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Coenzyme q10 and vital parameters: In acute myocardial infarction patients

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Abstract
Introduction: Acute myocardial infarction (AMI) may be associated with reperfusion induced & fee radical stress, lipid peroxidation, and antioxidant vitamin and coenzyme Q deficiency.These situations are predisposing actors for cardiac muscle necrosis, resulting in arrhythmias, ischemia, myocardial dysfunction, and coronary thrombosis, which continue as a chain reaction for several weeks after infrac1ion.
Methodology: On admission patients vitals and other systems were clinically examined. It was then followed by serial ECGs and 2D ECHO. Routine laboratory tests as per proforma were done. It was ensured that vitals and other systems were evaluated by the sane person.
Results: In our study the mean distribution of SBP was 1 16.46mmHg and 123.37mnmnHg in CoQI0 and placebo groups with p<O.O5 which *was* statistically significant but the decrease in SBP between these groups was clinically insignificant. The mean distribution of DBP was78.89mmHg and 76.75rnrnllg in CoQ1O and placebo group with p>O.05 which was statistically insignificant.
Conclusion: Most of the mortalities occurred in the first few this during which much action of CoQ10 can be expected. It is probably a chance factor that more deaths have occurred in the placebo group.

Keywords: CoQ1O, vitals, SBP

Introduction

Myocardial infarction is a myocyte cell death as a consequence or 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 eventuating in scar formation [1].

The clinical diagnosis or Ml requires an integrated assessment or history with some combination or indirect evidence of myocardial necrosis using biochemical, electrocardiographic, and imagine modalities. The sensitivity and specificity of the clinical tools for diagnosing Ml vary considerably and change at varying times after the onset of the infarction [2].

Epidemiological reports from the world Health organization and American Heart association beginning in the late l950s required the presence of at least two of 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.

Acute myocardial infarction (AMI) may be associated with reperfusion induced & fee radical stress, lipid peroxidation, and antioxidant vitamin and coenzyme Q deficiency [2-5]. These situations are predisposing actors for cardiac muscle necrosis, resulting in arrhythmias, ischemia, myocardial dysfunction, and coronary thrombosis, which continue as a chain reaction for several weeks after infrac1ion [2-6]. coenzyme Q has been demonstrated to enhance cell membrane stabilization in vitro and to exert bioenergetic and antioxidant effects by action as a free radical scavenger [7].

Coenzyme Q is a rate-limiting factor in mitochondrial respiratory activity, and a deficiency of coenzyme Q may result in a decrease in intracellular adenosine triphosphate [8].

In one experiment in a swine model [9]. Improved myocardial contractility after coenzyme Q supplementation during ischemia reperfusion injury was observed. Recent Studies1101 indicate that coenzyme Q can inhibit platelet aggregation and human vitronectin receptor expression [11]. Which are important predisposing mechanisms or coronary thrombosis after AMI. Coenzyme Q a naturally occurring vitamin-like agent that is normally present in cardiac cells and function as

An electron carrier in oxidative phosphorylation [12]. In general, it has no adverse effects. There is much clinical experience, with coenzyme Q therapy in angina pectoris, heart failure, arrhythmias and myocardial ischemia. However, no large-scale, randomized, and controlled trials have been published to date that prove its efficacy in coronary artery disease.

CoQ10 has been shown to be effective against chronic inflammation of the arteries and heart muscle tissue resulting in cardiac myopathy. In addition, studies by Japanese and Australian re-searchers, as well as by scientists in the US and elsewhere, have consistently shown the supplement's effectiveness against congestive heart failure and in preventing secondary cardiac events after patients have suffered an initial heart attack.

In Molecular cell biology [13]. Drs. Singh and Kumar published the results of another randomized, double-blind, placebo-controlled study showing CoQ10's benefits in combating atherosclerosis, increasing survival, and reducing the risk of subsequent cardiac events in heart attack patients, including those taking lipid-lowering drugs.

The scientists reported that among 73 patients receiving 120 mg per day of oral CoQI0 for one year after a first heart attack, the treated subjects suffered significantly fewer cardiac events than their untreated counterparts (24.6% v/s 45%). The CoQ1 0 group had a lower incidence of non-fatal heart attacks (13.7% v/s 25.3%) and significantly fewer deaths than the untreated patients.

Methodology

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

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.

Sample Size

All clinically and electrocardiographically defined MI patients admitted in MU-I during the study period. 98 patients were included in the study.

Data Collection

On admission patients vitals and other systems were clinically examined. It was then followed by serial ECGs and 2D ECHO. Routine laboratory tests as per proforma were done. It was ensured that vitals and other systems were evaluated by the sane person.

