

Bio-chemical variables in ischemic heart diseases

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Abstract

Background: The aim of this study is to evaluate the bio chemical parameters in study comprising cases of IHD, to evaluate various biochemical variables in sex and age matched controls, compare the variables in cases and controls the following variables were selected for study lipid profile and oxidative stress **Methods:** In present study, 60cases of nearly Ischemic heart cases in the age 20-65years who were admitted in cardiology unit. **Results:** out of 60cases recruited male patients were more risk in cardiovascular disease, in which 41-50 years of age group was in high risk and 20-30 was in least risk, mean values of Total cholesterol, LDL TG were significantly increased in CVD patients as compared to control ($p < 0.0001$). The mean level of HDL in CVD patients were significantly decreased as compared to control ($p < 0.0001$). serum MDA level in CVD patients were found to be significantly increased ($p < 0.001$) as compared to control. The serum level of TAC in CVD patients were found to be decreased compare to controls. **Conclusion:** In my study mean levels of serum cholesterol, TG, LDL-C and MDA in cases of IHD have increased significantly, when compared with controls. Mean serum HDL-C and TAC Levels have decreased significantly when compared with controls. our study shows that lipid levels are playing a significant role in IHD.

Key words: TG - Triglycerides, HDLc- High density lipoprotein cholesterol, LDLc- Low density lipoprotein cholesterol. Cardiovascular disease; Lipid profile; Enzymatic antioxidants; Oxidative stress.

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INTRODUCTION

Coronary artery disease (CAD), also known as coronary heart disease (CHD), Ischemic heart disease (IHD), or simply heart disease, involves the reduction of blood flow to the heart muscle due to build-up of plaque (atherosclerosis) in the arteries of the heart.¹ Types stable angina, unstable angina, myocardial infarction, and sudden cardiac death.¹² A common symptom is chest pain or discomfort which may travel into the shoulder,

arm, back, neck, or jaw.¹ Occasionally it may feel like heartburn. Usually symptoms occur with exercise or emotional stress, last less than a few minutes, and improve with rest.¹ Shortness of breath may also occur and sometimes no symptoms are present.¹ In many cases, the first sign is a heart attack.² Ischemic Heart disease is caused by lack of oxygen and blood due to inadequate circulation which result from an imbalance of supply and Demand. Common is arteriosclerosis of Epicardial coronary arteries. Reactive oxygen species (ROS) are highly reactive chemical species containing oxygen, including the superoxide (O₂⁻) and the hydroxyl (OH⁻) anions, and hydrogen peroxide (H₂O₂). Under normal physiological conditions, ROS levels are strictly controlled through the activity of antioxidant enzymes, including superoxide dismutase, catalase, and glutathioneperoxidase [Oxidative stress is defined as a dysregulation between the production of ROS and the endogenous antioxidant defense mechanisms, resulting in excessive ROS linked to multiple pathophysiological pathways in the heart.²⁰ several observational studies and clinical trials have

revealed the adverse effects of abnormal blood lipid and lipoprotein levels in the pathogenesis and progression of atherosclerosis and cardiovascular disease. Numerous population studies have shown an inverse correlation between plasma high density lipoprotein (HDL) levels and risk of CVD^{13,14}. In view of the pathological role of increased lipid profile, Oxidative stress and decrease antioxidants status in the development and progression of CVD, Hence, the present study is designed to determine level of lipid profile, markers of oxidative stress (MDA, TAC) and status of enzymatic antioxidants (TAC) in CVD patients, so that study would be helpful in assessment and prevention of cardiovascular disease. MDA were significantly increased in CVD patients as compared to control (p<0.0001) and HDL, TAC in CVD patients were significantly decreased as compared to control (p<0.0001). The present study reveals the importance of determining the lipid profile, free radicals and antioxidant status in CVD to enable the formulation of specific therapies for early intervention and better management of disease.

