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RESEARCH ARTICLE

MALONDIALDEHYDE LEVEL IN PATIENTS WITH MYOCARDIAL INFARCTION (A CASE CONTROL STUDY OF 30 PATIENTS)

Pooja Sorathia, *Ramesh Pradhan, Rosy Lekharu and Kiran Saxena

Department of Biochemistry, GCS Medical College, Ahmedabad-25, Gujarat, India

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ABSTRACT

Coronary artery disease (CAD) is the leading cause of death worldwide. Many recent studies have shown that anomalies in circulating lipid levels are a predisposing factor for coronary heart disease. Oxidative stress has been implicated in the patho-physiology of CAD. Lipid peroxidation measured as malondialdehyde (MDA) is an indication of the extent of free radical mediated tissue damage. The difference in the malondialdehyde levels between the case and control groups was highly significant (p value < 0.0001) suggesting Malondialdehyde as an important risk factor for myocardial infarction. The difference in Malondialdehyde levels observed between patients with LDL-C > 100 mg % and those with LDL-C < 100 mg % was statistically extremely significant (p value < 0.0001).

INTRODUCTION

Coronary artery disease is the leading cause of death worldwide. Many recent studies have shown that anomalies in circulating lipid levels are a predisposing factor for coronary heart disease. Oxidative stress has been implicated in the patho-physiology of a many disease or disorders which are initiated and /or exacerbated by pro-oxidants such as various drugs including alcohol and food additives (Halliwell 1989). Lipid peroxidation–measured as malondialdehyde-is an indication of the extent of free radical mediated tissue damage. Many recent studies have shown that free radical mediated damage and lipid peroxidation to be elevated in patients of coronary heart disease. Hence as a case-control study, this study concentrated at identifying the abnormalities in circulating levels of lipids and Malondialdehyde (MDA), in patients of coronary heart disease to reveal its role in myocardial infarction for diagnostic and prognostic purpose. Numerous risk factors have been identified and many of them are modifiable in the form of smoking, physical inactivity, diet, obesity, Dyslipidemia, hypertension. Maintaining the lipid profile within the normal range has been seen to reduce coronary events and mortality (James Shepherd *et al.,* 1995). Increased production of these free radicals will render the lipids susceptible to lipid peroxidation (Cheesman and Slater 1993). Lipid peroxidation is the result of free radical mediated damage to the lipid molecules (Vasudevan *et al.,* 2013).

Corresponding author: Ramesh Pradhan
Department of Biochemistry, GCS Medical College, Ahmedabad-25, Gujarat, India.

Malondialdehyde (MDA) is a breakdown product of peroxidation of long chain fatty acids which accumulates when lipid peroxidation increases (Ceconi *et al.,* 1992). The effects of lipid peroxides i.e. endothelial cell damage, uncontrolled lipid uptake, decreased prostaglandin synthesis and associated thrombogenecity are strongly implicated in the pathogenesis of atherosclerosis (Onvural *et al.,* 1998). Inadequate removal of ROS may cause cell damage by attacking membrane lipids, proteins and inactivating enzymes thus mediating several forms of tissue damage (Datla *et al.,* 2000). MDA causes toxic stress in cells and form covalent protein adducts and is used as a biomarker to measure the level of oxidative stress in an organism (Moore and Roberts 1998). MDA can also be generated during prostaglandin biosynthesis in cell (Hancock *et al.,* 1999).

MATERIALS AND METHODS

Study group included diagnosed patients of myocardial infarction in the age group of 30 – 60 years admitted at SSG Hospital Vadodara during the period between January 2009 to August 2009. Control group included persons between similar age group without ischemic heart disease. Inclusion criteria included age group between 30 – 60 years, severe chest pain lasting >30 minutes and not responding to sublingual nitroglycerin significantly and with specific ECG changes. A 12 hr fasting blood sample 5 ml blood was collected in a plain bulb, centrifuged within 5 minutes, and analyzed for Serum MDA (Thiobarbiturate method) (Beuge and Aust 1978), Total cholesterol (CHOD-POD), serum triacylglycerol (GPO), HDL-

Cholesterol (PTA, CHOD-POD). LDL-Cholesterol and VLDL-Cholesterol was calculated by Friedewald's formula (Richard *et al.,* 2006).

RESULTS

Out of 30 patients with myocardial infarction, 10 were females and rests were males. Chest pain was the most common presenting symptom followed by Dyspnea (63.33%) & perspiration (56.66%). Smoking (80 %) was the commonest risk factor associated with myocardial infarction in this study followed by, systemic arterial hypertension (20 %), positive family history for ischemic heart disease (3.33 %) and diabetes mellitus (3.33 %).

Table 1: Compared between the case and control group, the difference in total cholesterol levels was not statistically significant (p value = 0.259) while for HDL–C (p 0.0033) and LDL-C (p 0.0204) were significant. It suggested that low HDL –C and high LDL-C in case group were an important risk factor for coronary atherosclerosis .The difference in the serum triglyceride levels between the case and control groups was significant at 95 % confidence interval (p value = 0.0470). The difference in the malondialdehyde levels between the case and control groups was highly significant (p value < 0.0001) suggesting Malondialdehyde as an important risk factor for myocardial infarction.

