Coenzyme Q10 have been extensively studied. Among these, Coenzyme Q10 has demonstrated promising efficacy in migraine prevention, attributed largely to its antioxidant properties.[16] In this open-label study, Coenzyme Q10 was administered at a daily dose of 100 mg as an adjunct to standard migraine prophylactic therapy. A total of 68 patients were recruited, of whom 64 completed the study—32 in the Coenzyme Q10 group and 32 in the control group. Complete data were available for all 64 participants. The majority of the study population was middle-aged, with the mean age in the Coenzyme Q10 group being 39.75 ± 8.73 years and 39.53 ± 8.81 years in the control group. The study population was predominantly female. Of the 68 participants, 58 (90.62%) were women, and only 6 (9.37%) were men. This gender distribution aligns with the findings of Ray et al., who reported a female prevalence of 81.87% among migraine sufferers,[6] and with Ornello et al., who observed a female-to-male ratio ranging from 2:1 to 3:1, peaking during midlife.[17]

Baseline characteristics were comparable between the two groups. The primary outcome measures included the 50% responder rate in the monthly frequency of migraine attacks and changes in the mean frequency over time. Secondary outcomes assessed included the 50% responder rate and mean changes in the NRS pain score, monthly migraine

days, and analysis of associated symptoms. At the end of the treatment period, 50% of patients in the Coenzyme Q10 group achieved a ≥50% reduction in attack frequency compared to 21.87% in the control group. Sixteen out of 32 patients in the Coenzyme Q10 group showed a significant response, compared to 7 out of 32 in the control group. These findings are consistent with a study by Sándor et al., which used 300 mg/day of Coenzyme Q10 and reported a 50% responder rate of 47.6% in the intervention group versus 14.4% in the placebo group.[18]

Although improvement was noted in both groups from the first month, statistical significance in attack frequency was observed only at the third month. Similarly, a study by Rozen et al., involving 31 patients treated with 150 mg/day of Coenzyme Q10, found that 61.3% achieved a 50% reduction in migraine days.[19] A higher dose (400 mg/day) used in a study by Dahri et al. in 77 women yielded a 50% responder rate of 53.8%, further supporting its efficacy.[15]

In the present study, within-group comparisons using t-tests revealed a consistent decline in migraine frequency from baseline to the end of treatment. Baseline means were 5.62 and 5.68 in the Coenzyme Q10 and control groups, respectively, which declined to 2.72 and 3.56 by the third month. Between-group analysis showed significant reductions in the Coenzyme Q10 group from the second month onward, indicating the effectiveness of Coenzyme Q10 as an adjunctive therapy. This result concurs with findings by Dahri et al., who reported a reduction from 8.20 to 3.76 in migraine frequency.[15]

With regard to pain intensity, the 50% responder rate in NRS pain score was 40.62% in the Coenzyme Q10 group and 15.62% in the control group. A statistically significant difference was observed at the third month. The mean pain scores declined from 6.00 to 3.28 in the Coenzyme Q10 group and from 6.06 to 4.18 in the control group. Notably, a statistically significant difference between the groups was observed as early as the first month. These findings are supported by a study by Shoeibi et al., where pain scores were reduced from 8.5 to 2.5 with Coenzyme Q10 treatment.[20]

Monthly migraine days also showed a continuous decline from baseline to the third month in both groups: from 11.25 to 5.21 in the Coenzyme Q10 group and from 11.27 to 7.12 in the control group.

Intergroup comparisons showed significant reductions from the second month onward, consistent with the findings of Dahri et al.[15] However, a study by Sándor et al. reported a relatively weaker reduction in monthly migraine days with Coenzyme Q10.[18] In terms of associated symptoms—nausea, vomiting, photophobia, and phonophobia—a reduction was observed in all except vomiting by the end of the third month. This may be attributed to the small number of participants experiencing vomiting. These findings are in agreement with the study by Shoeibi et al., which used 100 mg/day of Coenzyme Q10 as add-on therapy.[20] No major adverse events were reported during the study. One patient experienced mild giddiness in the first month, which resolved spontaneously. No dropouts occurred due to adverse effects. This favourable safety profile is supported by previous studies: Sándor et al. reported one case of cutaneous allergy,[18] while Shoeibi et al.[20] and Dahri et al.[15] Reported no significant adverse events.

Following discontinuation of Coenzyme Q10, the frequency of attacks and monthly migraine days significantly increased at the two-month follow-up, while no significant changes were seen in the control group.

Pain scores showed no significant difference in either group post-discontinuation. Between-group comparisons at follow-up revealed no statistically significant differences in attack frequency, pain score, or monthly migraine days, suggesting symptom relapse after stopping the supplement. These findings support the antioxidant role of Coenzyme Q10 and its hypothesized mechanism in enhancing mitochondrial function. Given the emerging evidence linking mitochondrial dysfunction to migraine pathogenesis, Coenzyme Q10 may serve as an effective and well-tolerated adjunctive therapy for migraine prophylaxis. The present study was limited by its open-label design, which may have introduced observer and participant bias. The relatively small sample size and short duration of follow-up restrict the generalizability of the findings.

