



## MYOCARDIAL INFARCTION WITH ITS RELATION TO ASCORBIC ACID

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### ABSTRACT

The present study includes a total 66 patients with acute myocardial infarction and 66 age group and sex matched healthy controls. 53 (80.3%) were males and 13 (19.7%) were females. Mean age in patients was  $51.76 \pm 11.1$  and maximum patients were in the age group 40-60 years (75.76%). Mean plasma ascorbic acid in patients ( $0.37 \pm 0.064$  mg/dL) was lower than controls ( $0.58 \pm 0.16$  mg/dL). 50 (75.76%) patients had plasma ascorbic acid  $\leq 0.4$  mg/dL as compared to 5 (7.58%) controls. Low plasma ascorbic acid ( $\leq 0.4$  mg/dL), hypercholesterolemia ( $\geq 200$  mg/dL), increased triglycerides ( $\geq 150$  mg/dL), decreased HDL ( $< 40$  mg/dL), increased LDL ( $\geq 130$  mg/dL) and smoking were significantly associated with acute myocardial infarction. Mean plasma ascorbic acid was significantly lower in males, diabetics, hypercholesterolemic  $\geq 200$  mg/dL, serum triglycerides  $\geq 150$  mg/dL, serum HDL  $< 40$  mg/dL, serum LDL  $\geq 130$  mg/dL, body mass index  $\geq 25$  Kg/m<sup>2</sup>, smoking and history of alcohol intake. Plasma ascorbic acid was significantly lower in cases ( $n=9$ )  $0.36 \pm 0.07$  mg/dL than controls ( $n=17$ )  $0.72 \pm 0.12$  mg/dL without any conventional risk factors ( $p < 0.001$ ). AWMi occurred in 37 (56.06%), IWMI in 27 (40.91%), AWMi+IWMI in 2 (3.03) patients. Mean plasma ascorbic acid was  $0.31 \pm 0.04$  mg/dL,  $0.35 \pm 0.05$  mg/dL,  $0.41 \pm 0.07$  in patients with AWMi+IWMI, AWMi and IWMI respectively. Total of 19 patients died during 1 month follow-up. On admission 50 patients had plasma ascorbic acid  $\leq 0.40$  mg/dL and 16 had  $> 0.40$  mg/dL. A total of 8 patients died within one week in hospital, among them 7 had plasma ascorbic acid  $\leq 0.40$  mg/dL and 1 had plasma ascorbic acid  $> 0.40$  mg/dL. At one week 23 patients had plasma  $\leq 0.40$  mg/dL and 35 had  $> 0.40$  mg/dL. A total of 7 patients died in 2<sup>nd</sup> and 3<sup>rd</sup> week, among them 5 had plasma ascorbic acid  $\leq 0.40$  mg/dL and 2 had plasma ascorbic acid  $> 0.40$  mg/dL. At 3 weeks, 8 patients had plasma ascorbic acid  $\leq 0.40$  mg/dL and 40 had  $> 0.40$  mg/dL. A total of 4 patients died and among them 3 had plasma ascorbic acid  $\leq 0.40$  mg/dL and 1 had plasma ascorbic acid  $> 0.40$  mg/dL. The association was significant ( $p < 0.01$ ) with a relative risk of 15.2 and attributable risk of 93.42% indicating mortality was significantly associated in patients in whom plasma ascorbic acid remained persistently low i.e.  $\leq 0.40$  mg/dL. The mean plasma ascorbic acid was  $0.37 \pm 0.06$  mg/dL,  $0.44 \pm 0.08$  mg/dL, and  $0.50 \pm 0.09$  mg/dL on admission, at one week and after 3 weeks respectively.