MATERIALS AND METHODS

In the present study, 60 cases of newly diagnosed IHD in the age group of 20-65 years who were admitted to cardiology unit of Govt General Hospital were recruited for the study after obtaining written informed consent (study group). 20-65yrs Sex and Age matched persons were selected from the patient's attendants and hospital staff as controls

SAMPLE COLLECTION: 5ml of blood was collected from the subjects as well as controls after overnight fasting (12hr) by venipuncture. Serum was separated within 1 hr. The following parameters were analyzed on same day on semi-auto analyzer Trausassiae using ERBA Kits.

1. Total cholesterol (accurex kit)
2. Triglycerides (accurex kit)
3. High density lipoprotein Cholesterol (HDL-C) (Accurex)

4. Low density lipoprotein Cholesterol (LDL-C) (FRIEDEWARLD FORMULA)

Remaining serum was liquated and kept in deep freezer at -70. Centigrade, Following parameters were analyzed within 4 weeks,

1. Ferric reducing ability of plasma (FRAP) for TAC
2. Malondialdihyde [MDA] (TBARS)
3. Biochemical Analysis

Serum lipid profile (the level of total cholesterol, TG, LDL and HDL) was measured by kit supplied by Randox Laboratories Ltd, using fully automated clinical chemistry analyzer (Transasia EM-360, Boehringer Mannheim, Germany).

INSTRUMENTS USED: U.V Spectro photometer.

INCLUSION CRITERIA: All the patients diagnosed as MI based on clinical, electrocardiographic and laboratory criteria in the age group of 20 to 65 years.

EXCLUSION CRITERIA: The subjects having diabetes mellitus, Hypertension, endocrine disorders, obesity, alcohol abusers, and Cigarette smokers and alcoholics as well as patients on antioxidant supplements, lipid lowering drugs were excluded from the study. Biochemical variables were measured by standard methods. Mean (+ or -) S.D value of all biochemical parameters were calculated and mean difference was compared between the study group and controls by using student 't' test. (statistical analysis and reference ranges)

KITS: ACCUREX kits and FRIEDEWARLD FORMULA for LDL and TBARS for MDA

Estimation of total Antioxidant capacity by ferric reducing ability of plasma assay (FRAP).

statistical analysis: it is carried between group I (control) and

group II (cardiac patients) was done by using unpaired Student's t- test and results were expressed as Mean (M), standard deviation (S.D.). Difference between the control and study groups were considered significant when the p value determined by unpaired student's t- test was (p<0.001).

RESULTS AND DISCUSSION

Table 1: Gender wise distribution of control and CVD Patients group study group. (N = 120)

Gender	Control group (n=60)	CVD Patients (n=60)
Male	32 (53.3%)	44 (73.33%)
Female	28 (46.6%)	16 (26.67%)

Table 2: Age wise distribution of control and CVD Patients study group. (N=120)

Age group (25- 65 years)	Control (n=60)	CVD Patients (n=60)
20-30	16	8 (13.33%)
31-40	20	12 (20%)
41-50	14	18 (30%)
51-60	6	12 (20%)
61-70	4	10 (16.66%)

Table 3: Comparison of serum lipid profile in control and CVD patients

	Parameters Control (n=60)	CVD patients(n=90)	P-values
	Mean± SD	Mean± SD	
Cholesterol (mg/dl)	176.15 ± 29.60	223 ± 71.22	p< 0.001
LDL (mg/dl)	107.7 ± 25.96	150.97 ± 63.84	p< 0.001
HDL (mg/dl)	40.82 ± 3.09	37.47 ± 3.04	p< 0.001
Triglyceride (mg/dl)	135.95 ±44.92	175.63 ± 67.91	p< 0.001

Table 4: Comparison of serum MDA, TAC levels in Control and CVD patients

Parameters Control	CVD Patients	p-values
MDA (nMOL/Litt)	1.76 ±0.84 5.6 ±1.57	p<0.001
TAC(MicroMOL/Litt)	1.17± 0.36 0.57±0.18	p<0.001

p<0.001 is highly significant.

