ejpmr, 2018,5(1), 382-384

SJIF Impact Factor 4.161

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Research Article

ISSN 2394-3211

EJPMR

EFFECT OF HIGH ETHANOL CONSUMPTION ON LIPID PEROXIDATION, LIPID PROFILE AND ALCOHOLICS RELATED COMPLICATIONS

Sangita Paneri, P. Dey Sarkar, Meena Verma and Shakti Suslade

Dept. of Biochemistry, M.G.M. Medical College, Indore.

***Corresponding Author: Dr. Sangita Paneri**

Dept. of Biochemistry, M.G.M. Medical College, Indore.

Article Received on 20/11/2017 **Article Revised on 10/12/2017** **Article Accepted on 30/12/2017**

ABSTRACT

Damaging effects of Reactive oxygen species are well studied. They include oxidative attack on various cell constituents. Chronic ethanol Administration may leads to increased free radical damages like cardiomyopathy. cardiomyopathy. In this study we investigate Lipid profile and oxidative stress. This study has been carried out on 100 subjects, who are alcoholic. Levels of total Cholesterol, Triglycerides, LDL, VLDL significantly high, and level of HDL were found to be decreased. The chronic alcoholic individuals were compared with non alcoholic controls. Our result suggests that alcohol administration causes myocardial injury through the induction of oxidative stress. Alcoholic individuals are more susceptible to developing cardiovascular diseases. Depression of antioxidant system proves to be a marker for oxidative injury caused by the administration of ethanol.

KEYWORDS: OS – Oxidative Stress, MI – Myocardial Infarction, CVD – Cardio vascular disease, TAC-Total antioxidant capacity.

INTRODUCTION

Oxidative stress is defined as the imbalance between the oxidants and antioxidants, which results in to damage of different lipid membrane, organ systems and metabolic processes which has been suggested to be the cause of various human diseases.[1] OS develops due to generation of free radicals in the body. A free radical is defined as any species capable of independent existence that contains one or more unpaired electron in their outer orbital.[2] Oxygen free radicals are capable of damaging compounds of all biochemical classes including nucleic acids, proteins, lipids, lipoproteins, carbohydrates and connective tissue macromolecules.[3] Normally there is a balance between tissue oxidant and antioxidant scavenger system which includes enzymes like SOD, Catalase, GPX and antioxidant vitamins.[4]

Ethanol is the most psychoactive substance. Chronic alcoholism is a major health problem and causes multi organ disease and toxicity. Chronic ethanol administration is able to induce OS.[5] Alcoholic cardio-myopathy has been known for a long time but there is a little mechanistic insight into this clinical problem. Acute ethanol administration causes myocardial injury through, at least in part, the induction of OS. It is also associated with increase in the myocardial lipid peroxidation which is determined by measuring TBARS.[6] It is also known that ethanol consumption is able to induce OS in liver and in the extra hepatic tissues linked to the imbalance between the pro oxidants and the antioxidant system.[15] Reinke et al have observed that ROS play an important role in the onset of cardiac toxicity in chronically ethanol intoxicated animals.[7]

In our study, many parameters have been covered which may have patho physiology of MI. These parameters include lipid profile tests, oxidative stress molecule (MDA). These results give us a complete biochemical view of the susceptibility of MI in chronic alcoholic patients.

MATERIAL AND METHOD

The present study was carried out in the Department of Biochemistry, M.G.M. Medical College, Indore (M.P.) during the period of Jan 2017 to Dec 2017. study consisted of 100 chronic alcoholic individuals selected from who visited the OPD of M.Y. Hospital Indore and some by personal contact. 100 non alcoholic individuals served as controls. Proper history and consent of all the study subjects were taken.

Inclusion criteria – Males aged between 40-65 years, with habit of ethanol consumption.

Exclusion criteria – Males having above 65 years of age, Patients who were haemodynamically unstable in shock or heart failure were excluded. Patients who had known chronic kidney disease, Chronic liver disease, diabetes mellitus, Endocrine dysfunction, Malabsorption syndrome, Renal Rickets were excluded from the study.

5 ml overnight fasting blood sample was collected and subjected to biochemical estimations of lipid profile by enzymatic end point method in fully automated analyzer, and MDA level estimated by TBARS reaction (spectrophotometric) method.[8]

Statistical Analysis

Statistical analysis of data was done to compare each group. The data were expressed as mean ± standard deviation. Student 't' test for p value – p value less than 0.001 was taken as highly significant value and 0.05 as significant. Computer software was used for this analysis.

OBSERVATIONS

Table-1: Serum Lipid profile and MDA level of normal controls & alcoholic subjects.

S.No.	Parameters	Normal controls	Alcoholic subjects	p-value
		n =100	n =100	
1	MDA (umol/l)	3.4± 2.1	4.78±2.6	<0.05
2	TC (mg/dl)	152± 48.7	228± 44.0	<0.001
3	HDL (mg/dl)	46.4± 11.8	28± 9.2	<0.05
4	LDL (mg/dl)	84.2± 46	158± 54.8	<0.001
5	TG (mg/dl)	101± 58.2	190± 70.3	<0.001
6	VLDL (mg/dl)	22± 9.2	42± 11.2	<0.001

Data is represented as mean ± standard deviation. $p < 0.05$ and $p < 0.001$ v/s the control group.

RESULT

The result shows that there is rise in MDA level in alcoholic individuals as compared to normal controls.