**Keywords:** Myocardial infarction, ascorbic acid, Mass Index

### INTRODUCTION

Over last few years, search is going on for new risk factors, and as an outcome of these years of research, new risk factors are appearing on the horizon, like low plasma ascorbic acid levels, microalbuminuria, elevated lipoprotein (a), decreased apo-A1 hyperhomocysteinemia, low serum selenium levels, elevated plasma fibrinogen, etc. Atherosclerosis of coronary arteries commonly causes myocardial infarction and oxidative stress is thought to play an important role in atherosclerotic vascular disease. There is growing body of evidence for role of free radicals in mediating tissue injury during myocardial ischemia and in particular during the phase of myocardial reoxygenation and antioxidants may have a part in improving thrombolytic reperfusion of ischemic myocardium.<sup>1</sup>

In recent years, deficiencies of antioxidants have been extensively studied in the pathogenesis of atherosclerosis and coronary artery disease and subsequent mortality. One of these antioxidants is ascorbic acid (vitamin-C). The ability of vitamin C to donate electrons and sparing effect on other antioxidant vitamins makes it a potent water-soluble antioxidant that readily scavenges free radicals.<sup>2-6</sup> Vitamin C inhibit LDL oxidation and has been demonstrated to potentiate EDNO activity and normalize vascular function in patients with coronary artery disease and associated risk factors including hypercholesterolemia, hyperhomocysteinemia, hypertension, diabetes and smoking.<sup>7</sup> As there is paucity of Indian data regarding the levels of plasma ascorbic acid and their relationship with AMI and subsequent mortality, the role of plasma ascorbic acid in acute myocardial infarction needs further evaluation.

### Aims & Objectives

1. To determine the levels of plasma ascorbic acid in patients of acute myocardial infarction and compare them with age group and sex matched controls.
2. To study the relationship between acute myocardial infarction and plasma ascorbic acid levels.
3. To study the association of plasma ascorbic acid with conventional risk factors for coronary artery disease.
4. To assess the relation of plasma ascorbic acid to short-term mortality in acute myocardial infarction.

### METHODOLOGY

The present hospital based study was undertaken in MR Medical College, Basaveshwar Teaching & General Hospital and Government General Hospital, Gulbarga. Sixty six cases of acute myocardial infarction and equal number of age group and sex matched healthy controls were studied. All the subjects were interviewed, examined, investigated and followed up according to proforma that was predesigned and pretested. The study was approved by the Ethical Committee no. MRMC/IEC/01-02/46.

#### Methodology:

**Study Design:** Case Control Study (age group and sex matched) and prospective follow-up study of patients (hospital based).

**Selection of Cases:** All consecutive cases of acute myocardial infarction who were admitted to intensive cardiac care unit of Basaveshwar Teaching & General Hospital and Government General Hospital, Gulbarga fulfilling the

inclusion and exclusion criteria, during the period from October 2002 to August 2004 were included in the study.

**Inclusion Criteria:** Patients enrolled will be cases of first AMI and the diagnosis was made by the presence of at least two of the following three criteria.<sup>8</sup>

Typical clinical symptoms.

ECG diagnosis of acute myocardial infarction was made by the presence of serial ECG findings such as new pathological Q-waves or 1mm ST segment elevation in any two or more contiguous limb leads or a new LBBB or new persistent ST-T wave changes diagnostic of a non Q-wave myocardial infarction. For posterior wall myocardial infarction (Perloff) R-waves of 0.04 second in V<sub>1</sub> and contiguous right periodical leads and in acute phase, ST depression and R/S=1 in V<sub>1</sub> and V<sub>2</sub> were taken as criteria for diagnosis of acute posterior wall myocardial infarction.

Significant enzyme elevation (i.e., CPK-MB > two times normal).

Only patients who presented within 24 hours of onset of chest pain were included in the study.

**Exclusion Criteria<sup>9</sup>:**

1. Cases who had significant chronic illness (e.g. liver diseases, untreated hyper or hypothyroidism, renal diseases, bleeding tendencies, or malignancy) were excluded as these conditions would have led to change in lifestyle or alteration in the risk factors of AMI.
2. Past history of ischemic heart disease.
3. Hypertension
4. Subjects with intake of rich sources of vitamin-C just prior to study.
5. Cases who did not give informed consent were not included in the study.