The study was included total 120 subjects Table no 1 shows .Gender wise distribution All the subjects were divided into two groups, first group consists of 60 patients with cardiac diseases and second group consists of 60 healthy control subjects shows male patients were more risk in cardiovascular disease 44(73.33%) compare to female patient 16(26.67%), may be because of high body mass index . Table no 2 shows the age wise distribution of control and study group in which 41-50 years of age group was in high risk 18(30%) and 20-30 was in least risk 8(13.33%).

Table no 3 shows that the mean levels of lipid profile in CVD patients were T.chol (223.10±71.22 mg/dl), LDL (150.97 ± 63.84 mg/dl), HDL (37.47± 3.04 mg/dl), TG (175.63 ± 67.91mg/d). In control group the mean levels were T. chol (176.15 ± 29.60 mg/dl), LDL (107.7 ± 25.96 mg/dl), HDL (40.82 ± 3.09 mg/dl), TG (135.95 ±44.92 mg/dl). We found, mean values of Total cholesterol, LDL TG were significantly increased in CVD patients as compared to control (p<0.0001). The mean level of HDL in CVD patients were significantly decreased as compared to control (p<0.0001).

Table no 4 In the present study, serum MDA level in CVD patients (5.6 ±1.57 nmol/L) were found to be significantly increased (p<0.001) as compared to control (1.76 ±0.84nmol/L). . The serum level of TAC in CVD patients (0.57±0.18 µmol /L) were found to be decreased compare to controls (1.17± 0.36 µmol/L).

DISCUSSION

STABLE ANGINA

In "stable" angina, chest pain with typical features occurring at predictable levels of exertion, various forms of cardiac stress tests may be used to induce both symptoms and detect changes by way of eeg, echocardiography (using ultrasound of the heart) or scintigraphy (using uptake of radionuclide by the heart muscle).

ACUTE CORONARY SYNDROME

Diagnosis of acute coronary syndrome generally takes place in the emergency department, where ECGs may be

performed sequentially to identify "evolving changes" (indicating ongoing damage to the heart muscle). Diagnosis is clear-cut if ECGs show elevation of the "ST segment", which in the context of severe typical chest pain is strongly indicative of an acute myocardial infarction (MI); this is termed a STEMI (ST-elevation MI), and is treated .

RISK FACTORS

The major independent risk factors for the development of atherosclerosis are the plasma cholesterol concentration, triglyceride, cigarette smoking, hypertension and diabetes, which are by them self's risk factor for coronary heart disease³. The presence of any risk factor is associated with doubling the relative risk of developing atherosclerotic coronary artery disease. Cardiovascular disease (CVD) threatens to cripple India's workforce and stunt India's growth if timely and appropriate public health measures are not instituted. A large amount of epidemiological evidences also supports the relationship between serum low density lipoprotein cholesterol (LDL-c) and June, 2017/ Vol 5/Issue 06 ISSN- 2321-127X Original Research Article International Journal of Medical Research and Review Available online at: www.ijmrr.in 621 | P a g e coronary artery disease (CAD) in Indians⁴. Serum high density lipoprotein-cholesterol (HDLc) level has been found to have inverse relationship with the coronary artery disease (CAD)⁵. Diabetes mellitus is a common among Indians with coronary heart disease (CHD) both in their land of origin and abroad⁶. Individuals with non insulin dependent diabetes mellitus (NIDDM) are more likely to have multiple risk factors for CHD than age matched non diabetic subjects. Peoples with diabetes have a risk of CHD two to five times that of nondiabetic individuals⁷. Oxidative stress appears to be a probable clinically relevant factor in cigarette smoke related atherogenesis and increased lipid peroxidation is also a risk factor for myocardial infarction⁸. Effect of smoking are dyslipidaemia, increased free radicals, oxidative stress, oxidation of LDL, damage to endothelium and thus promotion of atherosclerosis. Hyperglycemia, (NIDDM) hypertension and cigarette smoking depletes natural

