ORIGINAL ARTICLE

Assessment Study on Abnormality in Serum Lipid Profile in Patients with High Risk Myocardial Infarction

NIDA KOMAL BUTT[1], MUHAMMAD AFZAL CHOUDHRY[2], MUHAMMAD UMER[3], *ROBINA RASHID[1], SAJID MEHMOOD[1]*

ABSTRACT

Myocardial Infarction is one of the leading causes of death worldwide. Serum lipid profile and BMI are found to be major risk factors in patients suffering from MI. smoking; faulty dietary habits and low physical activity or sedentary life style enhances the risk of myocardial infarction. However, these are modifiable risk factors and can be changed by altering the style of living. These factors also greatly modify the serum lipid profile. Of all the serum lipid profiles analyzed maximum patients showed an increase in LDL, Triglycerides and a significant decrease in serum HDL levels. Enzyme assays were also carried out. There was a significant raise observed in CK and LDH of majority of patients.

Keywords: Myocardial infarction, lipid profile, HDL, LDL

INTRODUCTION

In the previous years, considerable studies have been done regarding the determination and improvement of risk factors, such as high serum total low density lipoproteins (LDL), high density lipoproteins (HDL), cholesterol level, for cardiovascular diseases (CVD). Many studies have shown that low levels of high density lipoproteins and cholesterol are associated with an increased risk of coronary diseases and myocardial infarction (Adak and Shivpuri., 2010).

Myocardial infarction is a major cause of morbidity and mortality and unhealthiness across the globe (Shirafkan *et al.*, 2012). MI is a major risk factor after the age of 40 in men and 50 in women (Narayana *et al.*, 2011). A recent survey report estimated that approximately 12 million deaths occur worldwide due to myocardial infarction (Narayana *et al.*, 2011).

It is a disease caused as a result of less blood supply (oxygen and nutrients) to the heart, a state known as myocardial ischemia (Reddy *et al.*, 2011). In this state the blood supply to the heart is diminished resulting in disturbance of myocardial cellular repair mechanism and hence interfering normal function and homeostasis. If this state of ischemia prolongs, it results in irreversible myocardial cell damage and cell death (Reddy *et al.*, 2011).

----

[1]*Department of Biochemistry and Molecular Biology, Medical Biochemistry Laboratory,*
[2]*Department of Medicine, ABSTH*
[3]*Department of Anatomy,*
Nawaz Sharif Medical College, University of Gujrat, Hafiz Hayat Campus, Gujrat, Pakistan.
Correspondence to Dr. Sajid Mehmood Email: sajid.mehmood@uog.edu.pk; Cell: +92-53-3643112

Myocardial cells may die from lack of oxygen and this is called myocardial infarction (commonly called a heart attack). There is a difference between myocardial ischemia and myocardial infarction. Myocardial ischemia means myocardial cells have suffered a lack of blood supply and do not function optimally. This condition can be reversed if the blood supply to the tissue is improved, whereas, myocardial infarction means that the tissue has suffered a lack of blood supply for a long period of time and that tissue has undergone death. This condition is not reversible (Reddy *et al.,* 2012).

Critical myocardial ischemia can be caused by two reasons, an increase in metabolic demand of myocardial cells or decrease in delivery of oxygen and nutrients to the myocardium. An increase in metabolic demand takes place due to severe physical exertion, severe hypertension and severe aortic valve stenosis (Kumar *et al.* 2009, Reddy *et al.*, 2012).

The patients suffering from myocardial infarction have obstructive arteries. This obstruction occurs when a thrombus is superimposed on an unstable atheromatous plaque and results in coronary occlusion. This atheromatous plaque is made up of fat, cholesterol etc, within the walls of the coronary arteries that supply the myocardium or myocardial cells with oxygen and nutrients. This plaque fills up the lumen of the coronary artery hence, decreasing its diameter and narrowing the path for blood supply i.e, supply of oxygen and nutrients (Adak and Shivpuri., 2010). This leads to myocardial ischemia. After decades, this plaque may rupture and start limiting blood flow to the heart muscles. The acute rupture of an atheromatous plaque may lead to an acute myocardial infarction (Reddy *et al.,* 2012).