Allocation of groups

Allocation of the groups was done by randomization using lottery method with replacement-Group A (case group) and Group B (control group).

Intervention

Group a (case group): the active drug was provided in the form of capsules. One capsule was given daily for the period or 21 days. Each capsulecontaining200mgofCoenzymcQ10.

Group 13 (control group): conventional treatment with placebo for21days.

Results

Table 1: Sex distribution in 2 experimental groups

sex	CoQ10	placebo	Grand Total
Male	39(79.59%)	37(75.51%)	76(77.55%)
Female	10(20.40%)	12(24.48%)	22(22.44%)
Grand total	49	49	98

In our study group, the sex distribution was 39 and 37 in the male group and 10 and 12 in the female group for CoQ10 and placebo respectively. The sex distribution was comparable in both the groups. The total no. of males were 76 (77.55%) and the total no. of females were 22 (22.44%).

Table 2: Tobacco use

Tobacco	placebo	CoQ10	Grand Total
Yes	28(57.14%)	30(61.22%)	58(59.18%)
No	21	19	40
Grand total	49	49	98

Chi-square = 0.042 with I degree of freedom: P=0.837

In our study, the distribution of patients in placebo and CoQ10 with history of tobacco use/consumption was 28 and 30, and 21 and 19 in the group with no history of tobacco use. Both the groups were comparable. The total no. of patients with history of tobacco use/consumption were 58(59.18%).

Table 3: Alcohol consumption

Alcohol	Placebo	CoQ10	Grand Total
Yes	18(36.73%)	19(38.7%)	37(37.75%)
No	31	30	61
Grand Total	49	49	98

Chi-square 0.000 with 1 degree of freedom: P 1

In our study group the distribution of patients in placebo and CoQI0 with history or alcohol consumption was I 8& 19 and 31 &30 in the group with no history of alcohol consumption. l3oth the groups were comparable. The total no. of patients who were consuming alcohol in significant amount was 37 (37.75%).

Table 4: Vitals on Admiission

	Group	N	Mean	Std. deviation	Std. error mean	Student T test	Statistical significant
Heart rate	CoQ10	49	84.1224	18.47254	2.63893	P>0.05	NS
	Placebo	49	82.4082	17.30115	2.47159		
SBP	CoQ10	49	118.8571	20.16598	2.878085	p>0.05	NS
	Placebo	49	124.8980	26.05463	3.72209		
DBP	CoQ10	49	79.8367	13.11384	1.87341	p>0.05	NS
	Placebo	49	77.3469	17.32237	2.47462		

In our study the mean distribution of heart rate in the patients was 84.12 & 82.4 in CoQ1O and placebo group (p>O.O5) which was statistically insignificant.

The mean distribution of systolic blood pressure (SBP) was 118 mmllg and 124 mrnllg in CoQ10 and placebo group (p>0.05) which was statistically insignificant.

The mean distribution of diastolic blood pressure (DBP) was 79.83mm1Hlg and 77.34mml-lg in CoQ1O and placebo group (p>0.O5) which was statistically insignificant.

Table 5: Day 7 (Vitals)

	Group	N	Mean	Std. deviation	Std. Error mean	Student T test	Statistical significance
SBP	CoQ10	47	116.4681	11.88505	1.73361	p>0.05	Significant
	Placebo	45	123.3778	13.61698	2.02990		
DBP	CoQ10	47	78.8936	7.73258	1.12791	p>0.05	NS
	Placebo	45	76.7556	9.66991	1.44150		

In our study the mean distribution of SBP was 1 16.46mmHg and 123.37mnmnHg in CoQI0 and placebo groups with p<O.O5 which *was* statistically significant but the decrease in SBP between these groups was clinically insignificant.

The mean distribution of DBP was78.89mmHg and 76.75rnrnllg in CoQ1O and placebo group with p>O.05 which was statistically insignificant.

Table 6: Day 21(Vitals)

	Group	N	Mean	Std. deviation	Std. Error Mean	Student T Test	Statistical significance
HR	CoQ 10	47	84..6383	9.90482	1.44477	P<0.05	significant
	Placebo	45	77.5111	8.63034	1.28654		
SBP	CoQ 10	47	117.1489	10.63010	1.55056	P<0.05	significant
	Placebo	45	124.3111	12.22483	1.82237		
DBP	CoQ 10	47	78.7778	5.89485	0.85985	P>0.05	NS
	Placebo	45	75.7778	8.22659	1.22635		

In our study the mean heart rate (1-1k) was 84 and 77 in CoQI0 and placebo group (p<O.05) which was statistically significant but clinically insignificant.

