

Plasma Free Fatty Acid Concentrations as a Marker for Acute Myocardial Infarction

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ABSTRACT

Background: Acute myocardial infarction carries a high mortality among cardiac patients. The discovery of the fact that certain enzymes like CPK, LDH liberated into circulation following necrosis of the myocardial cells came as boon for physicians and patients. There has been a constant search of different parameters for the diagnosis and management of Coronary Artery Diseases (CAD).

Aim: The present study was undertaken to investigate a possible relation between the changes in plasma free fatty acid (FFA) concentration and acute myocardial infarction.

Material and Methods: Fifty cases (25 males and 25 females) of acute myocardial infarction were selected for the present study. All the patients were in the age group of 40-70 years. For the control group fifty (25 male and 25 female) subjects of same age group were selected from patient's relatives and friends.

Plasma free fatty acid concentration was estimated by Titrametric method of Trout et al., (1960), a modified version of Dole (1956).

Statistical Analysis: The statistical analysis of the data of the present study was done by using SPSS, version 14.0.1 was used.

Results: Our study showed a significant increase in plasma FFA in the first 24 hours of acute myocardial infarction with subsequent normalisation on the 7th day. The difference between the first and the seventh day was statistically significant.

Conclusion: The FFA were found raised in cases of acute myocardial infarction. On the basis of present study, it is worth to say that estimation of serum free fatty acid should be done routinely at the earliest opportunity in all cases of acute myocardial infarction.

Key words: Plasma free fatty acid, FFA, Acute myocardial infarction, Coronary artery disease, Marker

INTRODUCTION

The present day scientific & industrialized world has provided means for a better quality of life but unfortunately such facility has also led people to live a sedentary life style which is one of the most important causative factors for the development of coronary artery diseases.

Acute myocardial infarction is accompanied by profound metabolic changes. Circulating free fatty acids (FFAs) mainly originate from lipolysis in the adipose tissue; they contribute to insulin resistance and are elevated in obesity and type 2 diabetes [1-4]. Recent studies [5,6] suggested that FFAs also exert negative effects on the vessel wall by triggering endothelial apoptosis and impairing endothelium-dependent vasodilation [7,8]. The involvement of FFAs in atherosclerosis is supported by observations of an increased risk for cardiovascular disease associated with high levels of FFAs [9,10]. Oxidation of FFAs, which requires relatively more oxygen than glucose use, is the main energy source for the myocardium under physiological conditions [11]. However, elevation of FFAs as observed in myocardial ischemia has been shown to increase the ischemic damage of the myocardium and to be proarrhythmic [11-14].

The fact that free fatty acids are valuable related to myocardial ischemia is supported by the fact that a particular form of fatty acid can be used for evaluatory purpose of knowing viability of myocardium in chronically ischemic myocardium.

Recently, attention has been paid to the increased plasma free fatty acids (FFA) in patients with acute myocardial infarction [15-17]. The observations on the higher incidence of cardiac arrhythmias and sudden death in patients with myocardial infarction exhibiting strikingly raised serum FFA are particularly noteworthy [15-17]. Other authors have not found a relation between the initial rise of plasma FFA and the subsequent clinical course of acute myocardial infarction; however, they have noted a repeated increase of plasma FFA coinciding with the onset of complications [18]. High FFAs

were predictive for increased mortality in type 2 diabetics [19] and subjects with acute myocardial infarction [11,20], but to our knowledge there are very less data available showing relation between FFA and acute myocardial infarction. The present study was undertaken to investigate a possible relation between the increases of plasma FFA in acute myocardial infarction.

MATERIAL AND METHODS

The present study was conducted on patients of coronary artery diseases admitted in the Department of Medicine, Patna Medical College & Hospital, Patna, India. The duration of the study was 1 year. The study was approved by institutional ethics committee. Written informed consents were obtained from the subjects. The purpose of the study and the rationale behind it were explained to them.

The study was performed on total 100 subjects which included fifty cases (25 male and 25 female) of acute myocardial infarction and fifty healthy controls (25 male and 25 female). All the subjects were in the age group of 40-70 years. The control group subjects were selected among the patient's relative and friends in order to maintain the similarity in respect of socio-economic status.

Exclusion criteria: Subjects who suffered from diabetes mellitus, hepatic disorder, or taking drugs like corticosteroids, heparin were excluded from the control group.

METHODOLOGY

The cases of coronary artery disease were diagnosed on the basis of clinical history, suggestive echocardiographic changes, and significantly raised serum level of enzymes such as S.G.O.T, CPK & LDH. All the cases of study group were given identical diets consisting of low salt, low fat and high carbohydrate diet.

In case of study group, the serum free fatty acid level were estimated at the time of admission in the hospital (within six hour

of onset of clinical symptoms), after 24 hours, after 48 hrs and on the 7th day of hospitalization.

Lab analysis: Plasma free fatty acid concentration was estimated by Titrametric method of Trout et al., (1960), a modified version of Dole (1956).

STATISTICAL ANALYSIS

The statistical analysis of the data of the present study was done by using SPSS, version 14.0.1 was used. Since the study was interventional in nature, paired t-test was used to find out the statistical significance of the results. A t-value of ≥ 1.96 indicated that the results were significant statistically and a t-value which was less than 1.96 indicated that the results were not significant statistically.