Patients suffering from dyslipidemia are at a higher risk of myocardial infarction (Kumar *et al.*,

2009), as dyslipidemia is both atherogenic and thrombogenic (Adak and Shivpuri, 2010). Low levels of high density lipoproteins (HDL) are commonly observed in myocardial infarction patients (Kumar *et al.*, 2009). Higher levels of serum HDL are protective against ischemic stroke (Willey *et al.*, 2009). Elevated LDL levels have been established as risk factor for cardiovascular disease (Willey *et al.*, 2009). It is a modifiable risk factor (Bitla *et al.*, 2009).

LDL consists of a heterogenous group of particles. These particles have varying size and density. The smaller particles are more atherogenic as compared to the larger particles. The reasons for the atherogenicity of smaller LDL particles are reduced affinity for LDL receptors, increased binding for endothelial proteoglycans, better penetration of arterial intima (Landray *et al.*, 2002).

The small and dense LDL particles have been associated with coronary and carotid artery diseases and are considered to be a risk factor for myocardial infarction (Landray *et al.*, 2002). The patients suffering from untreated hypertension showed abnormalities in LDL sub fractions. LDL sub fractions are significantly higher in hypertensive patients as compared to non-hypertensive patients (Landray *et al.*, 2002).Important risk factors of myocardial infarction are previous cardiovascular disease, smoking, alcoholism, lack of physical activity, hypertension and abnormal levels of lipids in the blood serum. Abnormal lipid profile in case of myocardial infarction refers to raised levels of total cholesterol, triglycerides and low density lipoproteins (LDL) and a decrease in high density lipoproteins (HDL) (Narayana *et al.*, 2011, Pandey *et al.*, 2012). These are modifiable risk factors i.e.; they can be reversed by altering lifestyle of the patient. Age, sex and family history are considered as non-modifiable risk factors (Narayana *et al.*, 2011).

The enzymes playing an important role in the diagnosis of Myocardial Infarction are Creatine kinase, CK-MB, Lactate Dehydrogenase (LDH), Aspartate aminotransferases (AST), Alkaine aminotransferases (ALT). In myocardial infarction the levels of all these enzymes are significantly increased (Narayana *et al.*, 2011)Diabetes Mellitus is also considered as a major risk factor for coronary heart disease (CHD) and myocardial infarction (Chiariello and Indolfi, 1996). Evidence is present that asymptomatic myocardial infarction that is, painless myocardial infarction is much more frequent in diabetic patients than in non-diabetic. Myocardial Infarction is a major complication of diabetes mellitus.
.

Infact, the occurrence of myocardial infarction in diabetic patients is more severe and extensive in comparison to non-diabetic patients (Chiariello and Indolfi, 1996). It is a significant secondary cause because patients get an atherogenic combination of high TG's; high small, dense LDL fractions low HDL (diabetic dyslipidemia, hypertriglyceridemic hyperapo B). Patients suffering from type 2 diabetes are especially at risk (Krentz, 2003). The combination may be a consequence of obesity, poor control of diabetes, or both, which may increase circulating free fatty acids (FFAs), leading to increased hepatic very-low-density lipoprotein (VLDL) production. TG-rich VLDL then transfers TG and cholesterol to LDL and HDL, promoting formation of TG-rich, small dense LDL and clearance of TG-rich HDL (Krentz, 2003). Diabetic dyslipidemia is often exacerbated by the increased caloric intake and physical inactivity that characterize the lifestyles of some patients with type 2 diabetes (Krentz, 2003).

It is also studied that Hypertension and diabetes together increase the chances of myocardial infarction. This is because these both diseases occurring together cause cardiac fibrosis to a much greater extent than it is caused by any of the disease occurring alone. Diabetes also serves as a precursor for dyslipidemia and hypertension (Chiariello and Indolfi, 1996).

The present study was conducted to evaluate the abnormalities in lipid profile of patients suffering from myocardial infarction. Also the goal of the study was to evaluate other risk factors causing myocardial infarction. Risk factors such as smoking, alcoholism, non-vegetarian diet and diabetes mellitus was also recorded by using a proforma.