The mean SBP was 11 7rnmHg and 124.3 mmHg in CoQ10 and placebo group (p<O.05) which was statistically significant but clinically insignificant.

The mean DBP was 78.89mmHg and 75.77mnmHg in CoQ10 and placebo group with p>0.05 which was statistically insignificant.

Discussion

In our study the male to female ratio was 39:10 & 37:12 in CoQ1O & placebo group respectively. The total no. or males in our study was 76(77.55%) and the total no. of females was 22(22.44%). The ratio was comparable to that of Ram. B Singh et all study.

Before the age 45, cardiovascular disease afflicts few women. By age 60, however, it is the leading Cause or death in women. Although men exhibit a higher incidence of C4D & J\11 at every age, as Well as higher mortality from it the gap narrows substantially after natural and surgical menopause

A wide range of factors may explain the increased risk of CA1 after menopause. These include adverse changes in lipid and glucose metabolism that result in increase in LDL cholesterol and a decrease in I JDL levels, an increase in glucose intolerance, and the changes in all hemostatic factors and vascular function. These changes have been attributed to the decline in endogenous estrogen [14].

Despite observational data and physiological data suggesting benefits or hormone replacement therapy, data from recent randomized trials have not only failed to support a possible benefit of hormone therapy for CAD but indicate that combined estrogen and progestin may actually increase the CAD risk [15].

In the literature AM! Is the commonest followed by inferior and global Ml.

Even in our study AM! Was seen in 69 patients (70.4%), inferior MI in 24 patients (24.4%) & global Ml 5 patients (o.51%).

Tobacco use in various forms viz. smoking, chewing, snuffing etc. is itself an important risk factor for CAD & M1. There is linear correlation between amount of tobacco use and the increase it coronary related events

After discontinuation of tobacco use (specially smoking) the risk normalizes to that of general population after l5 years. In our study tobacco use ‘as observed in 58 patients.

Alcohol consumption is also a risk factor for CAD & MI when its total quantity exceeds more than two drinks per day in males &more than one drink per day in females. (A standard drink is generally considered as 12 ounces of beer, 5 ounces of wine, 1.5 ounces of 80-proof distilled spirit. Each of these contain 12 trams of alcohol).

In contrast to the adverse effects or excessive alcohol consumption, moderate consumption (20-30gfd) appears to be cardioprotective, It raises HI) L cholesterol and is associated with reduced incidence of CAD, stroke and metabolic syndromne13a1.v

In our study the no of patients who exceeded the above limits were 37(37.75%).

In general patients of acute MI tend to have tachycardia during initial few minutes associated with diaphoresis & anxiety. Heart rate tends to normalize once the pain subsides. Only in cases with dysrhythmia or CHF and inferior wall Ml abnormalities in heart rate can be observed.

In our study the heart rates were comparable among both the groups. No clinically significant variation was observed in both groups.

Hypertension is a predisposing factor for CAD & MI.16 no. of patients (16.32%) were observed to be hypertensive on admission as per JNC 7 criteria. Suboptimal treatment. Uncontrolled hypertension & accelerated hypertension tends to have higher association with CAD & MI and with other target organ damage.

Heart disease is the most common cause of death in hypertensive patients. Hypertensive heart disease is the result of structural and functional adaptations leading to left ventricular hypertrophy, diastolic dysfunction, CIF, abnormalities of blood flow due to atherosclerotic coronary

artery disease and microvascular disease, and cardiac arrhythmias. These can be reduced by well controlled hypertension [16].

F.L Rosenfeldt, S.J Haas et all showed that CoQ10 decreases both SBP & DBP. In our study the patients receiving CoQI0 were observed to have lesser SBP than placebo patients. (Mean SBP at 7 days 116 vs 123) even though this is statistically significant, clinically it appears to be insignificant because both the groups do not qualify to be hypertensives according to JNC 7 criteria. No significant difference was found with respect to DBP in both the groups.

Due to the absence of similar randomizations in the patient profiles, not much can be attributed to the positive effects of CoQ1O, it will require a large RCT with similar patient profiles & risk factor to study the effect of CoQ10 on mortality. Moreover most of the mortalities occurred in the first few this during which much action of CoQ10 can be expected. It is probably a chance factor that more deaths have occurred in the placebo group

Conclusion

The mortalities were similar in both study and placebo groups are 2 (4%) & 4 (8%) in coQ1O & placebo groups respectively. clinically even though it looks significant because more mortalities were observed in placebo groups.

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