**Selection of Controls:**

Age group and sex matched healthy controls were selected for each case. Controls were selected randomly from:

Patients attending OPD for minor ailments like headache, myalgia, refraction, cataract or for physical check up, or from Unrelated attendants or neighbourhood healthy people who visited the case, who had no history or clinical evidence of heart disease.

**Physical Measurement Methods:**

**Height<sup>9</sup>, Weight<sup>9</sup>, Body Mass Index (BMI):**

BMI  $\geq 25$  was considered as a risk factor for coronary artery disease and  $< 25$  was taken as normal.<sup>118</sup>

**Blood Pressure<sup>9</sup>:**Diagnosis of Hypertension: Systolic blood pressure  $>140$  mm Hg and diastolic  $>90$  mm Hg, based on the average of  $\geq$  readings taken on each of two or more visits after an initial screening or one who was known case of hypertension and was on anti-hypertensive medications.

**Chemical Measurements:**

Blood samples were collected within 24 hours, after onset of chest pain, before thrombolytic therapy for patients of acute myocardial infarction as thrombolytic therapy is known to alter plasma ascorbic acid levels by the mechanism of reperfusion injury.<sup>11</sup> Samples were also collected in patients after 1 week and after 3 weeks on follow-up.

For controls, overnight fasting blood samples were collected.

**Measurement of Plasma Ascorbic Acid<sup>12</sup>:**

**Procedure:** 2,4-dinitrophenyl hydrazine method for total serum ascorbic acid (ascorbic acid and oxidation products).

Plasma ascorbic acid  $\leq 0.4$  mg/dL was considered as low and  $>0.4$  mg/dL was taken as normal.<sup>13,14</sup>

**Measurement of Blood Sugar – GOD/POD Method:**

**Diagnosis of Diabetes:<sup>120</sup>**

**Statistical Analysis:**

Statistical analysis included the usual descriptive and univariate analysis. Z-test was used to compare continuous variables. For categorical variables, Chi-square test was used and unadjusted odd's ratios were calculated and 'p' values were computed. In addition to usual descriptive and univariate analysis, multivariate analysis was also performed.

## RESULTS

The study "Relation of Plasma Ascorbic Acid to Mortality in Acute Myocardial Infarction (AMI)" was carried out at Government General Hospital and Basaveshwar Teaching & General Hospital, Gulbarga attached to M.R. Medical College, Gulbarga over a period from October 2002 to August 2004.

Sixty-six cases of acute myocardial infarction admitted to ICCU were studied and compared with equal number of age group and sex matched healthy controls and patients were followed up for short-term mortality for one month and the following observations were noted.

**Table-1: Sex Distribution in Cases**

Sex	No. of Cases	Percent
Male	53	80.30
Female	13	19.70
Total	66	100.00

**Table-2: Age Distribution in Cases**

Age in Years	No. of cases	Percent
<40	06	9.09
40-45	15	22.73
46-50	13	19.70
51-55	11	16.67
56-60	11	16.67
61-65	04	6.06
>65	06	9.09
Total	66	100.00

**Table-3A: Comparison of Plasma Ascorbic Acid in Cases & Control**

	Cases (n=66)	Controls (n=66)	'Z' value	'p' value	Significance
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.37 $\pm$ 0.064	0.58 $\pm$ 0.16	10.50	<0.01	Significant
Range	0.28-0.54	0.38-0.91			

**Table-3B: Comparison of Plasma Ascorbic Acid in Cases & Control**

Plasma ascorbic acid	Cases (%) (n=66)	Controls (%) (n=66)	Total (%) n=132	'p' value
Low ( $\leq 0.4$ mg/dL)	50 (75.76%)	05 (7.58%)	55 (41.67%)	$\chi^2=63.12$ , OR=38.12
Normal ( $>0.4$ mg/dL)	16 (24.24%)	61 (92.42%)	77 (58.33%)	P<0.001

**Table-4: Comparison of Conventional Risk factors for AMI (confounding variables) in cases and controls (Univariate analysis)**