MATERIALS AND METHODS

Fasting blood samples of patients were collected from Aziz Bhatti Shaheed Teaching Hospital of Nawaz Sharif Medical College, University of Gujrat, Gujrat. A venous blood sample of n=52 patients suffering from myocardial infarction was collected. Approximately 5ml blood was drawn in disposable plastic syringes. The blood serum was then separated in eppendrof tubes and centrifuged. Lipid profile and following enzyme assays, LDH, ALT, AST and CK were carried out.of these patients in the Diagnostic Lab, Nawaz Sharif Medical College, University of Gujrat. All the serum samples were analyzed by using diagnostic kits for the quantitative estimation of following parameters

Low Density lipoproteins (LDL), High density lipoproteins (HDL), Triglycerides (Tg), Total Cholestrol, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Creatine Kinase (CK), Lactate dehydrogenase (LDH).

Low Density Lipoproteins (LDL): LDL was determined using invitro diagnostic reagent for quantitative estimation of LDL in serum with photometric systems using kit (Merck).

Principle: 1ˢᵗ step:

Sample was mixed with reagent R1, non-LDL lipoproteins were solubilized by detergent 1 and released cholesterol was subjected to enzymatic reactions to be eliminated:

Detergent 1+CO+CHE

Cholesterol, HDL, VLDL, Chylomicron $\longrightarrow$ Colorless products

2ⁿᵈ step: Reagent R2 was added, LDL was solubilized by detergent 2, then LDL cholesterol was measured by enzymatic reactions:

Detergent 2

LDL $\longrightarrow$ Solubilized LDL

CHE+CO

LDL Cholesterol + O_2 $\longrightarrow$ Cholest4-en-3-one + H_2O_2

Peroxidase

H_2O_2+ 4-AA + DSBmT $\longrightarrow$ Colored compound

Calculation:

$$\frac{(A2 - A1) \times n}{(A2 - A1)}$$ n= calibrator concentration

High Density Lipoproteins (HDL): HDL was determined using invitro diagnostic reagent for quantitative estimation using photometric methods using kit (Merck).

Principle: 1ˢᵗ Step: Sample was mixed with reagent R1 containing a selective accelerator, cholesterol of non-HDL lipoproteins was subjected to enzymatic reactions to be eliminated:

Accelator+CO+DSBmT+POD

LDL, VLDL, Chylomicron $\longrightarrow$ Non-reactive LDL, VLDL, Chylomicron

2ⁿᵈ Step:

HDL specific detergent

HDL $\longrightarrow$ Solubilized HDL

CHE + CO

HDL Cholesterol + O_2 $\longrightarrow$ Cholest-4-en-3-one + H_2O_2

Peroxidase

H_2O_2 + 4-AA + DSBmT $\longrightarrow$ Colored compound

Calculation:

$$\frac{(A2 - A1) \times n}{(A2 - A1)}$$ n= calibrator concentration

Triglycerides: Triglycerides level in blood serum was determined using invitro diagnostic reagent for quantitative estimation of Triglycerides in serum with photometric systems using kit (Merck).

Principle: Determination of triglycerides after enzymatic splitting with Lipoproteinlipase. Indicator is quinoneimine which is generated from4-aminoantipyrine and 4-Chlorophenol by hydrogen peroxide under the catalytic action of peroxidase.

LPL

Triglycerides $\longrightarrow$ Glycerol + Fatty Acids

GK

Glycerol + ATP $\longrightarrow$ Glycerol 3- phosphate + ADP

GPO

Glycerol 3 phosphate + O2 $\longrightarrow$ Dyhydroxy aceton Phosphate + H_2O_2

POD

H_2O_2 + Aminoantipyrine + 4-Chlorophenol $\longrightarrow$ Quinoneimine + $4H_2O$ + HCl

Calculation:

$$\text{Triglycerides concentration} = \frac{\textbf{Ast x Conc. of Standard}}{\textbf{Ast}}$$

Cholesterol: Cholesterol was determined using invitro diagnostic reagent for quantitative estimation of Cholesterol in serum with photometric systems using kit (Merck).

