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The effects of coenzyme q10 in acute myocardial infarction

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Abstract
Introduction: Epidemiological reports from the world Health Organization and American Heart Association beginning in the late 1950s required the Presence of at least two or the following for the diagnosis of myocardial infarction: characteristic symptoms, electrocardiographic changes, and a typical rise and fall in biochemical markers. This epidemiological approach was then generally adopted in routine clinical practice, although the rigor with which clinicians apply the electrocardiographic and biochemical criteria for infarction varies considerably.
Methodology: It was randomized placebo-controlled, interventional study. Study was conducted among all clinically and electrocardiographically defined MI patients admitted in Department or Medicine. All clinically defined MI patients were considered. Patients fulfilling the following inclusion criteria were included in the study.
Results: The region-wise distribution of Ml was 69 patients (70.40%) in AMI, 5 patients (5.1%) in global MI and 24(24.48%) in inferior wall MI in CoQ10 and placebo groups (p= 0.541) which was statistically insignificant and comparable among both groups.
Conclusion: CoQJ0 being a naturally occurring biological molecule it can have synergistic effects with fibrinolysis associated myocardial reperfusion injury and its complication.

Keywords: effects, coenzyme q10, acute myocardial infarction

Introduction

Myocardial infarction occurs when myocardial oxygen supply is inadequate compared to myocardial oxygen demand. Myocardial ischemia usually occurs in the setting or coronary atherosclerosis but May also reflect dynamic components or coronary vascular resistance. Coronary spasm can occur in normal coronary arteries, or, in patients with coronary disease, near atherosclerotic plaques arid in smaller coronary arteries. Other, less common causes or impaired coronary blood flow include syndromes that compromise the orifices of the coronary arteries or the arteries themselves, such as syphilitic aortitis, arteritides, aortic dissection, myocardial bridges, or congenital abnormalities of the coronary arteries [1].

The pathological diagnosis of myocardial infarction (Ml) requires evidence of myocyte cell death as a consequence of prolonged ischemia. Characteristic findings include coagulation necrosis and Contraction band necrosis, often with patchy areas of myocytolysis at the periphery of the infarct. During the acute phase of Ml, the majority of myocyte loss in the infarct zone occurs via coagulation necrosis and proceeds to inflammation, phagocytosis of necrotic myocytes, and repair eventuation in scar formation [2].

The clinical diagnosis of MI requires an integrated assessment of history with some combination of indirect evidence of myocardial necrosis using biochemical, electrocardiographic, arid imaging modalities. The sensitivity and specificity of the clinical tools for diagnosing MI vary considerably and change at varying times after the onset of the infarction [3].

Epidemiological reports from the world Health Organization and American Heart Association beginning in the late 1950s required the Presence of at least two or the following for the diagnosis of myocardial infarction: characteristic symptoms, electrocardiographic changes, and a typical rise and fall in biochemical markers. This epidemiological approach was then generally adopted in routine clinical practice, although the rigor with which clinicians apply the electrocardiographic and biochemical criteria for infarction varies considerably [4].

Since the original epidemiological efforts, considerable advances have occurred in the electrocardiographic and biochemical aspects of the definition of infarction. The electrocardiographic criteria for MI were codified, and scoring systems were developed for estimation of infarct size. Biochemical assays became available for markers more specific for cardiac damage. These include mass assays for the MB fraction of creatine kinase (CK) and immunoassays for cardiac-specific troponins. The cardiac-specific troponin assays have nearly absolute myocardial tissue specificity and have become the predefined biomarker for diagnosing MI. Advances in the techniques for diagnosing Ml were the impetus for a consensus document published jointly by several prominent cardiac societies around the world. The revised definition of MI has important implications nut only for clinical e of patients but also for tracking epidemiological trends, public policy, and clinical trials The paradigm shift to cardiac-specific troponins at the markers or choice for the diagnosis of MI requires new cut-off values for cardiac injury. The term normal range has been replaced by upper reference limit. Defined the 99th percentile or a normal reference control group [5].

Ubiquinone commonly referred to as coenzyme Ql0, was originally so named because of its omnipresence in virtually every cell of the human body. According to Dr. Karl Folkers,

a pioneer in the CoQ10 fermentation synthesis, CoQ10 should be properly renamed "vitamin Q," and thus take its rightful place in the pantheon of essential nutrients [6].

Two main theories have been set forth as to how CoQ10 assists in achieving optimal health and regeneration from illness. First is its ability to increase the amount of' energy available to those part of' the body whose cells most require it, including the heart, brain, kidneys, and skeletal muscles, among others. Second, many of the benefits derived from CoQ1O are thought to be a result of its potent antioxidant effects, as it scavenges dangerous free radical oxygen species that normally harm the body.