Variable	Cases (n=66)	Controls (n=66)	Total n=132	'p' value
Mean age in years ( $\pm$ SD)	51.76 $\pm$ 11.1	51.23 $\pm$ 12.6		Z=0.26 p>0.05 NS
Range	25-81	25-78		
Diabetes mellitus +	9	13	22	$\chi^2=0.87$
–	57	53	110	p>0.05 NS
Mean serum cholesterol mg/dL ( $\pm$ SD)	192.49 $\pm$ 34.44	177.06 $\pm$ 20.86		Z=3.11 P<0.01
Range	150-287	150-233		
Total cholesterol* $\geq$ 200	29	13	42	$\chi^2=8.94$
<200	37	53	90	P<0.01
Mean serum triglyceride mg/dL ( $\pm$ SD)	137.59 $\pm$ 50.65	110.95 $\pm$ 38.09		Z=3.41
Range	65-350	56-243		P<0.001 HS
S-triglyceride* $\geq$ 150	22	12	34	$\chi^2=3.96$
<150	44	54	98	P<0.05
Mean serum HDL* mg/dL	39.58 $\pm$ 3.08	43.32 $\pm$ 3.98		Z=6.04
Range	34-52	34-53		P<0.001
Serum HDL <40	34	11	45	$\chi^2=17.84$
$\geq$ 40	32	55	87	P<0.001
Mean serum LDL Mg/dL ( $\pm$ SD)	112.76 $\pm$ 31.83	111.77 $\pm$ 19.22		Z=0.21
Range	43-222.8	63.8-150		p>0.05 NS
Serum LDL* $\geq$ 130	27	10	37	$\chi^2=10.85$
<130	39	56	95	P<0.01
Mean BMI in Kg/m <sup>2</sup> ( $\pm$ SD)	24.90 $\pm$ 3.53	23.98 $\pm$ 3.01		Z=2.82
Range	17.3-31.4	19-32.2		P<0.01
BMI Kg/m <sup>2</sup> $\geq$ 25	38	29	67	$\chi^2=2.45$
<25	28	37	65	P>0.05
Smoking* +	31	17	48	$\chi^2=6.42$
–	35	49	84	P<0.05
H/o Alcohol intake +	12	9	21	$\chi^2=0.51$
–	54	57	111	p>0.05
Family H/o CAD +	7	10	17	$\chi^2=0.61$
–	59	56	115	p>0.05

+: Present, -: Absent

\*: Indicates significant association (p<0.01)

**Table-5: Showing Odds Ratio of various risk factors with Acute Myocardial Infarction**

(Plasma Ascorbic Acid, Serum Cholesterol, Serum Triglycerides, Serum HDL, Serum LDL, BMI taken as categorical variables)

Risk factors	Odd's ratio
Low plasma ascorbic acid ( $\leq$ 0.4 mg/dL)	38.125
Diabetes mellitus	0.64
Hypercholesterolemia ( $\geq$ 200 mg/dL)	3.19
Increased triglyceride ( $\geq$ 150 mg/dL)	2.25
Decreased HDL (<40 mg/dL)	5.31
Increased LDL ( $\geq$ 130 mg/dL)	3.88
Increased BMI ( $\geq$ 25 Kg/m <sup>2</sup> )	1.73
Smoking	2.55
Alcohol	1.41
Family history of coronary artery disease	0.66

**Table-6: Relationship of Plasma Ascorbic Acid with age**

Age category (years)	No. of Cases	Percentage	Mean plasma ascorbic acid (mg/dL)
< 40	12		0.46 $\pm$ 0.15
40 – 50	57		0.458 $\pm$ 0.15
51 – 60	43		0.52 $\pm$ 0.18
> 60	20		0.47 $\pm$ 0.13

Correlation coefficient between age and plasma ascorbic acid r=0.10.