Principle: The cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminophenazone in the presence of phenol and peroxidase.
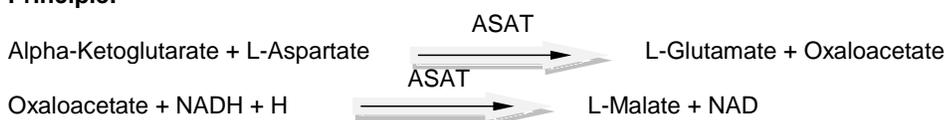
$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{CHE}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{CHO}} \text{Cholest-4-en-3-one} + H_2O_2$$

$$2H_2O_2 + \text{4-Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{POD}} \text{Quinoneimine} + 4H_2O$$
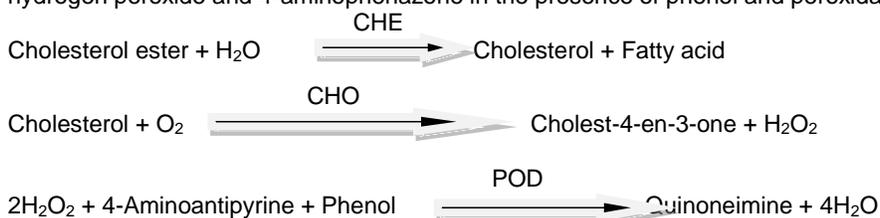
Calculation:

Concentration of cholesterol= $\dfrac{\text{A Sample} \times \text{Conc Std. [mg/dl]}}{\text{A Std} \quad \triangle}$

Aspartate aminotransferase (ASAT): ASAT was determined using invitro diagnostic reagent for quantitative estimation of ASAT in serum with photometric systems using kit (Merck).

Principle:

$$\text{Alpha-Ketoglutarate} + \text{L-Aspartate} \xrightarrow{\text{ASAT}} \text{L-Glutamate} + \text{Oxaloacetate}$$

$$\text{Oxaloacetate} + \text{NADH} + H \xrightarrow{\text{ASAT}} \text{L-Malate} + \text{NAD}$$

The rate of the NADH consumption was measured photometrically and was directly proportional to the ASAT activity in the sample.

Calculation: Enzyme activity [U/L] $\triangle$ = (A/ min) x F

	334 nm	340 nm	365nm
F	2184	240	3971

Alanine aminotransferase (ALAT): ALAT was determined using invitro diagnostic reagent for quantitative estimation of ALAT in serum with photometric systems using kit (Merck).
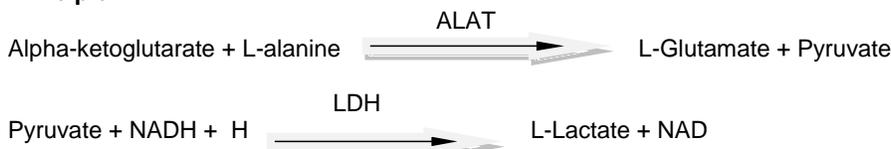
Principle:

$$\text{Alpha-ketoglutarate} + \text{L-alanine} \xrightarrow{\text{ALAT}} \text{L-Glutamate} + \text{Pyruvate}$$

$$\text{Pyruvate} + \text{NADH} + H \xrightarrow{\text{LDH}} \text{L-Lactate} + \text{NAD}$$

Calculations: Enzyme activity [U/L] = (A/min)   x   F

	334nm	340nm	365nm
F	2184	2143	3971

Creatine Kinase

Principle: Determination of the CK activity according to the following reactions:

$$\text{Creatine phosphate} + \text{ADP} \xrightarrow{\text{Creatine Kinase}} \text{Creatine} + \text{ATP}$$

$$\text{ATP} + \text{D-Glucose} \xrightarrow{\text{Hexokinase}} \text{D-Glucose-6-phosphate} + \text{ADP}$$

$$\text{G-6-P} + \text{NADP} \xrightarrow{\text{G-6-PDH}} \text{DGluconate-6-phosphate} + \text{NADPH} + H$$

Calculation: At 340 nm, with a 1cm light path cuvette:

Activity (U/L) =   A/min. x 4125

Lactate Dehydrogenase (LDH): LDH was determined using invitro diagnostic reagent for quantitative estimation of LDH in serum with photometric systems using kit (Merck).
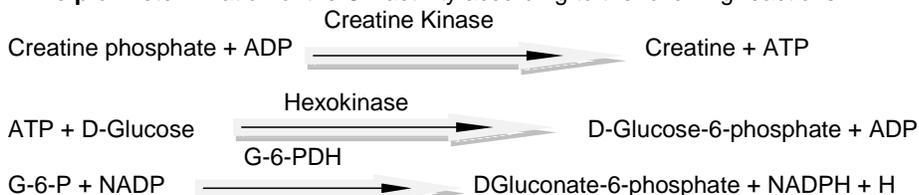
Principle:

$$\text{Pyruvate} + \text{NADH} + H \xrightarrow{\text{LDH}} \text{Lactate} + \text{NAD}^+$$