antioxidants and facilitates the production of reactive oxygen species (ROS) which has the ability to react with all biological molecules like lipids, proteins, carbohydrates, DNA etc and exert cytotoxic effects on cellular components.⁹ Biochemical and Pathological Consequences of Insulin Resistance Smoking is other risk factors for CHD increases the risk of cardiovascular diseases include systemic haemostatic and coagulatory disturbances, lipid abnormalities, increase in oxidative stress, and vascular increase in the concentration of serum total cholesterol, LDL anti-atherogenic HDL-c.¹⁰ Under certain normal conditions oxygen may accept only one electron (usually in the electron transport chain accepts four electrons and get converted to water) and this results which may initiate the chain reaction of free radicals is stable products of lipid peroxidation. Malonaldehyde levels are indicative of lipid peroxidation which is a oxidative degeneration of polyunsaturated fatty-acids. injury on membrane lipids. such as accelerated formation of reactive oxidase¹¹.

Smoking is other risk factors for CHD increases the risk of cardiovascular diseases include systemic haemostatic and coagulatory disturbances, lipid abnormalities, increase in oxidative stress, and vascular increase in the concentration of serum total cholesterol, LDL anti-atherogenic HDL-c.¹⁵ It is presumed that nicotine stimulates sympathetic increased secretion of catecholamine fatty acids, which further results in increased synthesis of hepatic triglycerides, along with VLDL stream¹⁶. High levels of LDL-c, VLDL coronary artery disease, while a low level of HDL artery disease¹⁷. In those with stable CAD it is unclear if PCI or CABG in addition to the other treatments improves life expectancy or decreases heart attack risk.¹⁸ Rheumatoid arthritis, SLE, psoriasis, and psoriatic arthritis are independent risk factors as well. Job stress appears to play a minor role accounting for about 3% of cases.¹⁹ In one study, women who were free of stress from work life saw an increase in the diameter of their blood vessels, leading to decreased progression of atherosclerosis.²⁰ In the heart, ROS play a fundamental function in cell homeostasis when present at low concentrations, since they regulate multiple physiological signaling pathways and biological processes. Oxidative stress is defined as a dysregulation between the production of ROS and the endogenous antioxidant defense mechanisms, resulting in excessive ROS linked to multiple pathophysiological pathways in the heart.²⁰ Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the heart's muscle cells. The heart's muscle cells may die from lack of oxygen and this is called a myocardial infarction (commonly referred to as a heart attack).²¹ Cardiac syndrome X is chest pain (angina pectoris) and chest discomfort in people who do not show signs of

blockages in the larger coronary arteries of their hearts when an angiogram (coronary angiogram) is being performed.²² The sensitivity of D-Dimer for the diagnosis of LAA thrombus is 100%. The specificity of D-Dimer for the diagnosis of LAA thrombus is 71.4%. So, patients with positive and elevated D-Dimer levels should undergo TEE for the diagnosis of LAA thrombus.²³ A number of theories exist regarding the elevated risk for adverse events for patients with CVD who develop COVID-19. In particular, better understanding of the relationships involving the ACE2 protein, antihypertensive agent use, and COVID-19 prognosis will have important implications for patients with both COVID-19 and CVD.²⁴ Dyslipidemia was an important risk factor for atherosclerosis and Coronary heart disease. Where high levels of cholesterol, triglycerides, (LDL-C), and decreased HDL cholesterol (HDL-C) in Plasma are associated with increased risk for CHD, Several mechanisms could explain the strong effect of lipid profile on the rate of CHD.²⁵