Methodology

Study Design

It was randomized placebo-controlled, interventional study.

Study Location

Study was conducted among all clinically and electrocardiographically defined MI patients admitted in Department or Medicine.

Study Duration

It was done during the period of one year.

Selection Patients

All clinically defined MI patients were considered. Patients fulfilling the following inclusion criteria were included in the study.

Inclusion Criteria

1. Selection of the cases for the study will be random
2. Diagnostic criteria for acute myocardial infarction are:

a. Appearance of Si' segment elevation:

b. Significant enzyme rises in the presence of evolution t typical electrocardiography pattern.

Exclusion Criteria

1. Ventricular aneurysm & hypertrophy.
2. Fixed ECG abnormalities not due to myocardial infarction.
3. Previous MI.
4. Valvular and other non-coronary heart disease.
5. Presence of serious non coronary disease example cerebrovascular accident that might contribute to mortality.
6. Angina pectoris with cardiac chest pain <30 min.
7. Diarrhoea, dysentery and persistant vomiting.
8. Acute MI non-thrombolysed.

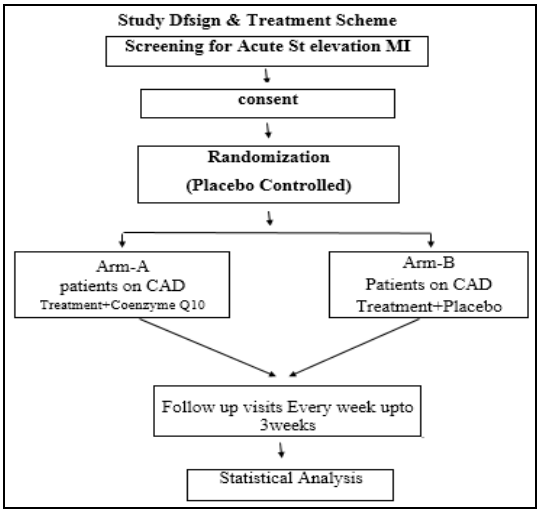

Fig 1

Results

There were 98 patients in the study. Patients were divided into two equal groups. Group (A) included patients with MI treated with Coenzyme Q 1 0 and conventional treatment Group (B) included patients treated with conventional treatment & placebo.

Table 1: Age distribution in the two experimental groups

Age group	coQ10 (%)	Placebo (%)	Grand Total (%)
30-39	10(20.4%)	7(14.28%)	17(17.34%)
40-49	13(26.53%)	12(24.48%)	25(25.51%)
50-59	12(24.48%)	17(34.69%)	29(29.59%)
>60	14(28.57%)	13(26.53%)	27(27.55%)
Grand Total	49	49	98

In our study group, the age group distribution was 10 and 7 in the age group of 3 0-39, 13 and 12 in the age group of 40-49, 12 and 17 in the age group of50- 59 and 14 and 13 in the age group of 60 and above in the CoQ1O and placebo group respectively. Distribution or age was comparable in both groups. The maximum no. of patients were in the age group of 50-59[29(29: 9%) l and the next most common age group is 60 and above 27(27.55%)].

Table 2: Lipid profile

	Groups	N	Mean	Std. deviation	Std. Error Mean	Student T Test	Statistical significance
Total. Lipids	CoQ 10	49	556.7143	122.21497	17.45928	p>0.05	NS
	Placebo	49	579.2449	126.86369	18.12338		
Total. cholestpron	CoQ 10	49	164.3673	37.99983	5.42855	p>0.05	NS
	Placebo	49	169.5306	41.09446	5.87064		
HDL	CoQ 10	49	46.5918	13.41783	1.91683	p>0.05	NS
	Placebo	49	46.0408	12.11053	1.73008		
LDL	CoQ 10	49	91.0816	33.33469	4.76210	p>0.05	NS
	Placebo	49	94.0612	37.15026	5.30718		
VLDL	CoQ 10	49	30.2959	13.85499	1.97928	p>0.05	NS
	Placebo	49	32.4347	14.70483	2.16669		

In our study group, the mean total lipids were 556.lmg/dl and 579.24mg/dl, the total mean cholesterol was 164.36 mgh.11

and
169.53mg/di, the mean l-IDL was 4659 mg/dl and 46.04 mg/dl, the mean [DL was 91.O8mg/dl and 94.O6mg/dl and the mean VLDL was 30.29mg/dl and 32.4 mg/dl in CoQ10 and placebo group respectively. It was comparable in both the groups (P>0.05).

