

SERUM ESTRADIOL AND TESTOSTERONE LEVELS FOLLOWING ACUTE MYOCARDIAL INFARCTION IN MEN

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Abstract : The present study examined serum testosterone and estradiol levels on the day of admission, 5th and on 10th day following acute myocardial infarction in men. Controls were matched for age and body mass index. Testosterone levels were low on the day of admission and remained statistically unchanged on 5th and 10th day as compared to controls. On the contrary, estradiol levels were significantly higher on the day of admission. A significant decrease in estradiol concentration in comparison to the levels on the day of admission was observed on 10th day post myocardial infarction. However, the estradiol levels on 10th day were significantly higher than control subjects. The results of the study suggest that in acute myocardial infarction, hyperestrogenemia is associated with hypotestosteronemia.

Key words : infraction estradiol testosterone

INTRODUCTION

It is well known that men suffer from higher incidence of ischemic heart diseases (IHD) than women and later is exposed to greater risk from coronary artery diseases after menopause (1-3). Results of clinical trials with estrogens following acute myocardial infarction have reported increase in mortality from IHD in men (4). Further, increase in feminization has been shown in the young male survivors of myocardial infarction (5). The above results suggest that estrogens might be an independent risk factor for myocardial infarction. Either low or normal testosterone levels with hyperestrogenemia in male survivors of myocardial infarction has been demonstrated (5-10). In the above cited

studies, hormonal levels were measured months after acute myocardial infarction. Therefore, in the present study testosterone and estradiol levels were measured at the time of admission and thereafter to elucidate the relationship between sex hormones in the acute phase of myocardial infarction.

METHODS

Thirty three male patients (age 56 ± 10.4) admitted in intensive care unit with acute myocardial infarction (AMI) constituted the study group. Equal number of normal healthy individuals (age 52 ± 8.60) were included in the control group. Patients and controls with any previous history of hypertension, diabetes, cardio-respiratory diseases, angina, myocardial infarction and

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smoking were excluded from the study. Diagnosis of AMI was made on the basis of presented history of ischemic pain, electrocardiographic changes and elevated creatine kinase levels. All patients received 350 mg of aspirin on admission and similar dose thereafter. Heparin was infused immediately with an intravenous dose of 5000 IU followed by 10,000 IU/day. Along with aspirin and heparin, 30 mg of isosorbide dinitrate was used in three divided doses. None of the patients received any thrombolytic therapy. Body mass index in control and patients was calculated by weight (kg)/height (m)².

Hormonal estimations: Venous blood samples were withdrawn between 7 to 9 AM for the measurement of estradiol and testosterone on the day of admission (0 day), 5th and on 10th day post acute myocardial infarction. Serum was stored at -30°C until assay. All estimations were carried out in single batch in duplicate with 'time resolved fluorimmunoassay' delfia kits (Pharmacia, Finland) to avoid interassay variations.

Data analysis: Values were presented as mean ± SEM. Hormonal levels on different

days following AMI were analysed by analysis of variance. Differences between means were analysed by test for least significant difference after taking 95% as level of confidence. Unpaired 't' test was used to compare age and body mass index between control and patients. Statistical significance was considered as $P < 0.05$.

RESULTS

TABLE I : Age and body mass index in controls and patients.

	Controls	Patients	P value
Age(years)	52±8.6	56±10.4	NS
Body mass index (BMI)	26.42±0.8	25.7±0.6	NS

n=33 in each group
NS - not significant

Table I shows that age and body mass index in patients and controls were not significantly different. Sex hormone levels following AMI is presented in Table II. It is evident from results that AMI patients on the day of admission (0 day) had significantly higher levels of estradiol and low levels of testosterone in comparison to controls. On the 5th day post AMI, estradiol and testosterone concentration showed no

TABLE II : Serum estradiol and testosterone levels following acute myocardial infarction

Hormones	Control	Patients		
		Days	Post	AMI
		0	5	10
Estradiol (pg/ml)	18.14±0.57	49.36±0.36*	48.70±0.24 ^{NS}	35.10±0.30**
Testosterone (ng/ml)	7.71±0.16	3.8±0.25*	3.84±0.16 ^{NS}	3.72±0.20 ^{NS}

Values are mean + SEM, n=33 in each group

*P<0.001 versus control,

**P<0.001 versus 5th, 0 day

NS- not significant versus 0 day.

significant changes whereas estradiol levels on 10th day were significantly less than 5th day. A significant increase in estradiol level in comparison to control was observed on 10th day post AMI (35.10 ± 0.30 vs 18.14 ± 0.57 , $P < 0.001$). On the contrary, the levels of testosterone on 10th day was not statistically different than control.

DISCUSSION

The results of the present study show that in confirmed acute myocardial infarction hyperestrogenemia is associated with hypotestosteronemia. It has been reported that obesity, drugs and cigarette smoking can cause hormonal imbalance in men with coronary artery diseases (11, 12). In our study, the patients were matched with control for age and relative body weight. Therefore, observed hormonal changes cannot be ascribed to either obesity or to smoking.

Adrenergic stimulation plays an important role in different cardiac diseases (13). Estradiol can increase synthesis and synaptic activities of adrenergic neurotransmitters (14, 15). Adrenergic receptor blockers have been shown to decrease the incidence of ventricular arrhythmias, rate of reinfarction and sudden death in the survivors of acute myocardial infarction (16, 17). Further, a significant correlation of estradiol with serum creatine

phosphokinase has been reported (18). The results of the above studies suggest that estrogens can act as adrenergic stimulant and patients with high estradiol levels at the time of myocardial infarction might be predisposed to higher risk of ventricular fibrillation and sudden death. Whether elevated estradiol levels in our study preceded acute myocardial infarction, is not known. No significant association between atherosclerotic coronary artery disease and free estradiol level has been reported (19).

In the present study, significantly low testosterone and high estradiol levels in the acute phase of myocardial infarction suggests enhanced conversion of testosterone into estradiol. Addition of noradrenaline to *in vitro* sertoli cell culture results in increased aromatization of testosterone to estradiol (20). Further, elevated noradrenaline levels in the patients with acute myocardial infarction has been reported (21). Therefore, the observed changes in testosterone and estradiol levels were probably due to increase in the process of aromatization.

In conclusion, the present study show that attack of acute myocardial infarction in men can alter the sex hormone levels. Whether, the hormonal change show enhanced aromatization of testosterone to estradiol or were due to some other unknown mechanism, requires further study.

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