**Table-7: Relationship of Plasma Ascorbic Acid with Sex**

	Males (n=106)	Females (n=26)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.47 $\pm$ 0.15	0.518 $\pm$ 0.18	Z=1.26
Range	0.28 – 0.91	0.28 – 0.83	>0.05 insignificant

**Table-8: Relationship of Plasma Ascorbic Acid with Diabetes Mellitus**

	Diabetics (n=22)	Non-diabetics (n=110)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.43 $\pm$ 0.099	0.49 $\pm$ 0.17	Z=2.24
Range	0.33-0.74	0.28-0.91	<0.05 significant

**Table-9: Relationship of Plasma Ascorbic Acid with Serum Cholesterol**

	Cholesterol $\geq 200$ mg/dL (n=42)	Cholesterol $< 200$ (n=90)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.43 $\pm$ 0.14	0.49 $\pm$ 0.17	Z=2.14
Range	0.28-0.91	0.28-0.87	<0.05 significant

**Table-10: Relationship of Plasma Ascorbic Acid with Serum Triglyceride**

	Triglycerides $\geq 150$ (n=34)	Triglycerides $< 150$ (n=98)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.41 $\pm$ 0.09	0.503 $\pm$ 0.17	Z=4.03
Range	0.28-0.74	0.28-0.91	<0.001

**Table-11: Relationship of Plasma Ascorbic Acid with Serum HDL**

	Serum HDL $\geq 40$ (n=87)	Serum HDL $< 40$ (n=45)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.52 $\pm$ 0.17	0.40 $\pm$ 0.12	Z=4.70
Range	0.28-0.91	0.28-0.87	P<0.001

**Table-12: Relationship of Plasma Ascorbic Acid with Serum LDL**

	Serum LDL $\geq 130$ (n=37)	Serum LDL $< 130$ (n=95)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.42 $\pm$ 0.12	0.50 $\pm$ 0.17	Z=3.04
Range	0.28-0.91	0.28-0.87	P<0.01 significant

**Table-13: Relationship of Plasma Ascorbic Acid with BMI**

	BMI $\geq 25$ (n=67)	BMI $< 25$ (n=65)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.44 $\pm$ 0.12	0.52 $\pm$ 0.18	Z=2.99
Range	0.28-0.83	0.28-0.91	P<0.01

**Table-14: Relationship of Plasma Ascorbic Acid with Smoking**

	Smokers (n=48)	Non-smokers (n=84)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.39 $\pm$ 0.07	0.53 $\pm$ 0.18	Z=6.48
Range	0.28-0.60	0.28-0.91	P<0.001

**Table-15: Relationship of Plasma Ascorbic Acid with Alcohol Intake**

	H/o Alcohol intake (n=21)	No H/o Alcohol intake (n=111)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.43 $\pm$ 0.10	0.49 $\pm$ 0.17	Z=2.21
Range	0.33-0.66	0.28-0.91	P<0.05 Significant

**Table-16: relationship of plasma ascorbic acid with family history of Coronary Artery Disease**

	Family H/o of CAD (n=17)	No Family H/o CAD (n=115)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.53 $\pm$ 0.19	0.47 $\pm$ 0.16	Z=1.24
Range	0.28-0.83	0.28-0.91	p>0.05 not significant

**Table-17: Relationship of Plasma Ascorbic Acid in cases and controls without any conventional risk factors**

	Cases (n=9)	Controls (n=17)	'p' value
Mean plasma ascorbic acid in mg/dL $\pm$ SD	0.36 $\pm$ 0.07	0.72 $\pm$ 0.12	Z=9.65
Range	0.28-0.54	0.54-0.91	P<0.001

**Table-18: Distribution of Plasma Ascorbic Acid in Different Sites of Infarction**

Site of infarction	N=66 (%)	Plasma Ascorbic Acid (mg/dL)	
		Mean $\pm$ SD	Range
AWMI	37 (56.06)	0.35 $\pm$ 0.05	0.28-0.48
IWMI	27 (40.91)	0.41 $\pm$ 0.07	0.28-0.54
AWMI+IWMI	02 (3.03)	0.31 $\pm$ 0.04	0.28-0.3
PWMI	--	--	--

AWMI-Anterior wall myocardial infarction., IWMI-Inferior wall myocardial infarction.  
PWMI-Posterior wall myocardial infarction.