Table 3: Mortality

Death	CoQ10	Placebo	Grand total
Yes	2(4%)	4(8%)	6(6.1%)
No	47	45	92
Grand total	49	49	98

Chi-square — 0.178 with 1 degree of freedom: P = 0.673

The distribution of mortality in our study group was 2 (4%) and 4 (8%) in CoQ10 and placebo groups respectively (p=O.673) which was statistically insignificant and comparable in both the groups.

Table 4: Distribution of MI

ECG	CoQ10	Placebo	Grand Total
Anterior Wallmi	32	37	69. (70.40%)
GLOBLE	03	02	5(5.1%)
Inferior wall MI	14	10	24(24.48%)
Grand total	49	49	98

Chi-square = 1.229 with 2 degrees of freedom: P = 0.541

The region-wise distribution of Ml was 69 patients (70.40%) in AMI, 5 patients (5.1%) in global MI and 24(24.48%) in inferior wall MI in CoQ10 and placebo groups (p= 0.541) which was statistically insignificant and comparable among both groups.

Table 5: 2DECHO

2D ECHO C.I.	CoQ10	placebo	Grand Total
20-29%	0	10(20.40%)	10(10.20%)
30-39%	10(20.40)	13(26.53%)	23(23.46%)
40-49%	30(61.22%)	19(38.77%)	49(50%)
>50%	9(18.36%)	7(14.28%)	16(16.32%)
Grand Total	49	49	98

Chi-square = 13.111 with 3 degrees of freedom: P 0 0o6

The cardiac output (CO) as indicated by LVEF was 0 and 10 between LVEF of 20-29%. 10 and 13 between 30-39%, 30 and 19 between 40-49% and 9 and 7 in >50% in COQ 10 and placebo group respectively (p=0.0O6). The values were both statistically and echocardiographically significant showing the patients who were receiving CoQ10 supplement did not have drop in LVEF compared to placebo group.

Table 6: ECG ON DAY 7& DAY21

Group	Arrhythmia				Grand total
	7days		21days		
	yes	NSR	Yes	NSR	
CoQ10	2	45	0	47	47
placebo	7	38	1	44	45
Grand total	9	83	1	91	92
Chi-square Test	p>0.05		p>0.05		NS

The ECG on admission, there Were no arrhythmias noted in patients belonging to CoQ10 and placebo groups respectively.
On day 7, arrhythmias were noted in 2(4%) and 7 (14.26%) patients belonging to CoQ10 and placebo groups respectively
On day 21, arrhythmias were noted in 0 and I (2%) patients belonging toCoQ10 and placebo groups ‘respectively.
On all the 3 days the incidence of arrhythmias was statistically. Insignificant but clinically significant o11 day 7 (since less number of patient had arrhythmias in the CoQ10 group).

Discussion

Age itself is an important risk factor for CAD & hence also for STEMI. The incidence of CAD & MI increases as the age increases& reaches its peak between 61h and 7th decade.
In our study 29 patients (29.59%) were in the age group of 50-59, and 27 patients (27.55%) were in the age group of 60 and above. The lowest age observed was 30 years & the highest age observed Was 78 years.
American heart association 1271 study showed the MI was commonest in the 6^{th} and 7^{th} decade (in about 4 or every 5 deaths due to heart disease occur in people older than 65 years) which is comparable to our study.
Dyslipidemia is associated with CAD & MI. the severity of which correlates well with the severity of dyslipidemia & its duration.
In our study 39 (39.79) no of patients were round to be dyslipidenic.
In our study 93 patients(94.89%)were found to have optimal total cholesterol,92 patients(93.87%)were found to have optimal level or IIDL, 93 patients(94.89%)were found to have optimal level of LDL & 61 patients(62.24%)were found to have optimal level of VLDI..
In our study 5 patients(5.1O%)were found to have deranged total cholesterol, 6 patients(6. 12%)were found to have deranged level of HDL, 5 patients(5.1O%)were found to have deranged level of LDL & 37 patients(37.75%)were found to have deranged level of VLI)L.
Both the study & the placebo groups had similar distribution of dyslipidemic patients.
The 2D Echocardiography is a sensitive Investigation to estimate the cardiac function viz stroke volume, ejection fraction (EF), cardiae output& cardiac index. LVEF is a direct indictor or left ventricular function.
We studied the EF of the patients of both the groups between 7 & 14 days after Ml. our study showed that there “is significant difference between CoQ10 and placebo groups with respect to drop in LVEF post MI i.e. patients who received CoQI0 did %t have fall in LVEF [30(61.22%) v/s 19(38.77%) between 40-49% and 0 v/s 10(20.4o%) in the LVEF between 2O-29% in the CoQ10 and placebo. Groups respectively] when compared to the placebo groups which were matched similarly. Our results were comparable to the results or Rain Singh *et al*. study
After Ml arrhythmias are common, which are the important factors contributing to mortalities. Arrhythmias may be in the form of ventricular ectopics, AF, SVT, VT, VF etc. with or without bundle brand1 blocks. Inferior wall MI may be associated with bradyarrhythmias because or involvement of conduction system.
These are supposedly due to myocardial reperfusion injury, they can occur immediately during or after thrombolysis. So