CONCLUSION

ISCHEMIC HEART DISEASE is one of the major health problems in the society. In this background, it is concluded that Patients with CHD have altered lipid profile, with higher levels of TGs, total cholesterol, VLDL and LDL and low level of serum HDL; this difference may play a role in the pathophysiology found in Patients with CHD.²⁵ It is one of the leading causes of death throughout the world in our country as well IHD is on the rise and causing great morbidity and mortality which ultimately is resulting in great burden on the society. It was found that, the level of total cholesterol, triglyceride, low density lipoprotein in CVD patients were significantly high as compared to control ($p < 0.001$). Level of high density lipoprotein was significantly low as compared to controls ($p < 0.001$). Level of TAC was significantly lower in CVD patients as compared to controls ($p < 0.001$). The marker of free radical induced injury i.e. Malonaldehyde (MDA) was significantly high ($p < 0.001$) in CVD patients as compared to controls. The levels of enzymatic antioxidants were also significantly lower in CVD patients as compared to controls ($p < 0.001$). It appears that, both increase in lipid profile and MDA with subsequent decline in TAC shows that CVD patients. The present study therefore reveals the importance of determining the lipid profile, free radicals and antioxidant status in CVD to enable the formulation of specific therapies for early intervention and better management of disease. The detection of risk factors in early stage and correction of disease condition will help the patients to improve, reduce further complications and Mortality rate.¹²