Reagents

R1   5x20 ml   R2   1x25 ml

Calculation: From absorbance readings calculated, (A / min) and multiply by the corresponding factor from table below:

($\triangle$A / min) x factor = LDH activity [U/L]

RESULTS AND DISCUSSION

The case study was performed on 52 MI patients after the confirmation of physician in Aziz Bhatti Shaheed Hospital, Gujrat. Their blood samples were drawn with their consent and serum lipid profile examined. The association between serum lipid profile and myocardial infarction is well established (Narayana et al., 2011).

Low density lipoproteins (LDL) are seen to be increasingly high among the patients suffering from myocardial infarction whereas; the serum levels of High density lipoproteins (HDL) show a significant decrease. A general rise in serum triglycerides was also observed. A high level of serum LDL and Triglycerides elevates the total cholesterol therefore, a rise in total cholesterol level of the patients was also observed.

The present study also shows a significant increase in the enzymes CK-MB, LDH and AST. The enzyme ALT did not show much alteration. When the plaque formed in the artery in acute ischemia ruptures in myocardial infarction, the levels of these enzymes and troponin-1 rises (Reddy et al., 2012). This study showed these enzymes can be used as indicators of myocardial infarction and can serve as important diagnostic enzymes.

Cardiovascular risk factors were assessed using a self administered questionnaire and various laboratory procedures. Blood pressure of the patients was s using a mercury sphygmomanometer. BMI was calculated using the formula weight (kg) divided by height(m^2). Socioeconomic status of the patients was also recorded. Dietary habits of the patients were classified according to two categories vegetarian and non-vegetarian depending upon the consumption of quantity of red meat. Smoking, alcohol consumption and drug history of the patient was also recorded.

Patients suffering from Myocardial infarction mortality rate are highest in underweight and obese people (Abdulla et al., 2012). Compared with normal weight [body mass index (BMI) 18.5–24.9kg/m2], obesity (BMI-35kg/m2) was associated with increased risk of death in patients with MI (Abdulla et al., 2012). Underweight (BMI, 18.5kg/m2) patients were in increased death risk regardless of MI (Abdulla et al., 2012).

In the present study it is established that people who are obese or have a higher BMI are at a higher risk of mortality by Myocardial infarction as compared to people having a normal BMI in accordance with the findings of Abdulla et al., 2012. BMI has been considered as the major regression factor and further analysis has been carried out in accordance with it. Other risk factors of MI i.e., Hypertension, Cholesterol, HDL and levels of cardiac enzymes have been plotted in graphs against BMI and are discussed later.

Smoking is one of the major modifiable risk factors in myocardial infarction (Prescott et al., 1998). Studies suggest that cigarette smoking seems to play the most important role for having a MI in individuals below the age of 36 (Panaqiotakos et al., 2007). Infact, smoking is the strongest discriminator for MI (Panaqiotakos et al., 2007). Relative mortality rate from Myocardial infarction in female smokers is higher as compared to male smokers because of constituents of tobacco smoke exerting anti-oestrogenic effects (Prescott et al., 1998). Studies have shown that risk of myocardial infarction is reduced by 50% within the first year of quitting smoking (Prescott et al., 1998).

A total of 52 patients were analyzed in the present study, out of which 26 were male and 26 female. Out of the 26 male patients 18 were found to be smokers and 8 were non-smokers Multivariate logistic regression analysis showed that smoking increased 6-folds the risk of having a Myocardial Infarction (Table. 5).

Another important risk factor of myocardial infarction is Hypertension (Picariello et al., 2011). There are two reasons that it is considered as a risk factor. First is that both diseases share the same genetic risk factors i.e., insulin resistance, sympathetic hyperactivity and vasoactive substances (i.e., angiotensin II). Second reason is that hypertension is a major factor associated with the development of atherosclerosis (Picariello et al., 2011).