for studies with respect to CoQ10 have shown to decrease dysrythmias in the pen and post thrombolytic period [7, 8]. In our study only on up to D7 the incidence of arrhythmias was less in CoQ1O group when compared to placebo group. Even though statistically insignificant it can be attributed to the administration of CoQI0 which has a negative effect on the incidence or reperfusion injury associated arrhythmias [9, 10].

Among 98 patients 7 patients were found to have hyperhormocystenemia out of which 3 and 4 were placed in placebo & CoQ10 group respectively.

The severity of CAD with respect to angioplasty were not studied. The other comorbidites like DM, impaired renal function& other factor were not studied. Strength of our study is both the groups were comparable with respect to various parameters viz age, sex, tobacco use, alcohol consumption and other parameters. All patients were evaluates for risk factor & no follow up was lost.

Conclusion

This case-controll study shows that CoQ10 a known free radical scavenger and cell membrane stabilising agent thus exerts a cardioprotective effect during post MI period.

Over 21 days most end points were similar in the interventional group showing better preserved LVEF, lesser no of arrythmias and lesser no of deaths.

References

1. Serebriuany VL, Ordonez JV, Herzog WR, Morten R, Mortensen SA, Folkers K, Gurbel PA. Dietary coenzyme Ql0 supplementation alters platelet size and inhibits human Vitronectin (CDSI/CD6 1) receptor expression. J Cardiovasc Pharmacol. 1997; 29:16-22.
2. Battino M, Fato R, Lemz G. Coenzyrne Q10 can control the efliciency of oxidative phosphorylation. lnt J Tissue Reac. 1990; 12:137-l4.
3. Baggio G, Gandini R, Plancher AC, passeri M, Carmosino G. Italian multicenter study on the safety and ef-cacy of coenzyrne Q1O as adjunctive therapy in heart failure. Mol Aspects Med. 1994; 1(5):287-294.
4. Singh RB, Niaz MA, Rastogi V, Rastgi) 3S. Coenzyme Q in cardiovascular disease. Jssoc Physiol 1nJ. 1998; 46:299-306.
5. Kamikawa T, Kobayashi A, Yamashita T, Hayashi H, Yamasaki N. Effects of coenzyme Q1O on exercise tolerance in chronic stable angina pectoris. Am J Cardiol. 1985; 56:247-257.
6. Mazzola C, Guffanti EE, taccarella A, Meregalli M, Colmago R, Ferrario N, Cantoni V, Marcherri G. Noninvasive assessment of coenzyme Ql0 in patient with chronic stable effort angina and moderate heart failure. Cure [her Res. 1987; 41:923-930.
7. Hofman-Bang C, Rehnqvist N, Swedberg K, Astrom H. Coenzyme Q10 as an adjunctive in treatment of congestive heart failure. J Am Coll Cardiol. 1992; 19:774-776.
8. Rosi E, Lombardo A, Tesla M, *et al*. Coenzyme Q10 in ischaemic cardiopathy. In: Folkers K, Litlarru GP, Yamagami T, eds. Biomedical and Clinical Aspects of Coenzyme Q, Amsterdam: Elsevier. 1991; 6:321-326.
9. Barbiroli B, Frassineti C, Martinelli P, lotti S, Lodi R, Cortelli P, Montngna P. Coenzyrme Q10 improves mitochondrial respiration in patients with mitrochondria1 cytopathies. An in vivo study on brain and skeletal muscle 1y phosphorous magnetic resonance spectroscopy. Cell Mo! 13i01. 1997; 43:741-749.
10. Yoshida T, Maulik G, Bagchi D, Dash SK, Engelman RM, Das DK. Increased myocardial tolerance to ischemia-reperfusion injury by feeding.pigs with coenzyme Q10. In: Das DK, Engelman R, Cherian KM, eds. Myocardial Preservation. Preconditioning and Adaptation. Ann NY Acad Sci. 1996; 793:414-4l9.