REFERENCES

1. Jump up to: "What Are the Signs and Symptoms of Coronary Heart Disease?". 29 September 2014. Archived from the original on 24 February 2015. Retrieved 23 February 2015.
2. Jump up to: "Coronary Artery Disease (CAD)". 12 March 2013. Archived from the original on 2 March 2015. Retrieved 23 February 2015.
3. Ross R. The pathogenesis of atherosclerosis--an update. *N Engl J Med.* 1986 Feb 20;314(8):488- 500.
4. Enas EA, Garg A and Davidson MA. Coronary artery disease and it's risk factors in 1st generation immigrant Asian Indian to the United State))of America. *Indian Heart Journal* 1996,48 (4)343-53.
5. Castelli WP and Andreson KA .Population at risk prevalence high cholesterol level in hypertensive patients Framingham study.*American Journal of Medicine* 1986, 80(2A) 23-28.
6. Bhoraskar AS and Raheja DS. Diabetes and cardiovascular disease Do Asian Indians have a high ethnic susceptibility. *Journal of the Association of Physicians of India* 1997,34 72-8.
7. Meigs JB, Singer DE and Sullivan LM. Metabolic control andPrevalent cardiovascular disease in non- insulin dependent diabetes mellitus (NIDDM); The NIDDM patient outcomes research group. *American Journal of Medicine* 1997,102(1) 38-47.
8. McLaughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Reaven G. Use of metabolic markers to identify overweight individuals who are insulin resistant. *Ann Intern Med.* 2003 Nov 18;139(10):802-9.
9. Venkatesan A, Hemalatha A, Bobby Z, Selvaraj N, Sathiyapriya V. Effect of smoking on lipid profile and lipid peroxidation in normal subjects. *Indian J Physiol Pharmacol* 2006; 50(3): 273-278.
10. Dincer Y, Akcay T, Aldemir Z and Likooova H . Effect of oxidative stress on Glutathione pathway in red blood cells from patients with insulindependent diabetes mellitus. *Metabolism* 2002, 51(10) 1360-1362.
11. Maritim AC, Sanders RA, Watkins JB 3rd. Diabetes, oxidative stress, and antioxidants: a review. *J Biochem Mol Toxicol.* 2003;17(1):24-38.
12. Study of lipid profile, enzymatic antioxidant and oxidative stress in cardiovascular diseases Binod Mahato*1, Chaudhary B. L.2 and Brij Nandan Singh3 1.Department of Biochemistry, Mayo Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.
2.Department of Microbiology, Mayo Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.
3.Department of Microbiology, G.S.V.M. Medical College, Kanpur, India.
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13. RH Glew, Williams M, Conn CA, Cadena SM, Crossey M and Okolo SN. "Cardiovascular disease risk factors and diet of Fulani pastoralists of northern Nigeria". *Am J Clin Nutr* 74(2001):730 - 736.
14. RH Glew, Kassam HA, Bhanji RA, Okorodudu A and VanderJagt DJ. "Serum lipid profiles and risk of cardiovascular disease in three different male populations in northern Nigeria". *J Hlth Popul Nutr* 20.2(2002):166-174.
15. Taskinen MR, Lahdenperä S, Syväne M. New insights into lipid metabolism in non-insulindependent diabetes mellitus. *Ann Med.* 1996 Aug;28(4):335-40.
16. Giugliano D, Ceriello A and Paolisso G . Diabetes mellitus, hypertension and cardiovascular disease and oxidative stress. *Metabolism*,1995, 44(3) 363-368.
17. Mukaopadhyya .Free radicals and diabetes, Role in aetiology and pathogenesis *Journal of Diabetic Association of India.*1994, 34 5-7.
17. Waqar A. Effect of tobacco smoking on the lipid profile of teenagemale population in Lahore City. *Int J Med Med Sci* 2010; 2(6):172-177.
18. Rezende PC, Scudeler TL, da Costa LM, Hueb W (February 2015). "Conservative strategy for treatment of stable coronary artery disease". *World Journal of Clinical Cases.* 3 (2)163–70. doi:10.12998/wjcc.v3.i2.163. PMC 4317610. PMID 25685763.
19. Moran AE, Forouzanfar MH, Roth GA, Mensah GA, Ezzati M, Murray CJ, Naghavi M (April 2014). "Temporal trends in ischemic heart disease mortality in 21 world regions, 1980 to 2010: the Global Burden of Disease 2010 study". *Circulation.* 129 (14): 1483–92. doi:10.1161/circulationaha.113.004042. PMC 4181359. PMID 2457335222)^ Moran AE, Forouzanfar MH, Roth GA, Mensah GA, Ezzati M, Flaxman A, *et al.* (April 2014). "The global burden of ischemic heart disease in 1990 and 2010: the Global Burden of Disease 2010 study". *Circulation.* 129 (14): 1493–501. doi:10.1161/circulationaha.113.004046. PMC 4181601. PMID 24573351.
20. The Role of Oxidative Stress in Cardiac Disease: From Physiological Response to Injury Factor Rossella D'Oria, Rossella Schipani, Anna Leonardini, Annalisa Natalicchio, Sebastio Perrini, Angelo Cignarelli, Luigi Laviola, and Francesco Giorgino11Department of Emergency and Organ Transplantation– Section of Internal Medicine, Endocrinology, Andrology and Metabolic Diseases, University of Bari Aldo Moro, Piazza Giulio Cesare, 11, I-70124 Bari, Italy Academic Editor: Gianna FerrettiReceived18 Nov 2019Revised11 Jan 2020Accepted22 Apr 2020 Published14 May 2020
21. Ambrose JA, Singh M (2015). "Pathophysiology of coronary artery disease leading to acute coronary syndromes". *F1000prime Reports.* 7: 08. doi:10.12703/P7-08. PMC 4311268. PMID 25705391.(19)
22. Kaski JC (February 2004). "Pathophysiology and management of patients with chest pain and normal coronary arteriograms (cardiac syndrome X)". *Circulation.* 109 (5): 568–72. doi:10.1161/01.CIR.0000116601.58103.62. PMID 14769677 .
23. Original Research (Original Article). 2020; 11(3): 117-123doi: 10.31838/jcdr.2020.11.03.27 Samir Rafla, Tarek Beshay.
24. Journal of the American College of Cardiology Volume 75, Issue 18, May 2020DOI: 10.1016/j.jacc.2020.03.031 JACC STATE-OF-THE-ART REVIEW.Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the COVID-19 PandemicElissa Driggin, Mahesh V. Madhavan, Behnood Bickdeli, Taylor Chuich, Justin Laracy, Giuseppe Biondi-Zoccai, Tyler S. Brown.
25. Lipid Profile and Coronary Heart Disease.December 2017 Authors:Maha Radhi Abess Majid Kadhum Hussain16.42 University of Kufa College of Medicine Zuhair Mohammed Ali Jeddo3 3.83 ,University of Kerbala.

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