OBSERVATIONS AND RESULTS

The difference in plasma FFA level in relation to sex was insignificant. This table shows the serum FFA level in cases of acute myocardial infarction at different time intervals. Serum FFA was highest at the time of admission and came down to normal level by the end of 48 hours [Table/Fig-1] and [Table/Fig-2].

Sex	No. of cases	Serum FFA levels in micro eq./L				t- value
		Range	Mean	SD	SEM	
Male	25	454-602	525.36	50.28	10.03	0.337 NS
Female	25	450-610	519.88	61.50	12.3	

[Table/Fig-1]: Serum free fatty acid levels in control group

Time interval of estimation	Sex	No. of cases	Serum FFA level in micro-eq/L			
			Range	Mean	S.D.	S.E.M
At the time of admission	Male	25	730-1360	1073.04	273.74	46.94
	Female	25	760-1380	1133.6	200.71	40.14
After 24 hours	Male	25	678-1090	853.09	151.06	32.25
	Female	25	700-1120	908.0	146.97	31.33
After 48 hours	Male	25	454-620	532.70	59.54	13.31
	Female	25	470-620	517.68	49.77	11.42
On 7th day	Male	25	454-616	536.22	63.69	15.01
	Female	25	460-618	512.11	54.53	12.51

[Table/Fig-2]: Serum FFA level in cases of stable angina pectoris at different time intervals

Time interval	Group	Mean serum FFA level	S.D.	't' value
At the time of admission	Control (male)	525.36	50.28	11.77
	Case (male)	1073.04	234.34	
	Control (female)	519.88	61.5	14.33
	Case (female)	1133.6	200.71	
After 24 hours	Control (male)	525.36	50.28	9.88
	Case (male)	853.09	50.28	
	Control (female)	519.88	61.50	11.81
	Case (female)	908	146.31	
After 48 hours	Control (male)	525.36	61.50	0.7
	Case (male)	532.70	59.54	
	Control (female)	519.88	50.28	0.49
	Case (female)	517.68	49.77	
on 7th day	Control (male)	525.36	61.50	0.83
	Case (male)	536.22	63.69	
	Control (Female)	519.88	50.28	0.82
	Case (female)	512.11	54.53	

[Table/Fig-3]: Table showing comparison of serum FFA level of control and cases of stable angina at different time interval

[Table/Fig-3] shows that serum FFA was highly elevated in Acute myocardial infarction. The rise was statistically significant. The rise was highest initially at the time of appearance of clinical symptoms

and it remain statistically significant till 48 hours, after 48 hours, it came to normal level.

DISCUSSION

In accordance with the reports of other authors (Gupta et al., Kurien and Oliver, Oliver, Rutenberg et al., our study showed a significant increase in plasma FFA in the first 24 hours of acute myocardial infarction with subsequent normalization on the 7th day [15-17]. The difference between the first and the seventh day was statistically highly significant. The highly significant correlation between the FFA, found in the acute stage of myocardial infarction, and the parallel decline of these values in time deserve particular attention.

The mechanism and the significance of the increase of plasma FFA concentration in acute myocardial infarction has been a matter of interest, especially so in connection with the role of FFA in the myocardial metabolism. The increased circulating plasma FFA promotes augmented myocardial oxygen consumption which may be particularly harmful for ischaemic myocardium. Kurien, Yates, and Oliver reported a relation between the post-heparin increase of plasma FFA and the incidence of cardiac arrhythmias in dogs with acute coronary artery occlusion [21].

It was also shown that in experimental conditions the FFA diminish the contractility of the hypoxic heart muscle [22]. Several recent clinical observations suggest that free fatty acids may also play an important role in the clinical course of acute myocardial infarction.

In the present study we were particularly concerned with the relation between plasma FFA concentrations in acute myocardial infarction.

Various possibilities have been put forward by different workers as to the cause of rise in plasma free fatty acid in cases of coronary artery diseases. One explanation is that in cases of coronary artery diseases, there is increased sympathetic drive, reason could be pain, anxiety, dreadfulness & stress.

It is known that noradrenaline is one of the most potent factors releasing FFA into the blood through the activation of tissue lipase and the subsequent hydrolysis of triglycerides. The highly significant increased levels of FFA which we have been able to show in the acute stage of myocardial infarction, as well as the subsequent parallel decrease of these levels, justifies the suggestion that the raised plasma FFA in acute myocardial infarction may be related to the increased noradrenaline production caused by the augmented activity of the sympatho-adrenal system in response to the infarction.

On the basis of above study it is worth to say that estimation of serum free fatty acid should be done routinely at the earliest opportunity in all cases of acute myocardial infarction. The cases with high serum FFA levels should be closely monitored for early and timely detection of acute myocardial infarction so that proper treatment can be provided to the patients.

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Date of Submission: **Sep 18, 2013**
Date of Peer Review: **Sep 23, 2013**
Date of Acceptance: **Sep 27, 2013**
Date of Online Ahead of Print: **Oct 10, 2013**
Date of Publishing: **Nov 10, 2013**

FINANCIAL OR OTHER COMPETING INTERESTS: None.