Table 4: Correlation analysis for parameters

	Cholesterol	TAG(Tg)	HDL	LDL	Creatine kinase	AST	ALT
Cholesterol	1						
TAG(Tg)	-0.036	1					
HDL	-0.008	0.274*	1				
LDL	-0.163	-0.155	0.008	1			
Creatine kinase	-0.145	0.018	0.133	-0.038	1		
AST	-0.011	-0.333*	-0.147	-0.115	0.016	1	
ALT	-0.104	-0.043	0.109	0.046	-0.071	0.266	1
Lactate dehydrogenase	-0.015	0.143	-0.129	-0.094	0.045	-0.101	0.129

* correlation is significant at P<0.05

Table 5: Case summary for basic health symptoms and gender wise distribution analysis

Variables	Male	Female	Total (n=52)	P–value*
Age group				
20 to 40 years	3	5	8	n.s
41 to 60 years	15	14	29	
Above 60 years	8	7	15	
Palpitation				
Yes	26	23	47	n.s
No	2	3	5	
Chest pain				
Yes	22	25	47	n.s
No	4	1	5	
Diabetic				
Yes	6	17	23	< 0.01
No	20	9	29	
Hypertensive				
Yes	20	24	44	n.s
No	6	2	8	
Joints Pain				
Yes	4	6	10	n.s
No	22	20	42	
Diet preference				
Vegetarian	9	20	29	< 0.01
Non-vegetarian	17	6	23	
Smoking				
Yes	18	0	18	< 0.001
No	8	26	34	
Alcohol				
Yes	2	0	2	n.s
No	24	26	50	
Corticosteroids				
Yes	26	26	52	n.s
No	0	0	0	
Use of lipid lowering drugs				
Yes	1	1	2	n.s
No	25	25	50	
Use of Disprin/Asprin				
Yes	2	8	10	< 0.05
No	24	18	42	
Continued Physical activity				
Yes	11	8	19	n.s
No	15	18	33	

*chi-square test p-value

Figure 1: Effect of BMI on systolic blood pressure detected in the study cohorts

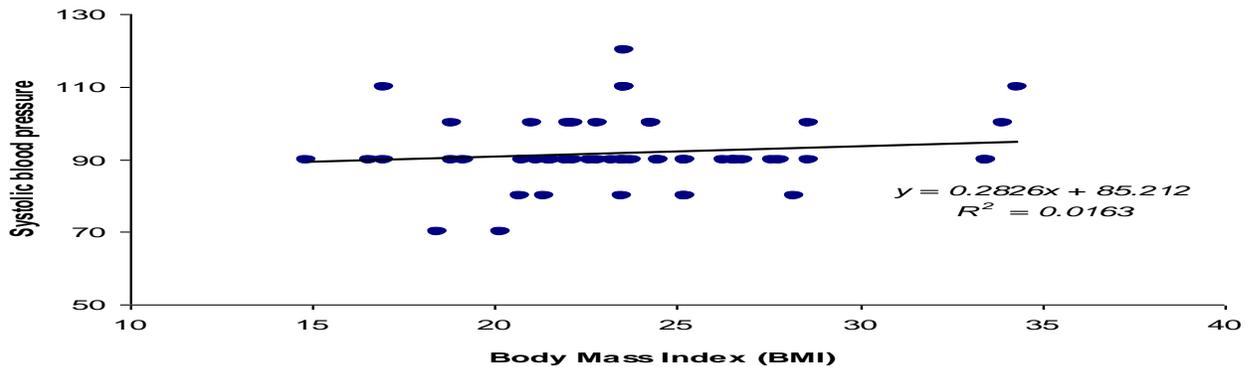$$y = 0.2826x + 85.212$$
$$R^2 = 0.0163$$