Additionally, the study did not assess long-term adherence or sustained efficacy beyond the follow-up period. Biochemical markers of oxidative stress or mitochondrial dysfunction were also not evaluated.

Conclusion

Based on the findings of the present study, Coenzyme Q10 at a daily dose of 100 mg demonstrated significant efficacy in reducing the frequency of migraine attacks, headache severity, monthly migraine days, and associated symptoms, with an acceptable tolerability profile. These results support its use as an effective adjunctive therapy in migraine prophylaxis. However, further studies with larger sample sizes and longer follow-up periods are warranted to validate and extend these findings.

References

1. Rizzoli P, Mullally WJ. Headache. The American Journal of Medicine. 2018; 131(1):17-24.
2. Ahmed F. Headache disorders: differentiating and managing the common subtypes. Br J Pain. 2012; 6(3):124-32.
3. Woldeamanuel YW, Cowan RP. Migraine affects 1 in 10 people worldwide featuring recent rise: A systematic review and meta-analysis of community-based studies involving 6 million participants. J Neurol Sci. 2017; 372:307-15.
4. Goadsby PJ. Pathophysiology of migraine. Ann Indian Acad Neurol. 2012; 15(Suppl 1):S15-22.
5. 5. Benjamin T, Gillard D, Abouzari M, Djalilian HR, Sharon JD. Vestibular and auditory manifestations of migraine. Curr Opin Neurol. 2022; 35(1):84-9.
6. Ray BK, Paul N, Hazra A, Das S, Ghosal MK, Misra AK, et al. Prevalence, burden, and risk factors of migraine: A community-based study from Eastern India. Neurol India. 2017; 65(6):1280-8.
7. Zameel Cader M. The molecular pathogenesis of migraine: new developments and opportunities. Human Molecular Genetics. 2013; 22(R1):R39-R44.
8. Jacobs B, Dussor G. Neurovascular contributions to migraine: Moving beyond vasodilation. Neuroscience. 2016; 338:130-44.
9. Cano A, Palomeras E, Alfonso S, Ortega D, Sanz P, Fossas P. Migraine without aura and migrainous disorder in children; International Headache Society (IHS) and revised IHS criteria. Cephalalgia. 2000; 20(7):617-20.
10. Dave J, Hakkinen I, Zhang P. Comprehensive list of preventative migraine headache medications without significant drug-drug interactions. Front Neurol. 2024; 15:1527897.
11. Guerzoni S, Pellesi L, Baraldi C, Cainazzo MM, Negro A, Martelletti P, et al. Long-term Treatment Benefits and Prolonged Efficacy of OnabotulinumtoxinA in Patients Affected by Chronic Migraine and Medication Overuse Headache over 3 Years of Therapy. Front Neurol. 2017; 8:586.
12. D'Onofrio F, Raimo S, Spitaleri D, Casucci G, Bussone G. Usefulness of nutraceuticals in migraine prophylaxis. Neurol Sci. 2017; 38(Suppl 1):117-20.
13. Rodick T, Seibels D, Jeganathan R, Huggins K, Ren G, Mathews S. Potential role of coenzyme Q10 in health and disease conditions. Nutrition and Dietary Supplements. 2018; 10:1-11.
14. Stuart S, Griffiths LR. A possible role for mitochondrial dysfunction in migraine. Mol Genet Genomics. 2012; 287(11-12):837-44.
15. Dahri M, Tarighat-Esfanjani A, Asghari-Jafarabadi M, Hashemilar M. Oral coenzyme Q10 supplementation in patients with migraine: Effects on clinical features and inflammatory markers. Nutr Neurosci. 2019; 22(9):607-15.
16. Buse DC, Manack AN, Fanning KM, Serrano D, Reed ML, Turkel CC, et al. Chronic migraine prevalence, disability, and sociodemographic factors: results from the American Migraine Prevalence and Prevention Study. Headache. 2012; 52(10):1456-70.
17. Ornello R, Ahmed F, Negro A, Miscio AM, Santoro A, Alpuente A, et al. Is There a Gender Difference in the Response to onabotulinumtoxinA in Chronic Migraine? Insights from a Real-Life European Multicenter Study on 2879 Patients. Pain Ther. 2021; 10(2):1605-18.
18. Sándor PS, Di Clemente L, Coppola G, Saenger U, Fumal A, Magis D, et al. Efficacy of coenzyme Q10 in migraine prophylaxis: a randomized controlled trial. Neurology. 2005; 64(4):713-5.
19. Rozen TD, Oshinsky ML, Gebeline CA, Bradley KC, Young WB, Shechter AL, et al. Open label trial of coenzyme Q10 as a migraine preventive. Cephalalgia. 2002; 22(2):137-41.
20. Shoeibi A, Olfati N, Soltani Sabi M, Salehi M, Mali S, Akbari Oryani M. Effectiveness of coenzyme Q10 in prophylactic treatment of migraine headache: an open-label, add-on, controlled trial. Acta Neurol Belg. 2017; 117(1):103-9.