Table 2: The difference in Malondialdehyde levels observed between male and female groups was not statistically significant. (p value = 0.3629). The difference in Malondialdehyde levels observed between patients with LDL-C $\geq$ 100 mg % and those with LDL-C < 100 mg % was statistically extremely significant (p value < 0.0001). Hence, in this study, the low levels of LDL-C did protect the patients against the Malondialdehyde induced coronary artery disease. The difference in Malondialdehyde levels observed between patients with HDL-C $\geq$ 40 mg % and those with HDL-C < 40 mg % was not statistically significant (p value = 0.9339). The difference in Malondialdehyde levels observed between patients with S. Cholesterol $\geq$ 200 mg % and those patients with S. Cholesterol < 200 mg %was statistically not significant (p value = 0.9917).

modifiable risk factor in patients of myocardial infarction. Bennet *et al.* (2008) have reported in their study that 64% patients had smoking history (Bennet *et al.,* 2008). Hypercholesterolemia is an important risk factor for atherosclerotic coronary disease in all age groups. Arun Kumar *et al.* (2009) have reported high cholesterol, LDL-C, low HDL-C in case compared to controls with significant p values. However the mean total cholesterol levels in our study was < 200 mg % in both case and control group (p value= 0.259) and thus total cholesterol was not a predominant risk factor for myocardial infarction in our study.

Lijia Chen *et al.* (1995) reported the mean HDL cholesterol and the SD observed in the cases and control group were 0.9 ± 0.2 mmol/l and 1.1 ± 0.4 mmol/l respectively. In this study the difference in the levels of HDL –C between the case and control groups was statistically significant (p value 0.0033) suggesting that low HDL – C in case group was an important risk factor for myocardial infarction. The mean LDL Cholesterol level of the control subjects compared with AMI (119.4 mg/dl) was significantly greater than that of subjects without AMI (83.6 mg/dl) [16]. The difference observed between the 2 groups in our study was statistically significant (p value = 0.0204). Thus in our study the LDL – C was a major risk factor for myocardial infarction noted in our patients. The mean TG level of the control subjects compared with AMI (129.0 mg/dl) was significantly greater than that of subjects without AMI (107.8 mg/dl) (ArunKumar *et al.,* 2009). An interaction between triacylglycerols and the total cholesterol/HDL-cholesterol (TC/HDL-C) ratio has been demonstrated in several studies (Sonia *et al.,* 2008). The difference observed between the 2 groups in our study was statistically significant (p value = 0.0470). Thus in our study the Serum Triacylglycerol was a major risk factor for premature myocardial infarction noted in our patients.

High Malondialdehyde levels appear to be clearly associated with an increased risk of cardiovascular disease. This was also reported by Arun Kumar *et al*. (2009), Kaur *et al*. (2008), Rashmi Raghuvanshi *et al.* (2007). In the present study also there is an extremely significant association of MDA concentration and risk of MI with a *p* value of <0.0001. The difference between the 2 groups in our study was highly

Table 1. Mean ± S.D of various biochemical parameters in myocardial patients.

	TC (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)	TG (mg/dl)	MDA (mmol/L)
Cases	180 ± 19.77	41.4 ± 3.47	106.15 ± 16.19	156.2 ± 17.22	4.62 ± 0.696
Control	174.56 ± 16.41	44.06 ± 3.26	97.29 ± 12.33	149 ± 9	2.21 ± 0.505
	p value 0.259	p value 0.0033	p value 0.0204	p value 0.0470	P value <0.0001

Table 2. MDA And Its Relation With Sex, Total Cholesterol & Its Fractions

	Males	Females	LDL-C ≥100 mg %	LDL-C <100 mg %	HDL-C <40 mg %	HDL-C ≥40 mg %	S.CHOL ≥200 mg %	S.CHOL <200 mg %
No of patients	20	10	18	12	6	24	4	26
MDA Mean	4.7 ± 0.745	4.45 ± 0.586	5.0 ± 0.567	4.05 ± 0.440	4.627 ± 0.708	4.6 ± 0.707	4.625 ± 0.45	4.621± 0.733
	p value = 0.3629		p value < 0.0001		p value = 0.9339		p value = 0.9917	

DISCUSSION

Contemporary studies by Deborah Zucker *et al.* (1997) and others have also shown that the male sex is more prone to myocardial infarction. (Cunningham *et al.,* 1989; Sonia *et al.,* (2008). Cigarette smoking is the most common and most

significant (p value <0.0001) suggesting Malondialdehyde as an important predictor of coronary artery disease (MI). The difference in malondialdehyde levels observed between male and female groups in MI was not statistically significant. (p value = 0.3629). MDA was significant compared to LDL-C

(p value <0.0001) but not so against HDL-C (p 0.9339) and total cholesterol (p 0.9917). Our study shows that low LDL-C protects patients against MDA induced CAD while high HDL or a low Cholesterol may not.

Conclusion

In patients with myocardial infarction, there is a male preponderance and smoking is the most common presenting symptom. Low HDL-C and high LDL-C, Cholesterol, TG are common risk factors and MDA has significant association with most – though not all - risk factors. Patients have to be taught the benefits of maintaining lipid profile in normal range and removing the modifiable risk factors as per AHA & ASA guidelines.

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