Figure 2: Effect of BMI on blood cholesterol levels detected among surveyed population
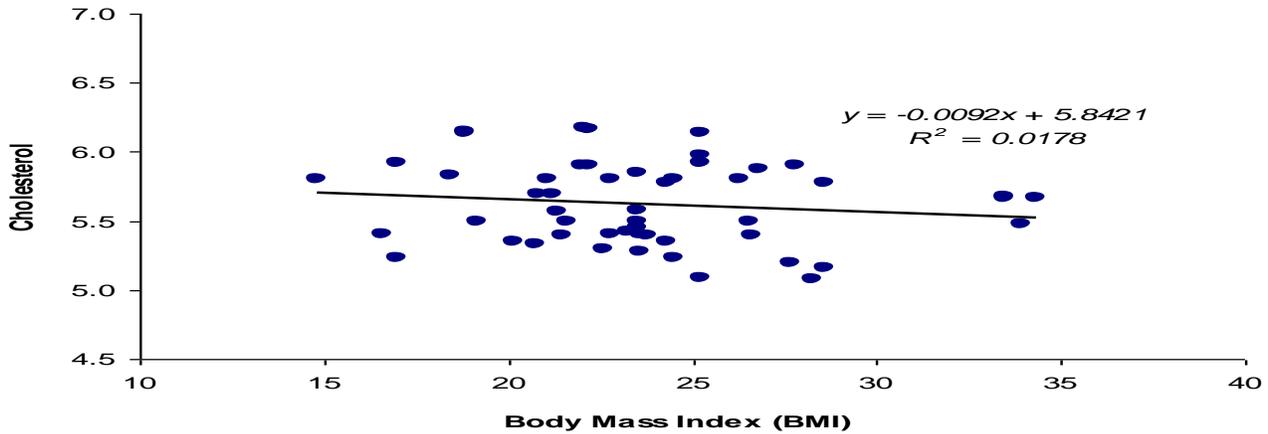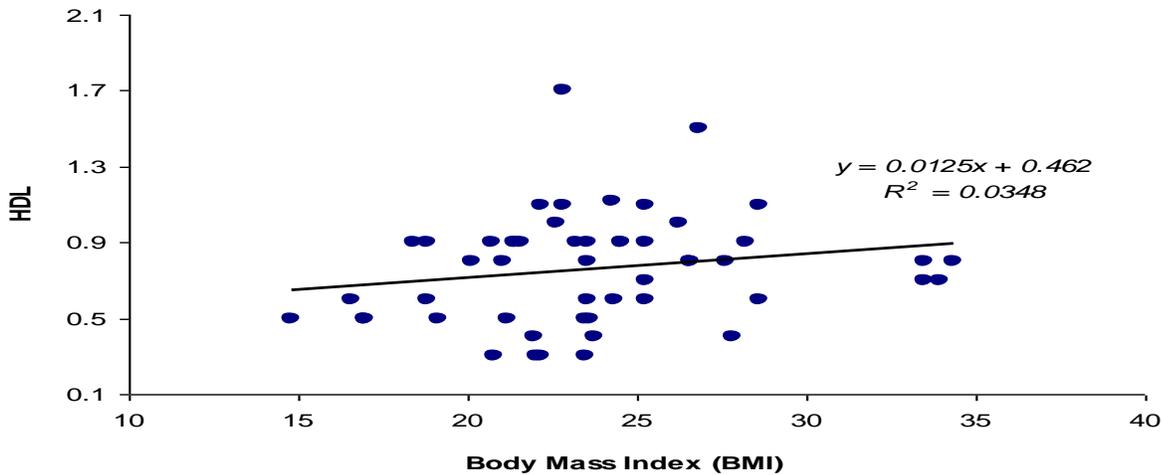
Fig. 3: Effect of BMI on estimated range of HDL in study cohorts.

In the present study 44 out of 52 patients were hypertensive (Table 2), showing that hypertension greatly increases the chances of myocardial infarction. Considering BMI as major category for regression a graph has been plotted against BMI and Hypertension.

The Figure 1 shows the relation between BMI and Hypertension. The increase in BMI increases the risk of hypertension as reported in Picariello *et al.*, 2011. 44 patients out of a total 52 patients assessed suffering from MI were found to have either a previous history of Hypertension before MI or suffered from hypertension along with MI. This finding is in accordance with findings of Picariello *et al.,* 2011

Serum lipid profile was undertaken as a major risk factor for patients of Myocardial infarction. Correlation analysis for lipid profile is shown in table.4, figure.2 and figure.3.

A significant decrease in total HDL and an increase in serum triglycerides and LDL of patients is observed as shown in Shirafkan *et al.* 2012.

Figure.2 and 3 show a correlation between Cholesterol and BMI and HDL and BMI of the subjects. Increased BMI indicates an increase in total cholesterol, but a general decrease in HDL is shown in patients of Myocardial infarction. This decrease in HDL suggests an increase in LDL and Triglycerides in the blood serum.