Decrease in the serum HDL is also observed in alcoholic individuals when compared to normal controls.

Serum total cholesterol, TG, LDL, VLDL were also found to be elevated in the study group when compared to the normal controls.

DISCUSSION

Our result indicates that there is an increase in the OS in the individuals with chronic ethanol consumption. There is an imbalance due to increased generation of free radicals and decrease in antioxidant scavenger system. The results were compared with the results of normal non alcoholic subjects. A negative correlation is obtained between MDA and the Total antioxidant capacity and catalase. Increase MDA and decrease antioxidants enzymes is a significant of oxidative stress. Lipid peroxidation is an auto catalytic process which ultimately results in the cell death.[11] It has long been known that there is imbalance between oxidant and antioxidants in individuals with MI. Direct evidences have been received for increase in free radicals originating from the failing myocardium.[12] At the same time studies have also suggested the risk of MI in the individuals with chronic alcohol consumption.[13]

Alcoholic cardiomyopathy in humans has been recognized for a long time. In association with the cardiac tissue injury, acute ethanol administration induces significant cardiac injury which was revealed by elevation of serum CPK and GOT activities alterations in cardiac tissues.[5]

Heavy drinking promotes oxidation of LDL, a pathogenic factor in atherosclerosis and coronary heart disease. Ethanol increases lipid peroxidation and the secreted lipoproteins may undergo oxidative modification during secretion or may be depleted of antioxidants.[14] Alcohol's ability to induce lipid peroxidation has been related to hypothesis concerning damages caused directly or indirectly by ethanol or the major metabolite acetaldehyde. Current hypotheses include direct impact of free radicals derived by ethanol; ethanol's ability to generate formation of oxygen free radical species, which are able to start lipid peroxidation either directly or by exhausting antioxidant substance and acetaldehyde's ability to stimulate lipid peroxidation either directly through free radical formation or through depletion of the concentration of antioxidant.[16]

In conclusion, this study shows that acute alcohol administration causes significant cardiac damage in association with hyper lipidemia and increased Oxidative Stress. There is also an imbalance between oxidants and antioxidant molecule in association with the lipid profile. Thus it suggests that individuals with chronic ethanol consumption are susceptible to develop MI and other CVD due to decreased total antioxidant capacity the increased oxidative stress.

REFERENCES

1. Ashok K. Tiwari- "Antioxidants: New generation therapeutic base for treatment of polygenic disorders." Current science, 25th April 2004; 86: 8.
2. Saygili E.I., Akcay T, Konukoglu D, Papilla C- "Glutathione and related enzymes in colorectal cancer patients." IJBRA, 2009; (7): 384-389.
3. Carrol CE- "Oxygen free radicals and human disease." Ann Int Med, 1987; 107: 526-45.

4. Neela Patil, Vishwas Chavan, N.D. Karnik- "Antioxidant status in patients with acute myocardial infarction." Ind. J. Clin. Bioch, 2007; 22(1): 45-51.
5. Subir kumar Das, Hiran K.R., Sukhes Mukherjee, D.M. vasudevan- "Oxidative stress is the primary event: effects of ethanol consumption in Brain." Ind J Clin Bioch, 2007; 22(1): 99-104.
6. Muralidhar Kanan, Lipeng Wang, Y. James Kang- "Myocardial oxidative stress and toxicity induced by acute ethanol exposure in mice." Exp. Biology and medicine, 2004; 229: 553-559.
7. Reinke LA, Lai EK, Dubose CM, Mccay PB- "Reactive free radical generation in vivo in heart and liver of ethanol fed rats: correlation with radical formation in vitro." Proc Natt Acad Sci USA, 1987; 84: 9223-9227.
8. Inal ME, Kaubak G, Sunal E- "Antioxidant enzyme activities and malondialdehyde levels related to aging." Clin Chem Acta, 2001; 305(1-2): 75-80.
9. L.Goth- "A simple method for the determination of serum catalase activity and revision of reference range." CCA; Clin Chemica Acta, 1991; 196: 143-152.
10. Miller, NJ, Rice Evans, C, M J Gopinathan, A Clinicalscience, 1993; 84: 407-412.
11. Akila, V. Prashant, H. Harishchandra, Vivian D'Souza, Benedictta D'Souza- "Age related changes in lipid peroxidation in elderly people." Ind J Clin Bioch, 2007; 22(1): 131-134.
12. "Increased hydroxyl radicals originate from superoxides in failing myocardium." Circulation research, Feb 4th 2000; 86(2): 152-157.
13. Charles P Vega MD, Univ of California Irvine- "The need for preventing CVD." Medscape today, 2005.
14. Joel de Leiris, Michel de Lorgeril and francios Boucher- "Ethanol and cardiac function." AJP-Heart and Circulation physiology, Sep 1 2006; 291(3): 4-1028.
15. I.Dupont, P. Bodenez, F Berthou, B. Simon, L.G. Bardou, D. Lucas- "Cytochrome P-450 2E activity and oxidative stress in alcoholic patients." Oxford journals, alcohol and alcoholism, 2006; 3: 98-103.
16. Flemming Nielsen, Boborg Mikkelsen, Jesper Bo Nielsen, Helle Raun Andersen and Phillip Grand Jean- "Plasma malondialdehyde as biomarker for Oxidative stress: reference interval and effects of life style factor." Clinical Chemistry, 1997; 43: 1209-1214.