Table-19: Correlation of Plasma Ascorbic Acid to Mortality in Patients with Acute Myocardial Infarction

		Mean plasma ascorbic acid mg/dL	No. of cases	No. of deaths	'p' value	Relative risk	Attributable risk
Plasma AA on admission (mg/dL)	≤0.40	--	50	7	$\chi^2=0.68$		
	>0.40	--	16	1	$P>0.05$	2.20	57.14%
	Total	0.37±0.06	66	8			
Plasma AA at 1 week (mg/dL)	≤0.40	--	23	5	$\chi^2=3.36$		
	>0.40	--	35	2	$p>0.05$	3.86	72.72%
	Total	0.44±0.08	58	7			
Plasma AA at 3 weeks* (mg/dL)	≤0.40	--	8	3	$\chi^2=10.69$		
	>0.40	--	40	1	$P<0.01$	15.20	93.42%
	Total	0.50±0.09	48	4			

\* Three patients alive but did not turned up for follow-up.

## DISCUSSION

The existence and importance of free radicals and its role in atherogenesis<sup>16,17</sup>, CAD and in mediating tissue injury during myocardial ischemia and in particular during the phase of myocardial re-oxygenation has been widely accepted. Since the first antioxidative defense line of plasma lipoproteins consists actually of vitamin-C<sup>2-5</sup>, its deficiency may become one of the most important events in relation to CAD. Moreover studies have shown that vitamin-C has sparing effect over vitamin-E and B vitamins such as folate (which normalizes moderate homocysteinemia).<sup>6</sup> Vitamin-C attenuates vasomotor dysfunction in epicardial coronary arteries<sup>41</sup>. Similarly high plasma vitamin-C is associated with high plasma HDL and HDL<sub>2</sub>.<sup>19</sup> Thus, low levels of plasma ascorbic acid may play a pathogenic role in coronary artery disease. The current understanding of events associated with myocardial ischemia suggest that within the ischemic myocardial region or area at risk, there is a population of cells that are reversibly injured and that reperfusion during a specified period (less than 3 hours) of time is capable of restoring the majority of jeopardized cells to normal state but the act of reperfusion itself leads to the reintroduction of molecular oxygen and the circulating elements of blood that will be associated with an explosive and self-limited destruction of some of the myocardial cells in the area at risk.<sup>20</sup> Ascorbic acid effectively reduces myocardial necrosis after ischemia, and hence also may have a part in improving reperfusion of ischemic myocardium.<sup>21</sup>

As the plasma ascorbic acid levels can be increased by supplementation, and the plasma levels are more conclusive indicators of the antioxidant status<sup>22</sup>, we carried out the present study. Our study showed that mean plasma ascorbic acid in cases of AMI was significantly lower than that of controls. Mean plasma ascorbic acid in cases was 0.37±0.064 mg/dL while that in controls was 0.58±0.16 mg/dL. This study diabetes was less common and was not significantly associated with acute myocardial infarction while smokers, serum cholesterol, low HDL, high LDL, BMI and smoking were significantly associated with acute myocardial infarction in univariate analysis. Significant difference between plasma ascorbic acid of cases and controls persisted even after controlling for all the confounding variables.

The present study also analyzed and tried to find the association between plasma ascorbic acid and the conventional risk factors for coronary artery disease. We found that men have lower plasma ascorbic acid as compared to females. The mean plasma ascorbic acid in males was 0.47±0.15 mg/dL and in females it was 0.518±0.18 mg/dL ( $p>0.05$ ). The difference was insignificant. Many

investigations have observed significantly lower plasma ascorbic acid in men compared to females.