REFERENCES

1. Karthikeyan G, Teo K K, Islam S, McQueen M J, Pais P, Wang X, Sato H, Lang C C, Sitthi-Amorn C, Pandey MR, Kazmi K, Sanderson JE, Yusuf S. Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians: an analysis from the INTERHEART Study. *J Am Coll Cardiol* 2009; 53:244–253.
2. Shirafkan A, Marjani A and Zaker F. Serum lipid profiles in acute myocardial infarction patients in Gorgan. *Biomedical Research* 2012; 23(1): 119-124.
3. Adak M, and Shivapuri JN. Serum lipid and lipoprotein profile abnormality in predicting the risk of coronary artery disease in non-diabetic patients attending

NMCTH, Birgunj. *Nepal Med Coll J.* 2010; 12(3):158-64.

4. Bitla, R. A, Pallavi, M, Vanaja, V, Suchitra, M. M, Reddy, V. S, Reddy, E. P and Rao, S. Acute Myocardial Infarction in a Southeast Indian Population: Comparison of Traditional and Novel Cardiovascular Risk Factors. *Research Journal of Medicine & Medical Sciences* 2009, 4 (2):202-206.

5. Narayana KS, Koora S, Shaker I A, Basha S S and Babu K S. Comprehensive levels of Serum Enzymes and Lipid Profile testing in MI and Stable Angina Subjects. *Indian J Basic and Applied Medical Research* 2011;1(1):13-21.

6. Reddy R K, Reddy S and Kumar AN. Lipid Profile levels on the second day of Acute Myocardial Infarction; is it the right time for estimation? *Internet Journal of Medical Update* 2012 January;7(1):52-5.

7. Kumar A, Nagtilak S, Sivakanesan R and Gunasekera S. Cardiovascular risk factors in elderly normolipidemic acute myocardial infarct patients--a case controlled study from India. *Southeast Asian J Trop Med Public Health.* 2009; 40(3):581-92.

8. Willey JZ, Xu Q, Boden-Albala B, Paik MC, Moon YP, Sacco RL, et al. Lipid profile components and risk of ischemic stroke: the Northern Manhattan Study (NOMAS). Arch Neurol. 2009;66:1400–1406.

9. Gaddam S, Nimmagadda K, Nagrani T, Nagi M, Wetz R V, Weiserbs K F, McCord D, Ghavami F Gala B and Lafferty J C. Serum lipoprotein levels in takotsubo cardiomyopathy vs. myocardial infarction. *Int Arch Med.* 2011; 4: 14.

10. Kumar PJ, and Clark ML. Cardiovascular diseases. Clinical Medicine 5th ed. W. B. Saunders, 2002. 766-783.

11. Ahmed IA. Myocardial-infraction based on intelligent techniques. *Am J Applied Sci* 2010; 7: 349-351.

12. Jahromi AS, Shojaie M, Dana S, and Madani A. Anti-cardiolipin antibody in acute myocardial infarction. *Am J Immunol* 2010; 6: 11-14.

13. Chizynski K, and Rozycka. Is hyperuecemia a cardiovascular risk factor? *Wiad Lek* 2006; 59(5-6):364-367.

14. Panagiotakos DB, Rallidis LS, Pitsavos C, Stefanadis C, and Kremastinos D. Cigarette smoking and myocardial infarction in young men and women: a case-control study. *Int J Cardiol.* 2007; 116(3):371-5.

15. Prescott E, Hippe M, Schnohr P, Hein OH and Vestbo J. Smoking and risk of myocardial infarction in women and men: longitudinal population study. *: BMJ* 1998;316:1043.

16. Abdulla J, Kober L, Abildstorm Z S, Christensen E, James WPT and Torp-Pedersen C. Impact of obesity as a mortality predictor in high-risk patients with myocardial infarction or chronic heart failure: a pooled analysis of five registries. *European Heart J* 2008; 29:594-601.

17. Fazlinezhad A, Shakeri MT, Taghavi S, Ziallhagh R, Abbaszadegan MR, Ghafarrzadegan K. Lipoprotein (a) level in acute myocardial infarction: comparison with healthy subjects. *Shiraz E-Medical Journal* 2005; 6(1-2).

18. Schwartz R S, Kullo IJ and Edwards DW. Hyperlipidemia and other risk factors. Mayo Clinical Cardiology Review. 2005 2nd ed. Murphy JG Lippincott Wilkins Pub pp115-136.