Our study showed that mean plasma ascorbic acid of diabetics was lower than that of non-diabetics. Mean plasma ascorbic acid in patients with diabetes was 0.43±0.09 mg/dL, which was significantly lower than that of non-diabetics subjects (0.49±0.17 mg/dL) ( $p<0.05$ ). And also showed significantly lower plasma ascorbic acid in subjects having total serum cholesterol ≥200 mg/dL (0.43±0.14 mg/dL) than subjects having total serum cholesterol <200 mg/dL (0.49±0.17 mg/dL) ( $p<0.05$ ). In this study lower plasma ascorbic acid in subjects having serum triglycerides ≥150 mg/dL (0.41±0.09 mg/dL) than subjects having serum triglycerides <150 mg/dL (0.50±0.17 mg/dL) ( $p<0.005$ ).

Our study showed lower plasma ascorbic acid in subjects having serum LDL ≥130 mg/dL (0.42±0.12 mg/dL), serum LDL <130 mg/dL (0.50±0.17 mg/dL) ( $p<0.01$ ). In the present study smokers were found to have significantly lower ascorbic acid (0.394±0.07 mg/dL) as compared to non-smokers (0.53±0.18 mg/dL) ( $p<0.001$ ). This finding is consistent with a number of studies.

In our study alcoholics had significantly lower plasma ascorbic acid as compared with non-alcoholics. The present study also analyzed and tried to find the association between plasma ascorbic acid and the short-term mortality after acute myocardial infarction. In our study, patients with plasma ascorbic acid ≤0.4 mg/dL had a higher mortality as compared to patients having >0.4 mg/dL. The mortality in patients with plasma ascorbic acid ≤0.4 mg/dL compared to >0.4 mg/dL were 7 and 1, 5 and 2, 3 and 1 at admission, after one week and after three weeks respectively.

The association between plasma ascorbic acid after three weeks and mortality was significant when compared in patients with ≤0.40 mg/dL and >0.40 mg/dL ( $p<0.01$ ) indicating that mortality was significant in patients in whom plasma ascorbic acid remained persistently low (relative risk 15.2 and attributable risk 93.42%). The mean plasma ascorbic acid in patients on admission were 0.37±0.06 mg/dL and the levels were found to increase on follow-up after one week i.e., 0.44±0.08 mg/dL and after three weeks i.e., 0.50±0.09 mg/dL.

Singh RB<sup>30</sup> et al concluded in a recent study that consumption of an antioxidant-rich diet may reduce the plasma levels of lipid peroxide and cardiac enzyme and increase plasma level of ascorbic acid. Antioxidant rich foods may reduce myocardial necrosis and reperfusion, injury induced by the oxygen free radicals.

Some of the studies have not shown any such association. Gale CR found that dietary intake or plasma ascorbic acid



concentration is strongly related to subsequent risk of death from stroke but not from coronary heart disease.

Thus, the results of our study are consistent with and support most other studies. It can be concluded from the above discussions that oxidative stress occurs in patients with acute myocardial infarction, and it can be hypothesized that alteration in the plasma ascorbic acid levels may be one of the mechanisms by which most of the conventional risk factors of coronary artery disease acts and also predicts cardiac mortality.

## CONCLUSIONS


The plasma ascorbic acid levels were significantly lower in cases of acute myocardial infarction than age group and sex matched healthy controls. The plasma ascorbic acid levels were significantly lower in males, diabetics, hypercholesterolemia (total cholesterol  $\geq 200$  mg/dL), hypertriglyceridemia ( $\geq 150$  mg/dL), decreased HDL ( $< 40$  mg/dL), increased LDL ( $\geq 130$  mg/dL), smoking, patients with BMI  $\geq 25$  Kg/m<sup>2</sup> and history of alcohol intake.

Plasma ascorbic acid  $\leq 0.40$  mg/dL was associated with increased mortality as compared to  $> 0.40$  mg/dL. Oxidative stress may be one of the important factor leading to reduced plasma ascorbic acid levels in patients with risk factors for coronary artery disease and acute myocardial infarction subsequently leading to death. Administration of ascorbic acid may help reduce oxidative stress in patients with acute myocardial infarction and associated risk factors and hence may help reduce mortality.

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